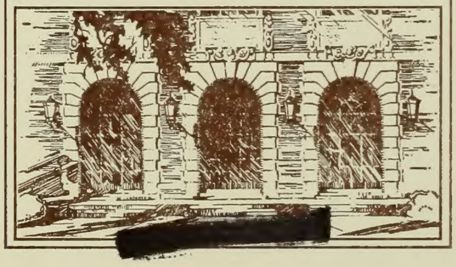
LIBRARY OF THE
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

616.99405
CAR
v.11
no.1-6



[redacted]

LIBRARY U. OF I. URBANA-CHAMPAIGN

[illegible]

CARCINOGENESIS ABSTRACTS

LIBRARY U. OF I. URBANA-CHAMPAIGN

.99405
R

JULY 1972-JANUARY 1973
Abstract & Citation Nos. 0001-0600

Vol. 11
No. 1

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health

LIBRARY U. OF I.

140

Volume XI, No. 1
uly 1972 - January 1973

Abstract and Citation Nos. 0001 - 0600

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

Editor
Robert Love, M.D.
Jefferson Medical College, Philadelphia

Associate Editor
George P. Studzinski, M.D.
Jefferson Medical College, Philadelphia

NCI Staff Consultants
Elizabeth Weisburger, Ph.D.
Sidney Siegel, Ph.D.

THE LIBRARY OF THE
SEP 25 1973
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Etiology Area
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication
approved by the Director of the Bureau of
the Budget on July 25, 1967.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For ournals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical eriodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

CTH	adrenocorticotropic hormone	mg	milligram(s)
DP	adenosine diphosphate	min	minute(s)
MP	adenosine monophosphate	ml	milliliter(s)
TP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	U	unit(s)
kg	kilogram(s)	UV	ultraviolet
LD_{50}	median lethal dose(s)	WBC	white blood cells (leukocytes), white blood count
m	meter(s)		
M	molar	wk	week(s)
mEq	milliequivalent(s)	wt	weight(s)
mM	millimolar	yr	year(s)
μM	micromolar		
mC,μC	milli-,microcurie(s)		

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	0001-0023	1
CHEMICAL CARCINOGENESIS	(Chem)	0024-0201	5
PHYSICAL CARCINOGENESIS	(Phys)	0202-0218	34
VIRAL CARCINOGENESIS	(Viral)	0219-0351	37
IMMUNOLOGY	(Immun)	0352-0537	58
PATHOGENESIS	(Path)	0538-0559	82
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	0560-0577	85
MISCELLANEOUS	(Misc)	0578-0600	89
AUTHOR INDEX			
SUBJECT INDEX			

001 OBSERVATION ON THE EPIDEMIOLOGY OF BREAST CANCER 1971. *(E.)* Macdonald, E. J. nderson Hosp., Houston, Texas). *S Afr Cancer Bull* (3):79-86, 1972.

iscoveries of factors involved in the etiology of ammary cancer are reviewed. Hormone dependency f breast cancer is discussed, together with the inding of a correlation between breast cancer nd androgen and corticosteroid metabolite xcretion. Studies are described which suggest hat the central nervous system (CNS) is important n malignant tumor development, CNS depression avoring tumor growth and CNS excitation hindering t. Data which suggest a connection between breast ancer and the S blood-antigen system are detailed. everal reports indicate that breast cancer incience is lower in communities with little immigraion. It is hypothesized that if a virus or group f viruses plays a part in causing breast cancer, he high incidence of this disease in regions of reat population motility might be based on the act that such population movement would result n an ever-changing viral picture and preclude ffective immunization of the resident population. n alternate hypothesis links low breast cancer ncidence with high economic status. Though conomic status may be relevant, it is believed ot to be a major contributing factor since atin women in every community surveyed to ate have lower breast cancer rates than Anglo omen and generally the economic status of Latins n the U.S. population is lower than that of Anglos. t may be that genetic characteristics play a part n reducing incidence among Latin women. 26 references)

002 THE LEEUWENHOEK LECTURE, 1971: TUMOUR VIRUSES AND THE SOCIOLOGY OF FIBROBLASTS. *E.)* Stoker, M. G. P. (Imperial Cancer Res. Fund Lab., London, England). *Proc R Soc Lond (Biol)* 181(1062):1-17, 1972.

A mechanism for viral carcinogenesis is proposed based on reported studies with cultured fibroblasts. These studies suggest that fibroblasts possess a control system which responds to the social environment of the cell and which regulates entry of resting cells into the growth cycle. Environmental factors which figure in the control system include the presence of growth factors, cell density, the local spatial relationships between individual cells, and cell membrane constitution. According to the theory of viral carcinogenesis, tumor viruses depend on DNA synthesis for their growth; the viruses themselves have only limited genetic information and they must use the enzymes and precursors needed for DNA synthesis in the host cell. Viruses that could induce resting cells to begin DNA synthesis would have an evolutionary advantage since few available cells are growing and synthesizing DNA at any one time. It is possible that tumor viruses induce the required DNA synthesis by the economical use of one or a few genes to perturb the host cell's control system, a system like that reported for fibroblasts. If the asocial cell that is produced survives the infection and continues to carry the functioning viral genes, the host cell is transformed into a cancer cell. (54 references)

0003 RNA-DIRECTED DNA SYNTHESIS. *(E.)* Temin, H. M. (McArdle Lab., U. Wisconsin, Madison). *Sci Amer* 226(1):25-33, 1972.

Work leading to the discovery of RNA-dependent DNA polymerase in animal and human tumor cells is reviewed. DNA polymerase in RNA tumor viruses may explain the cancerous transformations observed in virus-infected animals and also account for viral latency. All DNA polymerases are capable of transcribing information from RNA to DNA; and once an RNA virus has transferred its genetic information to DNA, it would be able to remain latent in a cell and be replicated by the cellular enzyme systems that replicate and repair the cell DNA. After some later activation the virus could reappear and cause "spontaneous" cancer. The discovery of RNA-directed DNA synthesis is particularly important for an understanding of cancer in man since it has removed the dichotomy between viral and genetic theories of the origin of cancer. It also means that simple biochemical tests can be used to determine whether newly discovered human viruses belong to the RNA viruses that induce tumors and cancerous transformation in animal cells. (No references)

0004 THE SIGNIFICANCE OF THE HL-A-ANTIGEN SYSTEM IN TUMOR RESEARCH. *(Ger.)* Kuwert, E. (Inst. Med. Microbiol. U. Essen, W. Germany) and J. Bertrams. *Z Krebsforsch* 78(2): 204-218, 1972.

A description of the HL-A system is presented together with a review of transplantation antigens and carcinogenesis in experimental animals. The relationship between HL-A antigens and tumor disease is presented. The histocompatibility antigens play an important role in tumor virus susceptibility in virus-induced carcinogenesis. The genetic control of the immune response is located on certain well-defined sites of the genome. Leukemogenesis in mice can be interpreted as a result of a congenital immune response lack under certain histocompatibility conditions. Results relative to a presumptive connection between HL-A phenotype (or haplotype) and tumor type of man are summarized. Most authors agree that certain differences in HL-A-antigen frequencies between tumor patients (especially with lymphogranulomatosis and leukemia) and healthy individuals of the same population indeed do exist. The biological significance of these statistical differences for the tumor growth, however, remains still unknown. When comparing HL-A antigens of tumor cells and other nucleated cells of the tumor carrier no additional HL-A specificities were observed. Several authors, however, discovered a loss of HL-A antigens on the neoplastic cell membrane. Apparently, only the cells carrying the same HL-A system as the normal cells or those with missing HL-antigens may undergo loss of allogeneic control and thus of immunity *in vivo*. (43 references)

0005 FOETAL "ANTIGENS" IN CANCER. *(E.)*

Alexander, P. (Chester Beatty Res. Inst., Sutton, Surrey, England). *Nature* 235(5334): 137-140, 1972.

Several types of "onco-fetal antigens" (OFA) have been detected in sera from cancer patients by use of heterologous antiserum raised against an antigen isolated from a tumor tissue. These include (1) α-feto protein, found in serum of primary hepatoma patients; (2) α_2H ferroprotein, found in young children with tumors of embryonic and nonembryonic origin; (3) carcino-embryonic antigen (CEA), found in patients with gastrointestinal tract tumors and also in patients with carcinomas of the lung and breast; (4) fetal sulphoglyco protein, found in the gastric juice of nearly all patients with stomach carcinoma; and (5) fetal alkaline phosphatase ("Regan iso-enzyme"), found in 4% of patients with a variety of different carcinomas. An OFA with the electrophoretic mobility of a α-globulin, but chemically and serologically distinct from α-feto protein and CEA, has been detected in a variety of malignant and benign tumors. Another OFA has been detected on the surface of mouse and hamster cells transformed by SV40 virus. These different antigens are classified as OFA-associated, OFA-specific, or possibly OFA-transplantation type. To evoke an immunologically mediated host reaction which is cytotoxic to tumor cells, the OFA must be of the transplantation type. That is, it must be antigenic in the tumor-bearing host and situated on the tumor cell membrane if it is to constitute a target by which immune mechanisms kill cells. (55 references)

0006 ETIOLOGY OF HUMAN BREAST CANCER: A REVIEW. *(E.)*

MacMahon, B. (Harvard Sch. Public Hlth., Boston, Mass.), P. Cole and J. Brown. *J Natl Cancer Inst* 50(1):21-42, 1973.

Data on known risk factors for human breast cancer are reviewed. The major predictors of risk are age, geographic area of residence, age at first birth, certain indicators of ovarian activity, history of benign breast disease, and familial history of breast cancer. Specific etiologic hypotheses are also reviewed, with consideration of the extent to which they explain, or are consistent with, the observed patterns of risk distribution. If a human mammary tumor virus exists, exposure is probably widespread, and other causal factors must account for the observed risk patterns. Excretion of low levels of androgen metabolites is associated with increased risk of breast cancer, but probably is only an indicator of other unidentified endocrine patterns. A hypothesis linking breast cancer risk to a particular pattern of estrogen metabolism in early repeoductive life, although unsupported by direct evidence, is compatible with most epidemiologic and experimental observations on the disease. The roles of exogenous estrogens, prolactin, and progesterone-suggested by animal models-remain essentially unevaluated in humans. A small number of human breast cancera are probably caused by exposure to high levels of ionizing radiation. Familial factors seem most strongly implicated in young women with bilateral disease, but even among such patients the nature of the familial factors, genetic or environmental, is unknown. (178 references)

0007 DISTINGUISHING THE ORIGIN OF TUMORS BY MEANS OF IMMUNOLOGICAL METHODS. *(Fr.)*

Grabar, P. (Pasteur Inst., Paris, France). *C R Acad Sci* [*D*] *(Paris)* 275(21):2447-2450, 1972.

Results in cancer etiology research attained by immunological methods are briefly summarized and discussed. The high specificity and sensitivity of the immunological approach provided evidence that part of the viral genome is incorporated into the transformed cell in virus-induced tumors. This portion of the viral genome is capable of inducing the synthesis of new antigens (neo-antigens) which never occur in healthy organism cells. Identical neo-antigens were detected in different animal tumors which had been induced by the same virus. In tumors, induced by chemical or physical agents, no exogenous nucleic acids were introduced into the cell and, therefore, only alterations of endogenous nucleic acids occur. These latter may lead to the formation of modified antigens or to the appearance of normal fetal tissue antigens as well as to the disappearance of certain normal cell constituents from the transformed cell. The antigenicity of chemically-induced tumors varies from one tumor to another even within the same line of animals. It could be concluded that whenever neo-antigens are detected in tumor cells a viral factor might be involved in the etiology of the related tumor. The absence of neo-antigens should indicate something other than viral factors being involved in the cause of neoplasia. A clear-cut borderline between neo-antigens and deeply-modified endogenous antigens is hard to draw, and the limitations of the immunological approach derived therefrom in cancer etiology studies are discussed. (15 references)

0008 POSSIBLE VIRAL ETIOLOGY OF HUMAN BREAST CANCER. *(E.)*

LePage, G. A. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.). *S Afr Cancer Bull* 16(3):87-89, 1972.

In 1936, Bittner discovered that a milk-borne virus s implicated in the high incidence of mammary ncer observed in susceptible mouse strains. cent electron microscope observations of human lk samples revealed the presence of virus particles sembling those in murine milk. Virus particle cidence was 5% for American women from families th no history of breast cancer, 60% for women th history of the disease in their families, d 39% for Parsi women in Bombay. Breast cancer counts for 49% of all cancers in Parsi women; n an age-adjusted basis, the Parsi incidence s 1.7 times greater than that of other women n Bombay. These observations suggest some ssociation between the particles and breast ancer. However, the combination of an increased cidence of the disease in American women and a reatly decreased incidence of breast feeding oes not support the hypothesis that human breast ancer is transmitted in milk as it is in the ouse. If the virus is transmitted vertically n the sex cells, as in some mouse strains, r through the placenta, immunotherapy would be seless because of immunotolerance. (6 references)

009 NITROSAMINES: A REVIEW. *(E.)* Sebranek, J. G. (Dept. Meat Animal Sci., U. isconsin, Madison) and R. G. Cassens. *J Milk ood Technol* 36(2):79-91, 1973.

itrosamines have become a recent problem for he food industry since it is thought that certain itrosamine precursors, such as sodium nitrite, which re commonly used in some foods, may lead to ignificant hazards in the food supply. This eview draws together recent information and evalua- es the problem of nitrosamines in foods. Chemistry f nitroso compounds is discussed and results of everal animal studies are cited to emphasize the xtreme toxic and carcinogenic potential of some f these compounds. The mechanism of action in umor development is considered. Although not et entirely clear, the most acceptable idea nvolves alkylation of cellular components such as NA, RNA, and proteins which leads to changes in ormal cell growth. Presence of nitrosamines n foods has been documented in several instances ut more recent analytical techniques involving as chromatography and mass spectrometry have ndicated that levels in foods may be lower than reviously believed. The potential for formation f nitrosamines from nitrite and secondary mines is discussed, in relation to food before onsumption and to formation in the body, par- ticularly the stomach, after consumption. Finally, nalytical procedures are reviewed and briefly valuated and some other possible sources of nitro- amines in the environment are considered. 131 references)

0010 THE CARCINOGENIC EFFECTS OF LOW LEVEL RADIATION. A RE-APPRAISAL OF EPIDEMIOLO- GISTS METHODS AND OBSERVATIONS. *(E.)* Stewart, A. (Dept. Social Med., Oxford U., England). *Health Phys* 24(2):223-240, 1973.

The Oxford survey and related studies of leukemia mortality have shown that it is unusual to be X-rayed within 6 months of conception, but not uncommon for a spontaneous cancer to be initiated during the first half of fetal life and to wind up as a stillbirth, a neonatal death or a fatal infection. These missed cases tend to be associated with early initiations, and with cancers which run an exceptionally acute course without tumor formation, so they have affected the recognition of leukemias more than other cancers, myeloid leukemias more than lymphatic ones, and spontaneous cancers more than radiogenic ones. The unrecognized cases are also associated with difficult births and difficult living conditions so they have affected X-rayed fetuses more than other fetuses, negroes more than caucasians, and A-bomb survivors more than other populations. A concentration of unrecognized cancers in the youngest age groups, and in populations with a high all-causes death rate, explains why studies of the delayed effects of prenatal irradiation which have allowed the children to be followed for less than 5 yr are not on a par with studies of cancer deaths before 10 yr of age, and why A-bomb survivors are in a class by themselves. The mistaken diagnoses do not account for the "extra" X-rayed cases in the Oxford survey. The age distributions and cell types of these cases suggest that they are a direct consequence of radiation-induced mutations, and may be the only numerically important group of childhood cancers to be caused by an external agent. (52 references)

0011 CHROMOSOMAL FEATURES OCCURRING IN HUMAN NEOPLASTIC DISEASES. *(Ger.)* Hossfeld, D. K. (Ruhr U. Tumor Res. Dept., Essen, W. Germany). *Z Krebsforsch* 78(2):123-128, 1972.

Current results on chromosome analyses of human tumors and leukemias are summerized. Except for the Philadelphia-chromosome (Ph^1) in chronic myelocytic leukemia no specific chromosomal anomalies in any other neoplastic disease are known to have diagnostic or prognostic significance. Aneuploidy ot tumor cells should not be considered an absolute criterion for malignant growth: it occurs in any tumor. (26 references)

0012 IMMUNOLOGICAL DISORDERS IN HODGKIN'S DISEASE. *(Ger.)* Scheurlen, P. G. (U. Med. Clin. Homburg/Saar, W. Germany). *Z Krebsforsch* 78(2):192-203, 1972.

Immunological disorders associated with Hodgkin's disease are reviewed. Systemic infectious diseases occur more often in Hodgkin's disease than in other cancer patients. Tuberculosis, fungal or viral diseases are most frequent while bacterial infections are rare. Infection susceptibility increases in the advanced stages of the disease. Transplant rejection and delayed sensitivity reactions are diminished. Serum immunoglobulin concentrations are within normal except for rare cases of hypogammaglobulinemia or monoclonal gammopathy. Antibody formation is impaired only if the primary contact with the antigen occurs after the onset of the disease. Only the concentration of β-1A-globulin, a constituent of the complement fixation system C'3, is increased in most of the Hodgkin's disease patients as in other cancer patients. The impairment of the immune defense processes cannot be explained by the decrease in lymphocytes occurring mainly during the late stages of the disease. A functional impairment of the lymphocyte must be involved: peripheral lymphocytes often show decreased blastic transformation and DNA synthesis upon phytohemagglutinin stimulation *in vitro*. The serum of Hodgkin's disease patients exhibits an inhibitory effect on the blastic transformation of normal donor mitogen-stimulated lymphocytes *in vitro*. A serum protein fraction, differing from the normal serum proteins and eliciting such inhibitory effects was separated by chromatography on Sephadex 200 from Hodgkin's disease and carcinoma patients. No other information as to the constitution of this inhibitory factor is available as yet. (88 references)

0013 CURRENT PROBLEMS IN CARCINOGENESIS. *(Ger.)* Hecker, E. (German Cancer Res. Ctr. Heidelberg, W. Germany). *Z Krebsforsch* 78(1):99-122, 1972.

Current problems related to the etiology of neoplastic disease are reviewed. Attention is focused on exogenous factors related to environmental pollution and plant and other food constituents and to endogenous factors dealing with intermediary metabolism products, hormones, and latent viruses. For practical and theoretical reasons it is proposed that in tumor etiology, the impact of one, physically or chemically unequivocally defined factor be referred to as solitary carcinogenesis and the impact of more than one physically or chemically unequivocally defined factors called syncarcinogenesis. Recent findings demonstrate that syncarcinogenesis may be caused either by several solitary carcinogens of identical organotropy (pluricarcinogenesis) or by a combination of solitary and cocarcinogens of identical organotropy (cocarcinogenesis). Of the physical factors involved in carcinogenesis, radioactivity, x-ray and UV radiation are referred to. Chemical carcinogenesis is discussed in terms of occupational exposure to known carcinogens as well as to drug induced neoplasia at the experimental level. Industrially occurring cocarcinogens such as phenols, n-dodecan, Tween 60, 1,8,9-trihydroxyanthracene, and ferric oxide are discussed. Carcinogens of plant origin are reviewed in terms of available data on senecionins, cycasine, and aflatoxins, and cocarcinogens such as croton oil and phorbol esters are discussed. Methylcholanthrene as a product of cholesterol and bile acid metabolism as well as the involvement of steroid hormones in carcinogenesis are discussed as endogenous factors. The presence of antibodies to genital type Herpes virus in sera of patients with cancer of the uterine cervix is referred to in discussing the viral ethiology of human tumors. Cytological and biochemical aspects are considered in terms of models most suited for investigations of biological and molecular mechanisms of carcinogenesis. (103 references)

0014 BIOLOGY OF MALIGNANT NEOPLASIA OF THE LYMPHORETICULAR TISSUES. *(E.)* Dorfman, R. F. (Stanford U. Sch. Med., Calif.). *J Reticuloendothel Soc* 12(3):239-256, 1972. (69 references)

0015 INITIATION OF IMMUNE RESPONSES AGAINST CANCER CELLS. *(E.)* Metcalf, D. (Royal Melbourne Hosp., Australia). *CA* 22(5):308-310, 1972. (No references)

0016 SYMPOSIUM ON MAMMARY NEOPLASIA. *(E.)* Feller, W. F. (Georgetown U. Sch. Med., Washington, D.C.). *CA* 22(5):329-335, 1972. (No references)

0017 CONTROL FOR ASBESTOS. *(E.)* Ahmed, A. K. (Minnesota Public Interest Res. Group), D. F. MacLeod and J. Carmody. *Environment* 14(10):16-22, 27-29, 1972. (77 references)

0018 WHY ARE SMALL-BOWEL TUMOURS SO RARE? *(E.)* Lowenfels, A. B. (Grasslands Hosp., Valhalla, N.Y.). *Lancet* (7793):24-25, 1973. (31 references)

0019 THE JAMES EWING LECTURE. LOOKING AT LUNG CANCER. *(E.)* Watson, W. L. (Memorial Hosp., New York, N.Y.). *Cancer* 31(1):6-9, 1973. (No references)

0020 AN HYPOTHESIS ON THE RELATIONSHIP OF CANCER TO AGE. *(E.)* Sobel, H. (Dept. Biochem., Colorado St. U., Fort Collins). *Proc Am Assoc Cancer Res* 13(March):32, 1972. (No references)

0021 PSYCHOSOMATIC FACTORS INVOLVED IN THE DEVELOPMENT OF MALIGNANT NEOPLASIA. *(Ger.)* Hedri, A. (No affiliation). *Praxis* 61:1076-1077, 1972. (No references)

0022 VIRUS-INDUCED BREAST CANCER IN HUMANS? *(Ger.)* Bandlow, G. (Inst. Hyg. U. Göttingen, W. Germany). *Hippokrates* 43(3):281-289, 1972. (10 references)

0023 CANCER AND ENDOCRINE DYSFUNCTION. *(E.)* Weller, M. (Dunston Hill Hosp., Gateshead, England). *Lancet* (7772):330-331, 1972. (25 references)

024 CHANGES IN FREE RADICAL CONCENTRATION IN CARCINOGENESIS BY DIETHYLNITROSAMINES.

Rus.) Shulyakovskaya, T. S., (Inst. Chem. Phys., cad. Sci. USSR, Moscow), V. V. Kondrat'yeva, S. A. lasova and A. N. Saprin. *Dokl Akad Nauk SSSR* 207(2): 61-463, 1972.

he changes in free radical concentration during a alignant transformation process were studied by eans of electron paramagnetic resonance in rat ivers subjected to diethylnitrosamine (DENA) reatment. Specifically, DENA (5 mg/kg as a 0.01% queous solution) was administered p.o. for 120 days o 500 Wistar rats. The animals were killed in arious periods to study their livers. The free adical concentration in the liver decreased in the irst days after the carcinogen administration, ue to the cytotoxic effect of the carcinogen. The ree radical concentration in the liver reached maximum level in 22 days, followed by a decrease, gradual increase (by 65% in comparison to the ontrol), and a maximum increase in 90-110 ays. Histologically, the last period corresponded o the precancerous stage. There was a correlation etween the high level of the free radicals in he precancerous liver tissue and chemoluminescence f various lipid fractions isolated from the same issue. Most of the rats developed tumors 120-140 ays after the beginning of the experiment, when ecreased free radical concentrations were observed. hus, there were various phasic changes in the liver ree radical concentration: a decrease in the nitial period, an increase in later stages due to he non-specific response of the animals to the oreign substance, an increase in the precancerous tage, and finally a decrease in tumor cells hemselves. Further studies are required to better nderstand the role of the free radical processes in malignant transformation.

0025 INHIBITION OF ESTABLISHED TRANSPLANTS OF CHEMICALLY INDUCED SARCOMAS IN SYNGENEIC MICE BY LYMPHOCYTES FROM IMMUNIZED DONORS.

(E.) Borberg, H. (Sloan-Kettering Inst., New York, N.Y.), H. F. Oettgen, K. Choudry and E. J. Beattie, Jr. *Int J Cancer* 10(3):539-547, 1972.

Sarcomas were induced in BALB/c, C57BL/6 or (C57BL/6 x A)F_1 mice by s.c. injection of 3-methylcholanthrene and grafted onto recipient mice. Syngeneic and allogeneic mice, and sheep, were rendered immune to these tumor transplants by injection with irradiated (10,000 R) tumor cells. Lymphocytes from immune allogeneic or syngeneic mice, or from sheep, were injected i.v. into mice bearing established tumor grafts, and the effect on growing tumors was observed. Specific inhibition of established tumors was seen in mice given immune cells from each source. The inhibitory effect of allogeneic immune mouse lymphocytes was much less marked than that of syngeneic lymphocytes.

0026 EARLY ENHANCEMENT OF PLATING EFFICIENCY OF PRIMARY MOUSE EMBRYO CELLS BY THE CARCINOGEN METHYLNITROSOUREA.

(E.) Frei, J. V. (Fac. Med., U. Western Ontario, London, Canada) and J. Oliver. *Cancer Res* 32(12):2747-2752, 1972.

CFW/D mouse embryo cells were grown in mass cultures and replated regularly once a week. Plating efficiencies were measured in these cells and in cells treated with methylnitrosourea (MNUA) in doses up to 5 mM in culture medium. The proliferation of untreated cells showed a cyclical pattern of change, with peaks of proliferation at 2, 6 and 11 wks after initiation. In MNUA-treated cultures, plating efficiency study showed that cells proliferated more slowly than controls during week 0, at an equal rate with controls during week 1, and more rapidly than controls during and after week 2. In one experiment, in which cells were treated with 3mM MNUA, the plating efficiency was initially 1/4 log less than control; three weeks later, MNUA-treated cells showed plating efficiencies which exceeded controls by 3 logs. This indicated that the change in cell plating efficiency represented a true change in the MNUA-treated cells rather than a selection of certain cell variants. This change may have been independent of contact inhibition, and may be a step in the development of cancer in tissue culture.

0027 CARCINOGENIC ACTIVITY OF SOME BENZ(a)-ANTHRACENE DERIVATIVES IN NEWBORN MICE.

(E.) Roe, F. J. C. (Royal Cancer Hosp., London, England), A. Dipple and B. C. V. Mitchley. *Brit J Cancer* 26(6):461-465, 1972.

Newborn Swiss mice were given single s.c. injections of 200-277 μg of one of the following agents: 7-methylbenz(a)anthracene (group A), 7-bromomethylbenz(a)anthracene (group B), 4-chloro-7-bromomethylbenz(a)anthracene (group C), 7-bromomethyl-12-methylbenz(a)anthracene (group D), or arachis oil (group E, control). Treated mice developed injection site sarcomas, lung carcinomas, liver tumors and malignant lymphomas. Mice in groups D and A had the highest incidence of tumors; tumors were less frequent in group B, and least frequent in group C. In group C, the only activity noted was a slight rise in the risk for liver tumors among male mice.

0028 THE RELATIONSHIP OF ACUTE MESODERMAL CELL DEATH TO THE TERATOGENIC EFFECTS OF 7-OHM-12-MBA IN THE FOETAL RAT.

(E.) Crawford, A. M. (Dept. Path., U. Aberdeen, Scotland), J. F. R. Kerr and A. R. Currie. *Brit J Cancer* 26(6):498-503, 1972.

Pregnant rats were given 2.5 mg/100 g body wt 7-hydroxymethyl-12-methylbenz(a)anthracene (7-OHM-12-MBA), a dose known to cause gross developmental defects in fetal offspring such as spina bifida, encephalocoele and meningomyelocoele. The early effects of 7-OHM-12-MBA on 11-14 day embryos were examined and it appeared that defects in fetuses resulted from cell death in specific regions of the embryonic mesoderm. Large foci of single cell necrosis were found in paraxial mesoderm of embryos from rats given 7-OHM-12-MBA. The distribution of these foci was similar in embryos from rats treated on the same day of gestation but varied with day of treatment. Virtually no necrosis was seen in embryos examined 72 hr after maternal treatment, though these embryos showed gross developmental retardation. At each day of treatment with 7-OHM-12-MBA, the distribution of mesenchymal cell death corresponded precisely to the axial skeletal defects known to occur in fetuses.

0029 MAMMARY CARCINOGENESIS: INFLUENCE OF PARITY AND ESTROGENS. *(E.)* Black, M. M. (New York Med. Coll., N.Y.) and H. P. Leis, Jr. *N Y State J Med* 72(12):1601-1605, 1972.

A study of parity and estrogen pretreatment in a consecutive series of patients with benign and malignant lesions is reported. Prior estrogen therapy in the present study is defined as continuous treatment for a minimum of one year. Most breast cancers in both parous and nulliparous women appeared one to ten yrs after starting estrogen treatment (21 of 28 in nulliparous women and 18 of 23 in parous women). The benign lesions appearing in estrogen-treated women were also concentrated within the first ten years after the start of estrogen treatment (55 of 62). Nonestrogen-treated women under 50 yr old who had malignant lesions were nulliparous in 28% of cases while women in this group with benign lesions were nulliparous in 25% of cases. Twenty-three percent of estrogen-treated women with benign lesions were nulliparous. Notably, however, the percent of nulliparity among estrogen-pretreated patients with malignant lesions under 50 years of age was 61%. In patients with malignant breast lesions developing before age 50 yrs, prior estrogen therapy was more frequent among nulliparous patients than among parous patients (34% vs 11%). Of 40 estrogen-treated parous patients with benign lesions, 34 were parous before age 20 yr. However, in cancer patients with estrogen pretreatment, late and early parity were more equally distributed. Additional data appear to indicate breast lesions arising after estrogen treatment are three times as likely to be malignant in women who were nulliparous beyond the age of 27 than in women who were parous before that age. Further, breast lesions are more likely to be malignant in nulliparous women who have received estrogen treatment than in nulliparous women without such pretreatment. The findings suggest that distinctive relationships exist between parity and breast cancer in estrogen-treated women.

0030 SOME NEW FINE STRUCTURAL OBSERVATIONS OF ETHYLNITROSOUREA-INDUCED NERVE TUMORS IN RATS. *(E.)* Hirano, A. (Montefiore Hosp., Bronx, N.Y.), J. Hasson and H. M. Zimmerman. *Lab Invest* 27(6):555-560, 1972.

Pregnant rats were injected i.v. with 20 mg ethylnitrosourea on gestation day 18. Offspring were killed when five to seven months old. Three nerve sheath tumors from the fifth cranial nerve of these offspring, and two tumors from spinal nerve roots, were examined electron microscopically. Tumor cells showed most of the features previously observed by others. However, tumor blood vessels were found to be lined by a single layer of distinctive fenestrated endothelial cells. In this, the vessels resembled those seen in neurolemomas. Also, the walls of cysts within tumors were made up of single layers of tumor cells arranged to exhibit a distinct polarity. Nuclei of these cells were usually located basally, while cytoplasmic organelles accumulated in the apical zones of most cells.

0031 EFFECT OF NITROSAMINES ON NURSING SYRIAN GOLDEN HAMSTERS AND THEIR OFFSPRING. *(E.)* Mohr, U. (Inst. Exp. Med. Path., Hannover, Germany), J. Althoff, A. Emminger, H. Bredch and R. Spielhoff. *Z Krebsforsch* 78(1):73-77, 1972.

Nursing hamsters were given s.c. injections of 5, 10 or 20 mg/kg/day N-diethylnitrosamine (DEN) or 300, 600 or 1200 mg/kg/day N-dibutylnitrosamine (DBN). Tumor development in these hamsters and in their offspring was observed. Parents on DEN developed respiratory system tumors, including papillary nasal cavity lesions, and papillomas of the larynx, trachea and stem-bronchi. Nasal cavity tumors were not seen in parents given DBN, though DBN did induce other respiratory system tumors. Offspring of DEN- and DBN-treated nursing hamsters developed tumors of the renal cortex and respiratory system, including the larynx, trachea and stem-bronchi. The number of tumors increased in the F_1 generation as the doses of nitrosamine given to the parent generation were increased. It was found that DEN appeared unchanged and in measurable quantities in the milk of treated nursing hamsters.

0032 MORPHOLOGY OF EXPERIMENTAL CHORIONIC TUMORS IN RATS. *(E.)* Miyamoto, M. (Osaka U. Med. Sch., Japan), N. Nilsuwarn and S. Angsubhakorn. *Acta Pathol Jap* 22(2):343-352, 1972.

Chorionic tumors were induced in four rats given 0.25 or 2.5 mg 7,12-dimethylbenz(a)anthracene or 0.25 mg 4-nitroquinoline-1-oxide. One tumor was transplanted. Histological examinations of the chorionic tumors revealed varying histopathological pictures. Some tumors resembled normal placenta histologically. In others, treated rats showed a cystic mass with chorionic tumors developing from the wall of this mass.

033 CARCINOGENESIS IN TISSUE CULTURE. 20: ELECTROKINETIC CHANGES IN CULTURED RAT IVER CELLS ASSOCIATED WITH MALIGNANT TRANSFORMATION N VITRO.

(E.) Yamada, T. (Natl. Cancer Ctr. es. Inst., Tokyo, Japan), T. Takaoka, H. Katsuta, . Namba and J. Sato. *Jap J Exp Med* 42(4):377-388, 972.

he electrophoretic mobility patterns of normal at liver cells, spontaneously transformed liver ells, malignant rat hepatocarcinoma cells and rat iver cells transformed by single or repeated xposures to 4-nitroquinoline-1-oxide (4NQO) *in itro* were compared. Normal cells showed a uni- orm distribution of relatively low electrophoretic obility values. Hepatocarcinoma cells showed enerally higher mobilities distributed over a ider range. Mean mobilities of normal cells ere increased by neuraminidase treatment of cells, hile neuraminidase reduced mean mobilities of alignant tumor cells. Spontaneously transformed ells were intermediate between normal and malig- ant cells in mean electrophoretic mobility, istribution of values and susceptibility to euraminidase. 4NQO-transformed cells generally howed electrophoretic mobility patterns similar o those of spontaneously transformed cells. On eculturation of transplanted malignant, normal nd transformed cells, all cells showed patterns f electrophoretic mobility similar to those riginally seen in malignant tumor cells.

034 EFFECTS OF THYMECTOMY, SPLENECTOMY AND 3-METHYLCHOLANTHRENE ON NEOPLASIA EXPRESSION, INCIDENCE AND LATENCY IN AKR MICE.

(E.) Whitmire, C. E. (Microbiol. Assoc., Inc., Bethesda, Md.), R. A. Salerno and L. S. Rabstein. *Proc Soc Exp Biol Med* 141(3):890-894, 1972.

In light of recent studies demonstrating that both leukemia and 3-methylcholanthrene (3MC) induced tumors rarely develop together in Balb/c mice, a study was undertaken to determine the effects of thymectomy or splenectomy on the incidence of these neoplasms in female AKR mice. Thymectomy, but not splenectomy, has been shown to reduce the incidence of spontaneous leukemia in AKR mice. In intact and splenectomized mice, a single s.c. injection of 3MC (150 μg) increased the total incidence of neoplasms over that of non-3MC-treated animals. The increase was due to a higher incidence of s.c. tumors at the site of 3MC inoculation and not to a change in the incidence of leukemias. Thymectomy totally eliminated the development of leukemia in control AKR mice and greatly reduced its incidence in the 3MC-treated mice (1 of 18). Even though leukemia was eliminated, the incidence of sarcomas in thymectomized mice was not increased. Both thymectomy and splenectomy reduced the average latency period for sarcoma by four and two weeks resp. Complement fixation and gel diffusion tests revealed a high level of viral gs-1 antigen in all tissues taken from control and tumor-bearing mice and from 3MC-induced tumors of tumor-bearing mice. Since no enhancement of sarcoma induction occurred in the absence of leukemia, a high endogenous level of type-C RNA virus genome expression may at least partly be responsible for the relatively low incidence of sarcoma induction in 3MC-treated AKR mice.

0035 THE SUPPRESSION OF NON-SPECIFIC ESTERASE ACTIVITY IN MOUSE SKIN SEBACEOUS GLAND BY "CS" GAS.

(E.) Barry, D. H. (Huntingdon Res. Ctr, England), L. F. Chasseaud, B. Hunter, and W. E. Robinson. *Nature* 240(5383):560-561, 1972.

The compound *o*-chlorobenzylidenemalononitrile (CS), a riot control agent, was assayed to determine its carcinogenic potential. CS (1% in acetone) and two related αβ-unsaturated compounds, cinnamonitrile (5% in acetone) and cinnamaldehyde (0.1% in acetone), were each painted onto the skin of different CFLP female mice on three consecutive days. A fourth group of mice painted with acetone served as controls. On the third day after the final application, pieces of skin were taken from the center of each painted area and sectioned in a cryostat. Non-specific esterase activity was then assayed using an image analyzing computer. Of the three substances tested, only CS suppressed non-specific esterase activity, a property previously shown to be associated with carcinogenic activity. The extent of CS-induced enzyme suppression in painted mouse skin was similar to that produced by 7,12-dimethylbenz(a)anthracene. It was therefore suggested that CS be fully investigated for carcinogenic activity.

0036 THE EFFECT OF ASCORBIC ACID UPON BLADDER UPTAKE OF β-NAPHTHYLAMINE METABOLITES.

(E.) Alam, B. S. (Tulane U. Sch. Med., New Orleans, La.), F. E. Jaramillo, J. U. Schlegel and T. A. DeRouen. *Proc Soc Exp Biol Med* 141(3):1008-1013, 1972.

The effect of ascorbic acid was studied on the bladder reabsorption of various ^{3}H-labeled metabolites of β-naphthylamine, a potent bladder carcinogen. Buffered ^{3}H-labeled β-naphthylamine metabolites which had been extracted from the urine of a carcinogen-fed dog were treated with ascorbic acid, introduced by catheter into the empty bladder of a male rabbit, and the bladder was incubated for two hr. Measurements by scintillation counter of the amount of ^{3}H taken up by rabbit bladder tissues showed that less radioactivity was incorporated in the presence of ascorbic acid-treated β-naphthylamine metabolites than in the presence of non-treated metabolites. Correspondingly, more radioactivity was retained in the instilled bladder ascorbic acid-metabolite solution than in the nontreated metabolite solution. It was therefore concluded that the presence of ascorbic acid in the bladders of rabbits decreased the uptake of radioactivity from the urinary metabolites of ^{3}H-β-naphthylamine.

0037 EFFECTS OF ADJUVANTS ON BENZPYRENE TUMORIGENESIS IN HAMSTERS. *(E.)* Pinkerton, H. (St. Louis U. Sch. Med., Mo.). *Proc Soc Exp Biol Med* 141(3):1089-1091, 1972.

Three nonspecific immunoenhancing adjuvants, Friend's complete adjuvant (FCA), pertussis vaccine (Pert), and BCG, were studied for their ability to enhance benzo(a)pyrene (BP)-induced tumorigenesis in progeny born of female hamsters who had been injected (with one of the adjuvants) s.c. while pregnant. Male and female offspring which received three weekly s.c. injections of BP (3 mg each) proved to be equally susceptible to increased BP tumorigenesis. These results differ from an earlier study in which males showed a decreased susceptibility. BP-induced tumors in progeny of hamsters injected with one of the adjuvants developed earlier and were significantly greater in number than tumors in progeny of untreated animals. This effect is similar to that reported earlier for Freund's incomplete adjuvant. In contrast to their potentiating effect on tumor development in the progeny, two of the adjuvants (Pert and FCA) exhibited an inhibitory effect on tumorigenesis in both pregnant and nonpregnant female hamsters.

0038 CLEAR-CELL ADENOCARCINOMA OF THE CERVIX AFTER MATERNAL TREATMENT WITH SYNTHETIC ESTROGENS. *(E.)* Noller, K. L. (Mayo Clinic, Rochester, Minn.), D. G. Decker, A. P. Lanier and L. T. Kurland. *Mayo Clin Proc* 47(9):629-630, 1972.

Seven female patients with mesonephric adenocarcinoma of the cervix born after 1939 and seen at the Mayo Clinic after 1964 were evaluated with respect to maternal use of hormones during pregnancy. One mother did not respond and two others definitely had taken no hormones during their pregnancies. The mother of the child diagnosed at age seven had taken diethylstilbestrol (25 mg/day) during the first trimester. The mother of another girl, aged 15 at diagnosis, had taken diethylstilbestrol (5-15 mg/day) during the first trimester. The mothers of the remaining two girls, both diagnosed at age 19, also gave a history of hormone administration during pregnancy. The drug (Dienestrol) was known in only one case and the dosage was unknown for both. The incidence of cervical adenocarcinoma in these patients was much higher than that expected (0.54 case) based on the total at risk population seen at the Mayo Clinic. However, the fact that no cases of cervical or vaginal adenocarcinoma have as yet been observed among the 23,816 childern born at the clinic from 1943 through 1959, including 1,568 whose mothers had received estrogens during pregnancy, suggests that the risk of adenocarcinoma of the vagina or cervix following maternal use of stilbestrol may be low.

0039 MODIFICATIONS OF RIBONUCLEIC ACID BY CHEMICAL CARCINOGENS: MODIFICATION OF *ESCHERICHIA coli* FORMYLMETHIONINE TRANSFER RIBONUCLEIC ACID WITH *N*-ACETOXY-2-ACETYLAMINOFLUORENE. *(E.)* Fujimura, S. (Coll. Phys. Surg., Columbia U., New York, N.Y.), D. Grunberger, G. Carvajal and I. B. Weinstein. *Biochemistry* 11(19):3629-3635, 1972.

Formylmethionine transfer RNA ($\text{tRNA}^{\text{fMet}}$) from *E. coli* was reacted with *N*-acetoxy-9-(^{14}C) acetylaminofluorene (*N*-acetoxy-9-(^{14}C)AAF); this agent was known to bind AAF to the 8 position of guanosine (G) residues in nucleic acids. After separation of radioactive from nonradioactive molecules in the reaction, the modified $\text{tRNA}^{\text{fMet}}$ was cleaved by T_1 or pancreatic ribonuclease. DEAE-Sephadex chromatography indicated that the major site of AAF modification of $\text{tRNA}^{\text{fMet}}$ was the G residue on the 5' side of the dihydrouridine (position 20). The methionine acceptor and transformylase activities of the modified $\text{tRNA}^{\text{fMet}}$ decreased to 40% and had a K_m higher than that of unmodified molecules. AAF modification of G at position 20 in the dihydrouridine loop of $\text{tRNA}^{\text{fMet}}$ did not markedly affect A-U-G codon recognition.

0040 1,1-DIMETHYLHYDRAZINE (UNSYMMETRICAL) CARCINOGENESIS IN MICE. LIGHT MICROSCOPIC AND ULTRASTRUCTURAL STUDIES ON NEOPLASTIC BLOOD VESSELS. *(E.)* Toth, B. (U. Nebraska Coll. Med., Omaha). *J Natl Cancer Inst* 50(1):181-194, 1973.

Administration of 0.01% unsymmetrical 1,1-dimethylhydrazine in the drinking water of 5-week-old randombred Swiss mice throughout their lifetime induced tumors of blood vessels, lung, kidney, and liver. The incidences of tumors in these four tissues were: 79, 71, 10, and 6%, while in the untreated controls they were 2, 11, 0, and 0%, resp. The vascular tumors occurred in the liver, muscle, heart, lung, and fat. Macroscopic, light and electron microscopic, and histochemical examinations of the vascular lesions revealed a morphology typical of angiosarcomas. The gross and histologic involvement of the tissues with the tumors is described and illustrated. The ultrastructure of hemorrhagic areas, angiosarcoma with its components, and the neoplastic endothelial cells and their associated structures and membranes are shown in detail. The findings of this study prove unequivocally the carcinogenicity of this important rocket propellent and provide further evidence for a possible relationship between the chemical structure of hydrazine derivatives and tumor development at specific organ sites.

0041 ULTRASTRUCTURE OF EARLY ALTERATIONS OCCURRING IN THE RAT BRAIN AFTER ADMINISTRATION OF METHYLNITROSOUREA. *(Ger.)* Lageman, A. (Path. Inst. Med. Acad., Erfurt, E. Germany) and W. Dietz. *Experientia* 28(11):1344-1345, 1972.

Wistar rats (130-150 g wt) received 30 mg/kg methylnitrosourea (MNU) i.p. at a 4 wk interval and brain preparations obtained 12 days after the first and 8 and 14 days after the second administration of the carcinogen were subjected to electron microscopy. As opposed to controls all three categories of MNU-treated rat brain preparations revealed pericapillary cell proliferation. Enlarged perivascular spaces were filled with proliferating pericapillary cells, and delimited by internal and external subepithelial membranes. The latter was found to be damaged in brain preparations obtained 8 and 14 days after the second MNU injection, allowing the pericapillary proliferating cells to spread into the brain tissue. Considerable differences between individual cells in terms of cytoplasmic organelle content were observed. Some had very high ribosome content while others had an extended smooth endoplasmic reticulum. The nuclear shape differed from that of the control preparations and the chromatin was either concentrated at the nuclear membrane or distributed throughout the karyoplasm. The above alterations were interpreted as a specific brain perivascular mesenchymal reaction to the carcinogen.

0042 EFFECT OF DILANTIN IN MICE. II. LYMPHORETICULAR TISSUE ATYPIA AND NEOPLASIA AFTER CHRONIC EXPOSURE. *(E.)* Krüger, G. (Natl. Cancer Inst., Bethesda, Md.), D. Harris and E. Sussman. *Z Krebforsch* 78(3):290-302, 1972.

Four hundred and thirty two female mice of strains C_3H/f, $C_{57}Bl$, and SJL/J were divided into nine groups and fed normal mouse pellets, a liquid chocolate flavored diet (Metrecal), and the liquid diet with diphenyl hydantoin (6 mg/100 g body wt/day), respectively, over a period of 16 months. An additional 60 $C_{57}Bl$ mice were sensitized with bovine serum albumin (BSA) and complete Freund's adjuvant while on diphenyl hydantoin and studied for their humoral and cellular immune reactivity by passive hemagglutination microassays and foot-pad tests. Mice of all three strains showed lesions in their lymphoreticular tissues from the second month of the experiment on, varying from partial activation to atrophy. Primarily affected was the paracortical zone of lymph nodes. C_3H/f and $C_{57}Bl$ mice developed thymic lymphosarcomas with a frequency of 3 of 24 mice each. SJL/J mice also showed similar tumors early during the experiment, i.e. 6 of 42 mice during the 4th to the 8th month. After the 8th month SJL/J mice developed up to 90% reticulum cell sarcomas type B, a lesion known to occur in these mice in a high frequency; however, its occurrence was about 2-3 months earlier than usual. Immunologic studies in diphenyl hydantoin fed $C_{57}Bl$ mice revealed variable but not significantly decreased serum anti-BSA titers while the foot-pad tests were constantly negative for a delayed hypersensitivity reaction. It is concluded that diphenyl hydantoin has a supportive effect on both the development of spontaneous lymphoreticular neoplasms and the induction of these neoplasms. It is felt that its action is by interference with the normal immune response rather than by direct carcinogenicity.

0043 SPONTANEOUS AND X-RAY-INDUCED GENOTYPIC AND PHENOTYPIC RESISTANCE TO 5-IODO-2'-DEOXYURIDINE IN LYMPHOMA CELLS *IN VITRO*. *(E.)* Fox, M. (Christie Hosp., Manchester, England). *Mutat Res* 13(4):403-419, 1971.

A study of genotypic and phenotypic resistance to 5-iodo-2'-deoxyuridine (IUdR) in lymphoma cells *in vitro*, either spontaneous or x-ray-induced, is reported. At low doses of IUdR an exponential decline followed by a plateau with further reduction in survival after IUdR doses from $5.6 \times 10^{-5}M$ to $2.8 \times 10^{-4}M$ was seen in the survival curve of plated P_388 cells. X-irradiation of cultures before plating with a range of IUdR concentrations produced an increased frequency of resistant colonies throughout the range of doses of x-radiation and concentrations of IUdR. No heritable resistance to IUdR was found in any of the surviving colonies. A nonlinear increase in the number of x-ray-induced mutations followed x-ray exposure. If the cells were held for 24 hours in normal media prior to plating in IUdR the number of both real and apparently resistant colonies increased. Fluctuation tests at two different dose levels provided evidence that both true mutation to resistance and phenotypic resistance occur at random in the population.

0044 ACCUMULATION OF ^{3}H-ESTRADIOL IN TRANSPLANTED INTERSTITIAL CELL TUMORS IN MICE. *(E.)* Bollengier, W. E. (Yale U. Sch. Med., New Haven, Conn.), A. J. Eisenfeld and W. U. Gardner. *J Natl Cancer Inst* 50(1):195-200, 1973.

The accumulation of estradiol (E2) in three different lines of transplanted testicular interstitial cell tumors (ict) was determined at various times after s.c. administration of ^{3}H-estradiol (^{3}H-E2). These tumors differed in host-hormone dependency. At the extremes, one grew in both male and female hosts and hypophysectomized hosts and one grew well in female hosts, rarely in male hosts, and regressed after hypophysectomy. ^{3}H-E2 accumulated and was retained by all three lines of ict, in the same way as the hormone was retained in the uterus and pituitary gland. This accumulation was highest in the most hormone-dependent and lowest in the most autonomous ict. Autoradiography showed localization over the nuclei. Chromatographic studies revealed 85-95% (usually over 90%) of radioactivity to be estradiol. Pretreatment with large doses of diethylstilbestrol reduced accumulation of ^{3}H-E2 given one hour later.

0045 THE SELECTIVE EXCISION OF ARYLALKYLATED PRODUCTS FROM THE DNA OF *ESCHERICHIA COLI* TREATED WITH THE CARCINOGEN 7-BROMOMETHYLBENZ (a)ANTHRACENE. *(E.)* Venitt, S. (Beatty Res. Inst., London, England) and E. M. Tarmy. *Biochim Biophys Acta* 287:38-51, 1972.

Three strains of *Escherichia coli* B/r WP2, WP2 *uvrA*, WP2 *uvrA exrA*, and WP2 (wild-type) were treated with 7-bromomethylbenz(a)anthracene in doses calculated to prohibit DNA replication but not to affect cell viability. Excision of arylalkylated products from WP2 DNA by carcinogen was studied. The increments of dose needed to reduce survival of WP2 cells to zero were 22, 7.5 and 1 µg/ml, resp., for WP2, WP2 *uvrA* and WP2 *uvrA exrA*. Exposure to carcinogen for two hr did not produce detectable excision of arylalkylated products in WP2 *uvrA exrA*, and produced only 10% excision in WP2 *uvrA*. Up to 70% of arylalkylated products of WP2 were excised by 7-bromomethylbenz(a)anthracene. For WP2, the rate of excision was inversely proportional to the initial extent of the reaction. Chromatographic analysis of arylalkylation products indicated that, though the three main products (extranuclear aminosubstituted N^2-dGuo, N^4-dCyd and N^6-dAdo) were exciseable, N^2 substituted deoxyguanosine was excised more rapidly and to a greater extent than either N^4-substituted deoxycytidine or N^6-substituted deoxyadenosine.

0046 CELL PROLIFERATION AND DRUG RESISTANCE IN THE RESPONSE OF COLONY-FORMING UNITS OF RAT BONE MARROW TO REPEATED DOSES OF CYTOTOXIC AGENTS.

(E.) Dunn, C. D. R. (Inst. Cancer Res., Sutton, Surrey, England). *J Nat Cancer Inst* 48(3):639-649, 1972.

Rats were injected with 30 mg/kg busulfan i.p. 24 hr before receiving 4 mg/kg α-amino-*p*-(di-2-chloroethylamino)-phenyl butyric acid; at 36 hr after busulfan, rats were injected with bone marrow cells and the number of colonies formed by bone marrow in spleen cell cultures of treated rats was observed. Busulfan depressed marrow cell colony formation in spleen. A second dose of busulfan or of 1 mg/kg vinblastine (VB) 24 hr before spleen colony assay reduced the depressive effect of busulfan on colony formation. At the time of maximum suppression of the busulfan effect, VB and the ^{3}H-thymidine suicide technique in bone marrow cells indicated that colony forming units (CFU) of bone marrow were proliferating more rapidly than normal. The resistance of CFU to depression by busulfan after pretreatment with busulfan was also evident for dimethylmyleran (DMM). VB depressed CFU activity in bone marrow to a greater extent in rats previously given DMM than in rats given busulfan. This suggested that cell cycle time (t_c) was altered to different extents after DMM and busulfan; the ratio of period of DNA synthesis (t_s) in bone marrow cells to t_c was also thought to be affected differently by busulfan and DMM. When bone marrow donors were irradiated before treatment with DMM or busulfan, CFU activity of their marrow cells was less affected by busulfan than in unirradiated rats, while the effect of DMM was undiminished by irradiation. This suggested that changes in cell proliferation rate influenced the effects of busulfan or DMM differently, the depression of CFU by DMM being independent of cell proliferation rate, and the effect of busulfan being dependent on cell proliferation rate. An inverse correlation was found between CFU depression by busulfan and CFU ^{3}H-thymidine suicide in bone marrow cells. This effect was apparently not due to the environment of the bone marrow cells; it suggested that cells synthesizing DNA resisted the action of busulfan.

0047 TRACE METALS IN HUMAN OSTEOGENIC SARCOMA.

(E.) Janes, J. M. (Dept. Orthopedics, Mayo Clinic, Minn.), J. T. McCall and L. R. Elveback. *Mayo Clin Proc* 47(7):476-478, 1972.

The content of eight trace metals was determined by atomic absorption spectrometry of acid digests of 40 normal human bone and 81 human osteogenic sarcoma specimens. Compared with normal bone, osteogenic sarcoma showed increased levels of iron, zinc, copper and manganese and decreased levels of calcium, magnesium, cobalt and nickel. The pairs, (Ni, Mn), (Ni, Fe) and (Fe, Mn), were positively correlated with the control but not with the sarcoma group. The pairs, (Co, Cu) (Co, Fe), (Ca, Cu), (Ca, Co), (Ca, Fe), (Mg, Mn), (Mg, Co) and (Mg, Ni), showed a correlation only with the sarcoma group. Sarcomas and normal controls could also be separated with respect to their linear discriminant functions which were based on all eitht metals and which reflected the overall differences between the groups.

0048 THE EFFECTS OF HETEROLOGOUS ANTITHYMOCYTE SERA IN MICE. V. ENHANCEMENT OF PLASMA CELL TUMOR INDUCTION.

(E.) Mandel, M. A. (U. Hosps. Cleveland, O.) and J. J. DeCosse. *J Immunol* 109(2):360-365, 1972.

Experiments were conducted to determine the effect of rabbit antithymocyte serum (ATS) and other modalities known to suppress immune reactivity on induction of plasma cell neoplasms in BALB/c mice given Bayol F. Sixty percent of young BALB/c mice given three i.p. injections of 0.5 ml Bayol developed malignant plasmacytomas after 8-10 months. When mice were given four i.p. doses of 0.5 ml ATS prior to Bayol treatment, tumor incidence was higher and tumors appeared as early as three months after the last Bayol injection. An even higher incidence of tumor induction was seen in mice given a total of 10 ATS injections concomitantly with Bayol injections. Suppression of host reactivity by other modalities, including the chemical agents, Imuran and Cytoxan and by adult thymectomy did not increase the rate of tumor induction. Irradiation of mice at the time of Bayol treatment resulted in a slightly higher incidence of tumors. Mice treated by suppressive therapy, without Bayol, did not develop tumors. Tumor enhancement by ATS and Bayol appeared due to selective destruction of lymphoid cells by ATS. The majority of tumors produced IgA myeloma proteins; about 25% produced IgG myeloma protein. IgM tumors were uncommon.

0049 DOSE-RESPONSE STUDIES WITH CONCURRENT ADMINISTRATION OF PIPERAZINE AND SODIUM NITRITE TO STRAIN A MICE. *(E.)* Greenblatt, M. (U. Nebraska Coll. Med., Omaha) and S. S. Mirvish. *J Natl Cancer Inst* 50(1):119-124, 1973.

Male strain A mice were given 0.69-18.75 g piperazine/kg food and 0.05-2.0 g sodium nitrite ($NaNO_2$)/liter drinking water for 20-25 weeks, and were killed 10-13 weeks later. The yield of lung adenomas was significantly greater than in untreated controls when as little as 0.69 g piperazine/kg plus 1.0 g $NaNO_2$/liter, or 6.25 g piperazine/kg plus 0.25 g $NaNO_2$/liter, were given. A maximum of 30.7 adenomas/mouse more than the controls was observed with 6.25 g piperazine/kg plus 2.0 g $NaNO_2$/liter. Mononitrosopiperazine (MNP) at 34.5 mg/liter drinking water induced 3.8 adenomas/mouse more than the controls. Negative results were obtained with $NaNO_2$ alone, piperazine alone, 12.3 g sodium nitrate ($NaNO_3$/liter alone, or 12.3 g $NaNO_3$/liter plus 18.75 g piperazine/kg. The latter result indicated that nitrate was not reduced to nitrite under conditions suitable for reaction with the piperazine. When the piperazine concentration was raised from 0.69-18.75 g/kg food in 3 threefold increments at a constant level of nitrite, the adenoma yield was increased between successive doses by ratios of 1.4 to 2.5. A ratio of 3.0 was predicted by application of the kinetic equation for MNP formation (adenoma yield was assumed to be proportional to MNP production). When the nitrite concentration was raised from 0.25-2.0 g/liter water in 3 twofold increments at a constant level of piperazine, the adenoma yield was increased by ratios of 3.7 to 3.8, in agreement with the predicted ratio of 4.0.

0050 HEMANGIOMATOUS LESIONS OF MICE TREATED WITH NITROSODIMETHYLAMINE. *(E.)* Otsuka, H. (Sch. Med., Tokushima U., Japan) and A. Kuwahara. *Gann* 62(3):147-156, 1971.

The incidence and distribution of hemangiomatous lesions in mice treated with nitrosodimethylamine were compared in mice receiving the compound p.o. and by s.c. injection. DDD mice, 8- to 10-weeks old, received p.o. administration of 100 ppm nitrosodimethylamine in their diet for 16 to 92 days. BALB/c mice, also 8- to 10-weeks old, received s.c. injection of 0.15 mg weekly for one to 25 weeks. Adenomas and carcinomas in the lung, and benign and malignant hemangiomas, mainly in the liver and soft tissues, but occasionally in the lung, kidney and spleen, were observed. There were also cases of leukemia. The results demonstrated that s.c. administration of nitrosodimethylamine induces hemangiomas and hemangioendothelial sarcomas in considerably higher incidence than does oral administration, not only in the liver, but also in the soft tissues. Soft tissue tumors were most frequent in retroperitoneal areas, especially in pararenal soft tissues. A few animals developed benign and malignant angiomatous lesions in the kidneys, lungs, and spleen. It was concluded that hemangiomatous tumors can develop as a primary growth in the lungs, kidneys, and spleen. since hemangioendothelial sarcomas were seen in these parenchymal organs in a few animals in which there were no angiomatous lesions in other organs.

0051 ENHANCED TUMOR GROWTH IN ANIMALS PRETREATED WITH COMPLETE FREUND'S ADJUVANT. *(E.)* Meltzer, M. S. (Natl. Cancer Inst., Bethesda, Md.) and E. J. Leonard. *J Natl Cancer Inst* 50(1):209-218, 1973.

The ascites form of a diethylnitrosamine-induced hepatoma (line-1), injected intradermally into syngeneic guinea pigs, formed a local tumor which regressed. Regression was associated with development of tumor-specific cellular immunity. Tumor rejection was delayed in animals treated with complete Fruend's adjuvant (CFA) before tumor challenge. Studies on the mechanism of this delayed tumor regression showed: 1) CFA-treated animals had smaller 24-hour-skin reactions to, and prolonged rejection of, a second tumor challenge; 2) the CFA-induced defect was corrected by systemic transfer of peritoneal exudate cells from guinea pigs immunized to the tumor; 3) cutaneous inflammatory response to turpentine and local passive transfer of tumor immunity reactions were intact in CFA-treated animals; 4) the CFA-induced defect was systemic, i.e., it occurred even if CFA treatment was given in a way that spared nodes draining the site of tumor challenge; 5) injection of CFA into animals previously immunized to tumor did not impair tumor rejection; and 6) CFA treatment abrogated induction of tumor immunity in guinea pigs challenged with a mixture of syngeneic line-10 tumor cells and BCG. CFA treatment caused systemic impairment of primary responses to tumor antigen. Effector immune mechanisms, inflammatory responses, and previously existing antigenic or tumor immunity remained intact.

0052 MALIGNANT TRANSFORMATIONS OF C3H MOUSE CELLS *IN VITRO*. DEVELOPMENT OF TRANSPLANTABLE ASCITES TUMORS FROM MOUSE EMBRYO CELLS TREATED WITH 4-NITROQUINOLINE 1-OXIDE. *(E.)* Fujimoto, J. (City of Hope Natl. Med. Ctr., Duarte, Calif.). *J Natl Cancer Inst* 50(1):79-85, 1973.

Seven days after primary culture of C3H/HeJ mouse embryos, cells were treated with 0.0001 mM, 0.001 mM, and 0.01 mM 4-nitroquinoline 1-oxide (4NQO) for 24 hours. These treated cell lines are abbreviated MEC7, MEC6, and MEC5, respectively. Nontreated cells (MECC) were reserved as controls. MEC5 did not grow. The cultured cells were subcutaneously reimplanted periodically into the strain of origin for assay for malignant transformation. After 69 days culture, MEC7, MEC6, and MECC produced subcutaneous nodular tumors at the sites of injection in syngeneic mice. However, tumor latent period *in vivo* of 4NQO-treated cell lines was much longer than that of the untreated cells. Thus 4NQO delayed spontaneous malignant transformation *in vitro* of mouse embryo cells. High doses of this drug inhibited cell growth. At 205 days in vitro MEC7 developed into ascites tumors in 10 of 10 mice, though reinjected subcutaneously into the strain of origin. There was no tumor mass at the site of injection. Some of these ascites tumors as well as subcutaneous solid tumors were transplantable. One ascites tumor has gone through 40 passages. For etiology of these ascites tumors, 4NQO cannot be ignored, though spontaneous malignant transformation may have been a factor.

0053 RELATIONSHIPS BETWEEN COVALENT BINDING OF 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE TO LIVER PROTEIN AND INCREASES IN THE LEVEL OF HEPATIC AMP DEAMINASE.

(E.) Smith, L. D. (Biomed. Div., Sammuel Roberts Noble Fdn., Inc., Ardmore, Okla.), L. Holman and D. E. Kizer. *Chem Biol Interact* 4(5):311-320, 1972.

Experiments were performed to determine whether changes in liver AMP deaminase in rats given 3'-methyl-4-dimethylaminoazobenzene (3'-ME-DAB) were associated with direct interactions between 3'-ME-DAB or its metabolites and AMP deaminase. Female rats were given 3'-ME-DAB in diet (0.06%) or by intraabdominal injection (250 mg/kg body wt). Treated rats were killed and the extent of dye binding to the 105,000 g supernatant fraction of rat liver proteins was determined. 3'-ME-DAB metabolites were bound both to supernatant proteins and to proteins present in fractions of liver subjected to a five-step procedure for purification of AMP deaminase. Some azo proteins were apparently lost during purification of the enzyme. In subsequent experiments rats were injected with 3'-ME-DAB-ring ^{14}C and killed five days later. The 105,000 g liver supernatant fraction was subjected to sucrose gradient centrifugation. Gradients were analyzed for AMP deaminase and for radioactive bound dye. Most radioactivity was associated with proteins which had lower molecular wts than AMP deaminase. Some of the 105,000 g supernatant from rats injected with 3'-ME-DAB was put through the five-step AMP deaminase purification procedure and reacted with rabbit antisera specific for AMP deaminase. Most 3'-ME-DAB-ring ^{14}C-protein conjugates were readily distinguishable from AMP deaminase proteins. Disc gel electrophoresis of purified supernatant from rats injected with 3'-ME-DAB-ring ^{14}C confirmed that AMP deaminase and the azo proteins were different. The increase in AMP deaminase in precancerous rat liver was evidently not caused by a direct interaction of 3'-ME-DAB and liver AMP deaminase.

0054 A CORRELATION BETWEEN THE CARCINOGENICITY OF ISOMERIC *N*-HYDROXY-*N*-ACETYLAMINOFLUORENES AND THEIR *IN VITRO* EFFECT ON MITOCHONDRIA.

(E.) Hadler, H. I. (Dept. Biochem., Southern Illinois U., Carbondale) and B. G. Daniel. *Cancer Res* 33(1):117-122, 1973.

Four aromatic-substituted isomeric *N*-acetylaminofluorenes and their *N*-hydroxy derivatives were examined in an *in vitro* mitochondrial system which previously had been used to implicate an experimental confluence between oxidative phosphorylation and carcinogenesis. The four parent *N*-acetylaminofluorenes did not induce an adenosine triphosphate-energized mitochondrial volume change when combined with showdomycin. The adenosine triphosphate-energized mitochondrial volume changes induced by the hydroxamic acids plus showdomycin fell into two categories. Isomers 2 and 3 (i.e., *N*-hydroxy-*N*-acetyl-2-aminofluorene and *N*-hydroxy-*N*-acetyl-3-aminofluorene) induced marked effects. Isomers 1 and 4 (i.e., *N*-hydroxy-*N*-acetyl-1-aminofluorene and *N*-hydroxy-*N*-acetyl-4-aminofluorene) induced modest effects. Isomer 1 was more effective than Isomer 4. This sequence (2,3 > 1,4 and 1 > 4) is in agreement with the carcinogenicity of the four hydroxamic acids when tested in the rat.

0055 STIMULATION OF THE PHOSPHORYLATION OF MOUSE EPIDERMAL HISTONES BY TUMOR-PROMOTING AGENTS.

(E.) Raineri, R. (U. Wisconsin Med. Ctr., Madison), R. C. Simsiman and R. K. Boutwell. *Cancer Res* 33(1):134-139, 1973.

A single topical application of 0.017 µmole of the potent tumor promoter, 12-O-tetradecanoyl-phorbol-13-acetate to female Charles River CD1 strain mice (7 to 9 weeks old) resulted in a stimulation of the phosphorylation of mouse epidermal histones with an early peak at 2 hr followed by peaks between 1 and 3 days after treatment. The effect of phorbol and two phorbol esters on histone phosphorylation was found to be related to their tumor-promoting activity as well as to their capacity to stimulate RNA and DNA synthesis. Stimulation of histone phosphorylation was slight following treatment with the weak promoter, phorbol-12,13-dibenzoate, and nonexistent after treatment with phorbol, which has no tumor-promoting activity. A dose dependency relationship was established linking the ability of 12-O-tetradecanoyl-phorbol-13-acetate to stimulate histone phosphorylation with its ability to promote tumors. Cycloheximide prevented the increase in histone phosphorylation caused by 12-O-tetradecanoyl-phorbol-13-acetate but had no effect on histone phosphorylation in control mice.

0056 TUMORS OF THE NERVOUS SYSTEM IN MICE TREATED NEONATALLY WITH N-ETHYL-N-NITROSOUREA.

(E.) Searle, C. E. (Dept. Path. U. Birmingham, England) and E. L. Jones. *Nature* 240(5383):559-560, 1972.

The tumorigenic ability of N-ethyl-N-nitrosourea (ENU) was studied in newborn A, C57BL, IF and DBA/Bcr mice. Mice were injected s.c. with ENU (10-160 mg/kg body wt) in saline. All tumors which subsequently developed were examined histologically. Nervous system tumors were found in five IF and five DBA mice. None developed in the A or C57BL strains which appeared to be resistant to ENU tumorigenesis. Four of the tumors in the IF and DBA strains were located in the brain (cerebellum) of male mice which had received 80 mg/kg ENU, contrasting sharply with results seen in ENU-treated rats. Three of these tumors were found in IF mice and were classified as medulloblastomas. The cerebellar tumor in the DBA mouse was an oligoastrocytoma. The remaining tumors were found in the cranial, sciatic, brachial and spinal nerves and were predominantly schwannomas. The tumors arising in the cerebellum did so between 20 and 28 wk after ENU injection. The peripheral tumors all arose after a long latent period (≥40 days).

057 PROPHYLAXIS OF CHEMICALLY INDUCED PAPILLOMAS AND CARCINOMAS OF MOUSE SKIN BY VITAMIN A-ACID. *(E.)* Bollag, W. (F. Hoffmann-La Roche Co., Ltd., Basel, Switzerland). *Experientia* 28(10):1219-1220, 1972.

Studies were conducted on random-bred Swiss albino mice to determine whether or not retinoic acid possesses a prophylactic effect on papilloma and carcinoma induction following weekly cutaneous application of dimethylbenz(a)anthracene (DMBA) and croton oil. Biweekly gastric intubation of retinoic acid during the promotion phase of carcinogenesis caused a decrease both in the mean number of papillomas per animal and in the mean papilloma volume per animal. Whereas both values progressively increased with time in the control animals, the retinoic acid treated mice exhibited a gradual decrease in both mean papilloma volume and papilloma number. Retinoic acid treatment delayed the onset and resulted in a decreased incidence of carcinomas. Whereas only 16% of the surviving control animals showed no evidence of papillomas and carcinomas 288 days after first treatment with DMBA, 50% of the retinoic acid treated survivors were tumor free.

0058 GROWTH OF CARCINOMAS IN THE REGION OF THE CARTILAGE. *(E.)* Dontenwill, W. (Res. Inst. Cigarette Industry, Hamburg, Germany), H. J. Chevalier and G. Reckzeh. *J Natl Cancer Inst* 50(1): 291-293, 1973.

Carcinomas were induced in CFLP mice after application of an acetone solution of benzo[a]pyrene to the skin of the external auricle. Histologic preparations were examined of all tumors and the surrounding cartilage for study of the pattern of tumor growth in connection with the cartilage. Of 100 animals, 81 developed carcinomas, up to walnut size, enveloping the cartilage but never infiltrating the bridges of the cartilage of the ear. The cartilage had a high resistance for carcinoma infiltration. The findings show that the lack of carcinoma infiltration into the cartilage may not be assessed as argument against the presence of a malignant tumor.

0059 SUGAR CONTENT OF THE TOBACCO AND *p*H OF THE SMOKE IN RELATION TO LUNG CANCER RISKS OF CIGARETTE SMOKING. *(E.)* Elson, L. A. (Inst. Cancer Res. Sutton, England) and T. E. Betts. *J Nat Cancer Inst* 48(6):1885-1890, 1972.

More than 150 brands of cigarettes from various countries were examined in a study of the relation between sugar content of cigarette tobacco and pH of smoke. Cigarettes made from flue-cured, high sugar tobacco produced a smoke of acid pH that became progressively more acid during smoking. Cigarettes made from air-cured, low sugar tobacco produced a less acid smoke that became progressively more alkaline during smoking. The lower incidence of lung cancer in smokers of air-cured tobacco cigarettes may relate to the fact that nicotine is more readily absorbed at alkaline than at acid pH. To obtain the same degree of "nicotine satisfaction" as in smoking low sugar cigarettes, the smoker of acid smoke cigarettes must inhale deeply at a time when the tar content of the smoke is highest. The smoking public is unlikely to accept air-cured, low sugar cigarettes, but flue-cured cigarettes could be made safer by raising the pH of the smoke through additives such as urea and guanidine salts.

0060 THE EFFECTS OF CHEMICAL IRRITANTS AND TOBACCO SMOKE CONDENSATE ON THE CHORIOALLANTOIC MEMBRANE OF THE FERTILE HEN'S EGG. *(E.)* Comber, R. (British-American Tobacco Co., Ltd., Carshalton, Surrey, England) and P. Grasso. *Chem Biol Interact* 6(1):25-34, 1973.

Sequential histological studies have shown that various chemical substances, including tobacco smoke condensate, croton oil, formaldehyde, phenol, and acetic acid, produce a dose-related hyperplastic response following application to the chorioallantoic membrane (CAM) of 10-day incubated fertile hen's eggs. In all cases, the initial change was hemorrhage of the mesodermal layer 6 hr after treatment. This was followed at 16 hr by ectodermal necrosis, and by hyperplasia of all three layers in the adjacent area which was maximum at 72 hr as measured by micrometer. Application of 100 µg smoke condensate in 1 µl glycerol produced the same type of response as did 5 µg of croton oil in 1 µl of glycerol. The hyperplastic response from dark air-cured tobacco smoke condensate, including cigar tobacco in cigarettes, was lower than that from flue-cured tobacco. Carrageenan, hypertonic saline, colloidal silica, and glycerol alone produced no CAM hyperplasia when assayed 72 hr after treatment.

0061 CELL PROLIFERATION PATTERNS IN AN AUTOGENOUS RAT SARCOMA. *(E.)* Post, J. (New York U. Sch. Med., New York), R. J. Sklarew and J. Hoffman. *J Natl Cancer Inst* 50(2):403-414, 1973.

Male Wistar rats, 3 wk old, were given single s.c. injections of 30 mg methylcholanthrene in 1 ml corn oil in the interscapular area. The cell proliferation pattern of the methylcholanthrene-induced autogenous rat sarcomas was investigated by continuous labeling with titrated thymidine (^{3}H-TDR), double-labeling with ^{3}H-TDR and ^{14}C-TDR, and by following the serial decay of the grain count frequency distribution after pulse labeling with ^{3}H-TDR. The data showed that only 40-60% of the initial S cohort completed mitosis and entered the succeeding cycle. About 20-35% of the S cohort left the cycle for long residence in G2, and 20-40% of the residual S cohort, after mitosis, remained in G1 for long intervals. Cells subsequently re-entered the cycle from both nonproliferative compartments. The growth fraction was 39%, and the cells in $G2_0$ and in $G1_0$ were 38% and 23% of the population, resp. The transit times of two-thirds of the $G1_0$ cells were greater than 13 days, and for two-thirds of the cells in $G2_0$ the transit times exceeded 1 wk. The continual turnover of the proliferative and nonproliferative compartments was a major determinant of tumor growth.

0062 ONCOGENIC WATER-SOLUBLE POLYCATIONS. I. INDUCTION OF SARCOMAS IN MICE BY DIETHYLAMINOETHYL-DEXTRAN. *(E.)* Rice, J. M. (Natl. Cancer Inst., Bethesda, Md.), J. K. Davidson, R. M. Madison, E. W. Kingsbury and W. Turner. *J Natl Cancer Inst* 50(2):387-401, 1973.

Multiple s.c. or i.p. injections of diethylaminoethyl-dextran (DEAE-D), alone or mixed with polyinosinic-polycytidylic acid, caused development of soft-tissue sarcomas in female NIH General Purpose Swiss-Webster mice at or near the site of injection, approximately 30 wk after the beginning of treatment. The incidence of sarcomas was increased by a single i.p. injection of 1 μmole 1-ethyl-1-nitrosourea (ENU)/g body --a treatment which alone rarely causes s.c. tumors. Dextran sulfate, given by repeated s.c. injections, was less effective than DEAE-D, and dextran was completely inactive. The incidence of other types of neoplasia induced by ENU (pulmonary adenomas, mammary carcinomas, and malignant lymphomas) was not significantly affected by repeated injections of any dextran derivative. Several animals with soft-tissue sarcomas also developed malignant lymphomas. Tissue cultures derived from a number of sarcomas contained no virus detectable by electron microscopy or the XC test, but typical C-type oncornavirus in these cultures was often readily inducible by 5-iodo-2'-deoxyuridine. The possible role of endogenous oncornaviruses in the sarcomagenic action of DEAE-D, which is known to enhance infectivity of such viruses *in vitro* and *in vivo*, is discussed. The proposed use of DEAE-D as an adjuvant for injectable vaccines appears questionable because of its oncogenic properties.

0063 INDUCTION OF BLADDER CANCER IN HAMSTERS BY REPEATED INTRATRACHEAL ADMINISTRATIONS OF 2-ACETYLAMINOFLUORENE. *(E.)* Oyasu, R. (Northwestern U. Med. Sch., Chicago, Ill.), T. Kitajima, M. L. Hopp and H. Sumie. *J Natl Cancer Inst* 50(2):503-506, 1973.

To determine if bladder tumors are induced by a known bladder carcinogen repeatedly administered intracheally, a saline suspension of 2-acetylaminofluorene (AAF) was instilled intratracheally into young male and female golden hamsters once or twice a wk for 17 months. The total amount of AAF administered to experimental animals was 340 mg by the end of eight months and 1100 mg by the end of 17 months. Of the 48 hamsters surviving eight months of treatment, 18 developed bladder tumors. All the tumors were either stage 0 or 1 transitional cell carcinomas with or without focal squamous cell carcinomas. The earliest carcinoma *in situ* (stage 0) was observed at 13 months and 3 wk, and the earliest invasive carcinoma (stage 1) at 13 months and 2 wk.

0064 CHONDRAL DYSPLASIA INDUCED BY ZIRCONIUM AND HAFNIUM. *(E.)* Shelley, W. B. (U. Pennsylvania Sch. Med., Philadelphia). *Cancer Res* 33(2):287-292, 1973.

Eighty female white ICR mice, 12 weeks of age, were given a single intradermal injection of one of a variety of metallic compounds. The salts used were aluminum chloride (hexahydrate), beryllium sulfate (tetrahydrate), cadmium acetate (dihydrate), chromium potassium sulfate (dodecahydrate), cobalt chloride (hexahydrate), hafnium oxychloride (octahydrate), nickel chloride (hexahydrate), and zirconyl chloride (octahydrate). Dissolved in a 0.003 M concentration in an aqueous 0.9% sodium chloride solution, each metallic salt was injected (0.02 ml) into the dorsa of the ear lobes of 10 mice that had been lightly anesthetized with ether. Two months later the animals were sacrificed, and stained serial sections of the ears were analyzed. A similar study of 6 of the metallic salts was done on sixty 16-week-old female CBA/J agouti mice. Five months later these mice were also sacrificed for gross autopsy observation and for a serial microscopic study of the ear injection sites. All 20 ICR and CBA/J mice that received zirconyl chloride injections showed progressively enlarging papules at the injection sites in the ear. Histologically, each mouse showed significant ecchondromas, i.e., outgrowths from the cartilaginous plate. The growths were present at both 2 and 5 months, but were not found at 2 weeks in a special group of 10 CBA/J mice given injections of zirconyl chloride. All of these chondrocytic masses appeared to arise from the upper surface of the plate corresponding to the side of the ear that had been treated by injection. The excrescences were of various forms and sizes, presenting as either small hyperplastic mounds, wedge-like thickenings 4 to 6 times the width of the plate, irregular solid dysplasias, or exuberant serpentine and tongue-like extensions through the dermis. The new chondrocytes, appearing to arise in thickened perichondrium, were embedded in matrix which was well vascularized. Enchondromas were not seen. The same findings were seen in the external ears of each of the 20 ICR and CBA/J mice that was given a single injection of hafnium oxychloride. The histological patterns were virtually identical with those seen following the intradermal injection of zirconyl chloride. The remaining 100 mice, each of which was given a single injection of soluble salts of aluminum, beryllium, cadmium, chromium, cobalt, and nickel showed no abnormal gross or histological changes in either the 2- or 5-month series.

0065 FUNCTION OF BONE MARROW IN SUPPRESSING THYMIC LYMPHOMAS INDUCED BY 7,12-DIMETHYLBENZ[a]ANTHRACENE. *(E.)* Ottonen, P. O. (Cancer Res. Lab., U. Western Ontario, London, Canada) and J. K. Ball. *J Natl Cancer Inst* 50(2):491-496, 1973.

The neonatal injection of 7,12-dimethylbenz[a]-anthracene (DMBA) into the low leukemic strain of CFW mice resulted in a 100% incidence of thymic lymphomas. Injection of normal, syngeneic bone marrow cells 1-21 days post DMBA treatment markedly reduced the tumor incidence but did not affect the mean latent period. At 30 days post bone marrow injection there was evidence of extensive repopulation (approximately 80%) of both thymus and bone marrow in DMBA-treated animals given normal bone marrow 5-21 days after DMBA. When normal bone marrow was given 1 or 35 days after DMBA treatment, the number of donor-derived cells at 30 days post bone marrow injection was less extensive (approximately 50%) in both bone marrow and thymus. Therefore, the establishment of a successful chimera depended on the interval between DMBA treatment and bone marrow injection. At tumor development (90 or more days post DMBA treatment) the cells of both thymus and bone marrow of mice given bone marrow 5-35 days post DMBA treatment (87-176 days post bone marrow injection) but showing no visible signs of thymic lymphoma were still predominantly of donor origin (approximately 90%). However, at 100-150 days post bone marrow injection, no donor cells were found in the thymus and bone marrow of mice given normal bone marrow 1 day post DMBA injection. Direct cytologic examination of the induced thymic lymphomas showed that all were derived from host cells, even though in the same mice (provided the tumor had not disseminated) the bone marrow was still donor in origin. The exception to this occurred in mice given normal bone marrow 1 day post DMBA treatment. In these mice the dividing cells in the bone marrow of tumor-bearing mice were all host in origin, even through the tumors were confined to the thymus. Since the injected bone marrow resulted in a 50% reduction in the tumor incidence, it appears that the ability of normal bone marrow to reduce the incidence of thymic lymphomas is not related to its ability to establish a permanent chimera.

0066 RAT COLON CARCINOMAS ASSOCIATED WITH AFLATOXIN AND MARGINAL VITAMIN A. *(E.)* Newberne, P. M. (Dept. Nutrition Food Sci., Massachusetts Inst. Technology, Cambridge) and A. E. Rogers. *J Natl Cancer Inst* 50(2):439-444, 1973.

Rats of the Charles River CD and the Fischer strains were exposed to aflatoxin B_1 through a low concentration (0.1 or 0.015 ppm) added to their diet or by gastric intubation (400 or 375 µg in 10 or 15 daily doses, resp.). The diet was semisynthetic with refined sources of carbohydrate; some groups of animals were given deficient or marginal levels of vitamin A. Liver carcinomas were induced in a frequency expected from past experience with aflatoxin, without significant effect of the dietary concentration of vitamin A. On the other hand, 11 of 260 rats exposed to aflatoxin developed colon carcinomas, most of which were also associated with low dietary and hepatic concentrations of vitamin A.

0067 HAMSTER RESPIRATORY CARCINOGENESIS INDUCED BY BENZO[a]PYRENE AND DIFFERENT DOSE LEVELS OF FERRIC OXIDE. *(E.)* Sellakumar, A. R. (Eppley Inst. Cancer Res., Omaha, Neb.), R. Montesano, U. Saffiotti and D. G. Kaufman. *J Natl Cancer Inst* 50(2):507-510, 1973.

Dose-response relationships for administration of suspensions of different dosages of ferric oxide and a constant quantity of benzo[a]pyrene (BP) given intratracheally to Syrian golden hamsters were examined in a model system for respiratory carcinogenesis. Each group received 10 instillations at 2-week intervals. The amounts of ferric oxide were 3, 6, and 9 mg per dose, all with 3 mg of BP. As previously noted in this animal model, squamous cell carcinomas were the predominant morphologic type of induced tumors, and the bronchi were the most frequently affected. Essentially identical results were observed for the three experimental groups. Doses of ferric oxide beyond a 1:1 ratio with BP did not enhance tumor yield. These results demonstrated that beyond a certain critical ratio of BP to ferric oxide, the proportion and quantity of ferric oxide administered were unrelated to the incidence of induced tumors.

0068 LACK OF CORRELATION BETWEEN GROSS CHROMOSOME ABNORMALITIES AND CARCINOGENESIS WITH 7,12-DIMETHYLBENZ[a]ANTHRACENE. *(E.)* Ottonen, P. O. (Cancer Res. Lab., U. Western Ontario, London, Canada) and J. K. Ball. *J Natl Cancer Inst* 50(2):497-501, 1973.

The effect of a highly carcinogenic dose of 7,12-dimethylbenz[a]anthracene (DMBA) on the induction of microscopically visible chromosome abnormalities during the preleukemic period and in thymic lymphomas was investigated. A dose of DMBA (30 µg) sufficient to induce thymic lymphomas in 100% of inbred CFW/D mice produced chromosome abnormalities in bone marrow and thymus which did not differ significantly in number and type from those in the cells of the control tissues. Increasing the dose to 60 μg did not cause a detectable increase in chromosome abnormalities. Studies of 37 thymic lymphomas induced by 30 μg DMBA indicated that of the early arising tumors (the first 50%), 61% had normal-appearing chromosome karyotypes. Late-arising tumors had a higher incidence of abnormal chromosome complements, primarily involving the addition of one to three extra chromosomes. It was also possible to cytologically identify the sex of 36 of the 37 tumors on the basis of whether or not they contained the unpaired male (Y) chromosome.

0069 FREQUENCY OF FISH TUMORS FOUND IN A POLLUTED WATERSHED AS COMPARED TO NONPOLLUTED CANADIAN WATERS. *(E.)* Brown, E. R. (Chicago Med. Sch., Ill.), J. J. Hazdra, L. Keith, I. Greenspan and J. B. G. Kwapinski. *Cancer Res* 33(2):189-198, 1973.

Data are presented on the incidence of tumors found in 2121 fish examined from the Fox River watershed (4.38%) as compared to 4639 fish examined in Canada (1.03%). The water systems were compared as to pollution potentials, and the Fox River was found to be a highly polluted system. Among the pollutants found were mercury, lead, arsenic, toluene, crude oil, gasoline, benzanthracene, chlorinated hydrocarbons, phosphates, sulfates, and coliform bacteria. Factors such as dissolved oxygen content, temperature, and nutritional variation were considered similar in both water systems. It is concluded that these pollutants were responsible for the greater frequency of tumors. No attempts were made to determine the effects of pollution on the long term human users of the water or to determine the virus contents of the water system under study. Plans are now being made to study the waters for fish viruses and, incidently, those enteric viruses that one might expect in a river system that is termed "a floating sewer."

0070 MUTAGENIC PROPERTIES OF BENZO(*a*)PYRENE AND ITS METHYLATED DERIVATIVES IN RELATION TO THE MOLECULAR MECHANISMS OF HYDROCARBON CARCINOGENESIS. *(E.)* Fahmy, O. G. (Royal Cancer Hosp., London, England) and M. J. Fahmy. *Cancer Res* 33(2):302-309, 1973.

The carcinogen benzo(*a*)pyrene is mutagenic in *Drosophila*, although this activity appeared to require crucial conditions for the metabolic activation of the compound. The genetic properties of the hydrocarbon were substantially altered as a consequence of various methyl or methylol substitutions on its metabolically significant molecular sites: positions 1, 3, and 6. The 3-methyl and 6-hydroxymethyl derivatives were more mutagenic than the parent hydrocarbon, and dimethyl substitution caused dramatic changes in this activity, the 3,6-compound being most effective; whereas its 1,6-analog was virtually inactive. The mutagenicity of this chemical series also depended on the organization and physiological state of the biological targets, both at the cellular and genic levels. Variations occurred between derivatives as regards the patterns of yield of different mutational classes in the successive germ cell stages during spermatogenesis. Differences were also discernible in the selective mutagenicities of some compounds for the ribosomal RNA genes, as indicated by the *bobbed/X* chromosome mutability ratios. On the whole, however, these ratios were much lower than for other aromatic carcinogens but were of the same order as those associated with alkylation carcinogenesis. The genetic and carcinogenic activities of the examined benzo*(a)*-pyrenes were compared, and both phenomena were considered in relation to the molecular mechanisms proposed for their chemical reactivation. Evidence was adduced in favor of the suggestion that these compounds were metabolically converted into complex ionic species with multiple reactive centers, which could differentially attack the cellular macromolecules. The carcinogenically significant events appeared to involve reactions with DNA analogous to those responsible for alkylation mutagenesis.

0071 ROLE OF HORMONAL ENVIRONMENT, PARTIAL HEPATECTOMY, AND DOSE OF ETHYLNITROSOUREA IN RENAL CARCINOGENESIS. *(E.)* Vesselinovitch, S. D. (Pritzker Sch. Med., U. Chicago, Ill.), L. Itze, N. Mihailovich, K. V. N. Rao and B. Manojlovski. *Cancer Res* 33(2):339-341, 1973.

For evaluation of factors modifying renal carcinogenesis, several groups of 6-week-old C57BL X C3H F_1 mice of both sexes were exposed once to 60 or 120 μg of ethylnitrosourea (ENU) per g of body wt. This treatment was preceded in some groups by partial hepatectomy and/or ovariectomy. Animals were killed at 90 wk of age, and the incidence of renal tumors was evaluated. Ethylnitrosourea induced papillary cystadenomas and solid granular and clear cell-type tumors. Animals treated with 60 μg ENU/g body wt developed kidney tumors in 7.7% of male and 3.7% of female mice. A higher dose of ENU (120 μg/g body wt) increased tumor incidence to 28.0% and 9.8% of males and females, resp. Only the combination of partial hepatectomy and bilateral ovariectomy significantly augmented development of kidney tumors.

0072 TRANSPLANTABILITY AND BIOLOGICAL BEHAVIOR OF MOUSE LIVER TUMORS INDUCED BY ETHYLNITROSOUREA. *(E.)* Kyriazis, A. P. (Pritzker Sch. Med., U. Chicago, Ill.) and S. D. Vesselinovitch. *Cancer Res* 33(2):332-338, 1973.

A study was made of the transplantability, morphology, and biological behavior of hepatomas induced by ethylnitrosourea in C57BL X C3H F_1 newborn and adult mice. Eight tumors induced in newborn and five in adult mice were transplanted to isogeneic hosts, and their behavior was followed for a period of 1 yr. Grafts from the neonatally induced hepatomas grew in 75% of the recipients and were serially transplanted for 22 generations. Transplantation time was reduced from the original 8.4 wk to 2 wk. These transplants grew expansively, invaded locally, and metastasized to regional and distant lymph nodes and the lungs, killing the recipients within 6 wk. Hepatomas that originated in mice treated with ethylnitrosourea as adults grew in 30% of the recipients, but growth was slower. The original transplantation time of 18.0 wk was reduced to 8.1 wk during the observed five transplant generations. No metastases were observed thus far in this series.

0073 SECRETORY AND MITOTIC RESPONSE OF THE MALE RAT PITUITARY GLAND TO REPEATED DOSES OF OESTROGEN. *(E.)* Lloyd, H. M. (Dept. Med., U. Queensland, Brisbane Australia), J. D. Meares and J. Jacobi. *Int J Cancer* 11(1):90-94, 1973.

Pituitary wt, pituitary mitotic index and pituitary and serum concentrations of growth hormone and prolactin were measured at 14-day intervals in male rats given 10 mg diethylstilbestrol dipropionate s.c. every 14 days for a total period of 70 days. Mean pituitary weight reached a maximum of 21 mg on day 42 and no pituitary gland became a tumor according to a criterion of wt greater than 30 mg. Mitotic index was greatly increased until day 70 when it diminished significantly. Pituitary concentration of growth hormone decreased to maintain a low constant level from day 28 on while pituitary prolactin concentration rose to a maximum on day 42. Serum growth hormone was elevated on day 14 and then gradually diminished, while serum prolactin continued to increase until day 42. Pituitary wt and serum prolactin were significantly correlated and pituitary mitotic index was negatively correlated with pituitary growth hormone concentration. The state of apparent equilibrium in mass reached by the pituitary glands from day 42 until day 70, despite greatly increased secretory and mitotic activity, suggests that further disturbance of pituitary function is necessary to bring about net gain in cell numbers and lead to tumor formation.

0074 INHIBITION OF MITOCHONDRIAL 5'-ENDONUCLEASE ACTIVITY BY CARCINOGENIC AMINES AND THEIR N-OXIDIZED DERIVATIVES. *(E.)* Morais, R. (Montreal Cancer Inst., Canada), L. A. Poirier and C. DuPuis. *Chem Biol Interact* 5(6):391-399, 1972.

he carcinogens *N*-hydroxy-2-aminofluorene (N-hydroxy-AF), *N*-benzoyloxy-*N*-methyl-4-aminoazobenzene (*N*-enzoyloxy-MAB) and *N*-acetoxy-*N*-2-acetylaminofluorene *N*-acetoxy-AAF) at concentrations of 0.3, 1.2 and 1.5 mole/mg of mitochondrial proteins resp., inhibited 90% of liver mitochondrial 5'-endonuclease activity in Wistar rats. Under the same experimental conditions, their precursors *N*-methyl-4-aminoazobenzene (MAB), 2-acetylaminofluorene (AAF) and *N*-hydroxy-*N*-2-acetylaminofluorene (*N*-hydroxy-AAF) inhibited the activity to a much lesser extent; 2-aminofluorene (AF) was without effect. The inhibition with *N*-hydroxy-AF, *N*-benzoyloxy-MAB and *N*-acetoxy-AAF was permanent; that exerted by MAB, AAF and *N*-hydroxy-AAF depended on their continued presence in the reaction medium. *N*-acetoxy-AAF readily bound mitochondrial proteins while their precursors bound slightly or not at all. The activity of the alkaline ribonuclease of microsomes and nuclei as well as that of the lysosomal acid ribonuclease was unaffected by the carcinogens. The activity of the supernatant alkaline ribonuclease, however, was stimulated by AF, *N*-hydroxy-AF, *N*-benzoyloxy-MAB and *N*-acetoxy-AAF. This increased activity was not observed in the presence of *p*-chloromercuribenzoate (*p*-CMB), a compound which reacts with the natural protein inhibitor of the supernatant enzyme.

0075 GLUCOCORTICOID-BINDING MACROMOLECULES IN RAT AND MOUSE THYMOCYTES. *(E.)* Kaiser, N. (Dept. Exp. Therap., Roswell Park Memorial Inst., Buffalo, N.Y.), R. J. Milholland and F. Rosen. *J Biol Chem* 248(2):478-483, 1973.

The physicochemical properties of glucocorticoid receptors were studied in rat and mouse thymocytes. The steroid-receptor complex formed when thymocytes were incubated with [1,2,4-^{3}H]triamcinolone acetonide ([^{3}H]TA) was assayed in a 27,000 X *g* supernatant fraction using sucrose density gradient analysis. After incubation of rat thymocytes at 0 C for 30 min most of the bound radioactivity was associated with the 27,000 X *g* supernatant obtained by extracting the cells with a buffer containing a low concentration of salt (<0.001 M); two [^{3}H]TA-receptor complexes which sedimented at $\sim$3.5 S and $\sim$7 S were observed in this fraction. At 0 C the buffer containing high salt (0.15 M KCl) extracted [^{3}H]TA-receptor complex sedimenting at $\sim$4 S. When incubated at 37 C for 30 min, the bulk of bound radioactivity was found in the 27,000 X *g* pellet from which it could be extracted only with high salt buffer to give a [^{3}H]TA-receptor complex which sedimented at $\sim$4 S. Mouse thymocytes differed from rat thymocytes in that following incubation with [^{3}H]TA at 0 C for 30 min and extraction with the low salt buffer, they yielded only one complex sedimenting at $\sim$7 S. Pretreatment *in vivo* with TA increased the *in vitro* binding of the [^{3}H]TA to the 3.5 S receptor at the expense of binding to the 7 S receptor. It is concluded that the qualitative distribution of the glucocorticoid receptors is species specific and is dependent on the incubation temperature and the ionic strength of the buffer used for extraction and sucrose density analysis.

0076 EFFECT OF HUMAN PLACENTAL LACTOGEN ON GROWTH OF CARCINOGEN-INDUCED MAMMARY TUMORS IN RATS. *(E.)* Nagasawa, H. (Natl. Cancer

Ctr. Res. Inst., Tokyo, Japan) and R. Yanai. *Int J Cancer* 11(1):131-137, 1973.

The effect of exogenous administration of human placental lactogen (HPL) on 7,12-dimethylbenz(a)-anthracene (DMBA)-induced mammary tumors was studied in female Sprague-Dawley rats. Twice daily s.c. injections of 1 or 2 mg HPL for 20 days (45-85 days after a single oral dose of 20 mg DMBA) markedly stimulated the appearance and growth of mammary tumors and of normal mammary gland growth not only in intact but also in adreno-ovariectomized rats. These results suggest that placental lactogen plays an important role in the growth of DMBA-induced mammary tumor of rat and that it acts on mammary tumorigenesis during pregnancy both directly and indirectly through its promotion of progesterone secretion by the ovary.

0077 BERYLLIUM-INDUCED ULTRASTRUCTURAL CHANGES IN INTACT AND REGENERATING LIVER.

(E.) Goldblatt, P. J. (U. Pittsburgh Sch. Med., Pa.), M. W. Lieberman and H. Witschi. *Arch Environ Health* 26(1):48-56, 1973.

Four groups of eight rats were partially hepatectomized and immediately following the operation were given an i.v. injection of 15,30, or 60 µmols $BeSO_4$ per kilogram in saline or an equal volume of saline. Two animals from each group were killed at 6, 12, 24, and 48 hours after the operation. In a second experiment, ten rats were partially hepatectomized, and 30 µ mols $BeSO_4$ per kilogram was injected into two animals at 0, 8, 12, and 22 hours after the operation. The two controls received saline at 0 time. All animals in this second group were killed 28 hours following the hepatectomy. That is, two animals were exposed to Be for 28 hours, two for 20, two for 16, and two for 6 hours prior to death. The 28-hour point was selected since in young rats mitoses are generally more frequent at this interval than at 24 hours. Light microscope observations revealed liver necrosis in animals surviving 48 hours, and abolition of mitoses in animals treated 20 or 16 hours before death. Ultrastructural alterations in both intact and partially hepatectomized animals examined 24 hours following Be injection included vacuolization and dense deposits in lysosomes, loss of fibrils and appearance of dense plaques in some nucleoli, and distortion of bile canaliculi. The lysosomal changes and inhibition of mitosis correlated well with previous biochemical studies.

0078 FACTORS AFFECTING DIMETHYLNITROSAMINE FORMATION FROM SIMPLE PRECURSORS BY RAT INTESTINAL BACTERIA.

(E.) Klubes, P. (George Washington U. Sch. Med., Washington, D.C.), I. Cerna, A. D. Rabinowitz and W. R. Jondorf. *Food Cosmet Toxicol* 10(6):756-767, 1972.

$[^{14}C]$Dimethylamine and sodium nitrite incubated together in the molecular proportion 1:4 with the bacterial flora of the rat intestine under anaerobic conditions at pH 7.0 give rise to $[^{14}C]$dimethylnitrosamine, which can be readily detected by thin-layer chromatography and estimated by scintillation counting. Glucose is required for the formation of dimethylnitrosamine from precursors dependent on the metabolic activity of the bacterial flora from the caecum, small intestine or large intestine. By omission of glucose from the system, or by the use of a boiled bacterial preparation, nitrosamine formation is suppressed but not completely abolished. $[^{14}C]$Dimethylnitrosamine formation is also suppressed to a similar extent by inclusion of the antibiotic neomycin as a component of the standard incubation system. There appears to be a residual non-enzymatic formation of $[^{14}C]$dimethylnitrosamine from its precursors under these circumstances, even though the reaction conditions at pH 7.0 are far from optimal for spontaneous interaction of dimethylamine and sodium nitrite. Enzymatic formation of dimethylnitrosamine can be enhanced when standard incubations are performed in the presence of riboflavin. Overall yields of nitrosamine can also be enhanced by increasing the nitrite levels in the system, but this tends predominantly to increase the non-enzymatic nitrosamine formation. Preformed $[^{14}C]$dimethylnitrosamine incubated with caecal bacteria under standard conditions for 20 hr appears to be stable and is recoverable in 95% yield from incubation mixtures.

0079 DEOXYCORTICOSTERONE AND CORTICOSTERONE BIOSYNTHESIS IN FEMALE SPRAGUE-DAWLEY RATS AFTER 7,12-DIMETHYLBENZANTHRACENE ADMINISTRATION.

(Fr.) Joubert, F. (E. U. R. Med., Medical Biochem. Lab., Caen, France) and M. A. Drosdowsky. *C R Soc Biol (Paris)* 165(11):2095-2098, 1971.

Conversion of progesterone into deoxycorticosterone and corticosterone by regenerated adrenal tissues from 7,12-dimethylbenz(a)anthracene-treated rats was studied. Fifty day-old female Sprague-Dawley rats were given intragastrically 20 mg DMBA dissolved in 1 ml of olive oil. Adrenalectomy was performed following regeneration of the adrenal tissue 15 days after DMBA administration. Adrenal portions were then incubated in a Krebs-Ringer bicarbonate solution at a 7.4 pH in the presence of tritiated progesterone and an $NADPH^+$ generating source in an O_2/CO_2 (95/5) atmosphere at 37 C for two hours. Incubation was stopped with ethanol. Adrenal perparations from DMBA-treated rats exhibited a progesterone conversion of 34% into corticosterone and a 1.16% conversion into deoxycorticosterone. Related data in intact rat adrenals amounted to 20.75 and 6.7%, resp., indicating that a shift from deoxycorticosterone towards corticosterone synthesis occurs upon DMBA treatment. Studies attempting to verify these results on larger populations as well as to explore estrogen biosynthesis under similar conditions are in progress.

0080 CHEMICAL INDUCTION OF SUBCUTANEOUS TUMORS IN BALB/c AND SWISS MICE INFECTED WITH

WILD TYPE C RNA VIRUSES DERIVED FROM BALB/c TISSUES. *(E.)* Salerno, R. A. (Microbiol. Associates, Inc., Bethesda, Md.), G. M. Ramm and C. E. Whitmire. *Cancer Res* 33(1):69-77, 1973.

The effect of exogenous type C RNA viruses derived from BALB/c mice on the chemical induction of s.c. tumors in mice was determined in relation to host strain, age at treatment, sex, carcinogen dose, and virus strain. Viruses that were cocarcinogenic in Swiss mice were anticarcinogenic or without effect in BALB/c mice under similar treatment conditions. Co- and anticarcinogenic effects of viruses on tumor induction by 100 μg 3-methylcholanthrene occurred when the chemical was given at newborn but not weanling age. In BALB/c mice, the mean effect of all viral inocula was related to sex and to the dose of 3-methylcholanthrene administered. Virus decreased the induction of tumors by 25 μg 3-methylcholanthrene in males or by 100 μg 3-methylcholanthrene in females; however, virus increased tumor induction by 25 μg 3-methylcholanthrene in females or by 100 μg 3-methylcholanthrene in males. Viruses derived from tumor-bearing BALB/c mice were more co- and anticarcinogenic than viruses derived from nontumorous mice. Mixing viruses isolated from neoplasms in equal proportions with viruses isolated from normal mice averaged the effects of the individual isolates. This study indicated that wild type C RNA virus may significantly influence chemical carcinogenesis; however, the type of modifying effect depends on the mouse strain, sex and age at treatment, carcinogen dose, and virus strain.

0081 ON THE ISOTOPIC $^{13}C/^{12}C$ RATIO STUDIED IN A MALIGNANT TUMOR OF THE RAT. *(Fr.)*

Mosora, F. (Dept. Atomic and Molecular Phys., U. Liège, Belgium), M. Lacroix and J. Duchesne. *C R Acad Sci* [*D*] *(Paris)* 275(15):1701-1702, 1972.

The isotopic $^{13}C/^{12}C$ ratio was compared between healthy muscular and sarcomatous tissues in female rats with methylcholanthrene-induced cancer. Neoplasia was induced in the hind limb by a single s.c. injection of 0.5 mg methylcholanthrene. CO_2 resulting from the combustion of tissue samples (quadriceps muscle) was determined by mass spectrometry according to a procedure referred to elsewhere. Healthy tissues revealed isotopic $^{13}C/^{12}C$ ratios amounting to 8.3 ± 0.2 ppt whereas the sarcomatous tissue samples had a 9.5 ± 0.2 ppt ratio, indicating a ^{13}C enrichment of 1.2 ppt. Since the average tumor size amounted to 15 g the isotope contribution attributed to the administered carcinogen was minimal.

0082 MACROAUTORADIOGRAPHIC AND RADIOMETRIC STUDIES ON THE DISTRIBUTION OF 3-METHYLCHOLANTHRENE IN MICE AND THEIR FETUSES. *(E.)*

Takahashi, G. (Chest Dis. Res. Inst., Kyoto U., Japan) and K. Yasuhira. *Cancer Res* 33(1):23-28, 1973.

Pregnant and nonpregnant mice received injections of 3-methylcholanthrene-^{14}C by several routes and were killed after various intervals. Macroautoradiographs of while-body sections were examined, and the radioactivity in several organs was measured. The distribution of the compound in various tissues and its clearance were elucidated from the data obtained. This carcinogen is metabolized in the liver, secreted in the bile, and excreted in the feces. That some of the compound can be reabsorbed from the intestine is evident from examinations of mice given the chemical by gastric intubation. The kidneys also play a role in metabolizing the carcinogen. The lungs of mice that had been given an i.v. injection showed many capillary emboli composed of crystalline deposits of the carcinogen. A small but definite amount of the carcinogen injected into pregnant mice appeared in the fetuses. In fetuses near term, it seemed to be metabolized in the same manner as in adult mice, since the carcinogen or its metabolites were detected in the liver, in the contents of the intestines, and in the kidneys. No significant difference in specific radioactivity was recognized between tissues that were susceptible and those that were resistant to the carcinogen. This was true in adult as well as fetal animals, and it was established that only a small amount (possibly less than 0.26%) of the carcinogen injected into a pregnant mouse is transferred to the fetus and that only 2 ng of the carcinogen/mg of lung tissue is sufficient to initiate pulmonary tumors in about three-fourths of the offspring.

0083 THE DISTRIBUTION OF TRITIUM-LABELED AFLATOXIN B_1, DIHYDRO-AFLATOXIN B_1, AND TETRAHYDRODESOXO-AFLATOXIN B_1 IN LIVER CELLS CULTURED *IN VITRO* FROM THE CHICKEN EMBRYO. *(E.)*

Terao, M. (Chiba U. Sch. Med., Japan), M. Yamazaki and K. Miyaki. *Z Krebsforsch* 78:303-309, 1972.

The distribution patterns of ^3H-aflatoxin B_1, B_2 and tetrahydrodesoxoaflatoxin B_1 ($4HB_1$) in the cultured liver cells originated from the 12-day-old chicken embryo were demonstrated. The fact that the hepatocytes were more heavily labeled than the mesenchymal cells by the incorporation of ^3H-aflatoxins correlate well with the observation that the hepatocytes were more susceptible than the mesenchymal cells to the aflatoxins. There were no significant differences in the pattern of incorporation of ^3H-aflatoxin B_1, B_2 and $4HB_1$ into the hepatocyte, but the three mycotoxins show a wide variation in their inhibitory action in RNA synthesis. It was concluded that the toxicity of aflatoxins to the liver cells *in vitro* is closely related to the membrane permeability and the active portion of the molecule of aflatoxins, such as the double bond in the difuran structure and the carbonyl group in the cyclopentenone portion of B_1.

0084 STRUCTURE-ACTIVITY RELATIONSHIPS IN POLYCYCLIC AROMATIC HYDROCARBONS: INDUCTION OF MICROSOMAL ARYL HYDROCARBON HYDROXYLASE

AND ITS POSSIBLE IMPORTANCE IN CHEMICAL CARCINOGENESIS. *(E.)* Franke, R. (Germany Acad. Sci., Berlin, West Germany). *Chem Biol Interact* 6(1):1-17, 1973. *Biol Interact* 6(1):1-17, 1973.

The quantitative structure-activity relationships for microsomal arylhydrocarbon hydroxylase (AHH) induction by polycyclic hydrocarbons have been analyzed. Multiple regression analyses based on data obtained from the literature indicated that, for a polycyclic hydrocarbon to have good AHH-inducing ability, it must possess a reactive K-region and an inert L-region, and it must be able to participate in hydrophobic as well as charge transfer interactions, the latter being only of secondary importance. These structure-activity relationships necessary for AHH induction are very similar to those necessary for carcinogenic activity. It thus appears that, as with AHH induction, the formation of a reactive K-region metabolite which then reacts with a biological receptor is the mechanism for chemical carcinogenesis in which AHH induction may be the first step.

0085 CARCINOGENIC NITROSAMINES RESULTING FROM SIMPLE ALIPHATIC TERTIARY AMINES AND NITRITE.

(Ger.) Schweinsberg, F. (Hyg. Inst., U. Tübingen, W. Germany) and J. Sander. *Hoppe-Seylers Z Physiol Chem* 353(11):1671-1676, 1972.

Treatment of tertiary amines with sodium nitrite in a citrate/hydrochloric acid buffer at 25 or 100 C yields nitrosamines after dealkylation to secondary amines. The nitrosation of the simple aliphatic tertiary amines trimethyl- and triethylamine produced the best yields at pH 3.3, as with the secondary amines. The amount of diethylnitrosamine found in the nitrosation of triethylamine at 100 C was 200 times smaller than in the reaction with diethylamine. The addition of diethylamine and nitrite to the diet of rats did not produce tumors, as was shown in an earlier publication. The addition of 0.5% triethylamine and 0.5% nitrite to the diet of SIV$_{50}$ rats caused no toxic effects. These experiments were started one year ago; no tumors have been observed so far. The concentrations of amine and nitrite that occur in the environment are normally much smaller than the concentrations used in the animal experiments. Because of the strong dependence of the reaction on the nitrite concentration only minute amounts of carcinogenic nitrosamines will be formed *in vivo* or under conditions corresponding to food preparation. The amounts of nitrosamines generated under conditions of such small nitrite amounts seem to be too low for tumor induction.

0086 CHARACTERIZATION OF LIVER RNA FRACTIONS ALKYLATED BY DIMETHYLNITROSAMINE *IN VIVO*.

(E.) Hennig, W. (Inst. Pharmacol. Toxicol. U. Marburg, W. Germany), W. Kunz, K. Petersen, B. Schnieders and F. W. Krüger. *Z Krebsforsch* 76(3): 167-180, 1971.

The extent of RNA methylation occurring *in vivo* upon dimethylnitrosamine (DMNA) administration was studied in young male NMRI mice receiving an i.p. injection of 25 or 50 mg/kg ^{14}C-DMNA (acq. solution). Liver RNA extraction was performed according to the temperature-dependent phenol extraction procedure at 20 and 65 C 12 hr postinjection. Sucrose density gradient centrifugation and column chromatography were applied for the separation and characterization of the RNA fractions and constituents. Intact mouse livers yielded a 72/28 ratio between the RNA extracted at 20 and 65 C, resp. The RNA fraction, extracted at 20 C prevailed in cytoplasmic whereas the fraction obtained at 65 C contained mainly nuclear RNA. The temperature-dependent RNA ratio was close to the control ratio in extracts from 25 mg/kg ^{14}C-DMNA-treated mouse livers. Livers from mice receiving 50 mg/kg ^{14}C-DMNA yielded an 82/18 RNA ratio indicating a dose-dependent shift of the tightly bound RNA towards the labile RNA. This shift was found to be associated with a decrease in large molecular RNA constituents and an increase in small molecular RNA constituents, according to sucrose gradient centrifugation data. Sedimentation profiles of RNA extracts from 50 mg/kg ^{14}C-DMNA-receiving mouse livers showed OD peaks in the 4s, 4-12s, 12-20s, 20-30s, 35s, and 45s sedimentation zones. The bulk of the ^{14}C label was found in the RNA of the 20 C extract. The RNA extracted at 65 C showed minor label content in the 4-12s and above 30s sedimentation zones. Methylalbumin diatomite column chromatography of the RNA extracts yielded nucleotides and oligonucleotides, t-RNA, 5s RNA, DNA and RNA hybrids, ribosomal RNA and newly formed RNA, referred to as D-RNA and R-RNA. Pulse labeling with ^{3}H-orotic acid revealed two peaks of maximum DMNA-generated label in the region corresponding to the newly formed RNA. Transfer RNA and its physiological methyl group receptors (the 5s fractions) showed only minor label content. Most of the ^{14}C-DMNA-generated label was concentrated in the 7-methylguanine base of the label carrying RNA fractions. It is postulated that alkylation of the RNA chain occurs during the transcription process or during the subsequent phase of conversion into low-molecular functional units; DMNA-generated alkylation of RNA constitutes the basic feature of nitrosamine cytotoxicity. No relationship between nitrosamine cytotoxicity and carcinogenicity could be established as yet.

0087 THE NATURE OF ESTROGEN AND PROLACTIN EFFECT ON MAMMARY TUMORIGENESIS. *(E.)*

Sinha, D. (Dept. Breast Surg., Roswell Park Mem. Inst. Buffalo, N.Y.), D. Cooper and T. L. Dao. *Cancer Res* 33(2):411-414, 1973.

Increased release of pituitary prolactin as a result of placement of electrolytic lesions in the median eminence of the hypothalamus greatly accelerated the growth rate of 7,12-dimethylbenz(a)-anthracene-induced mammary tumors in female Sprague-Dawley rats. Ovariectomy in these rats induced a rapid regression of tumors in spite of high plasma levels of prolactin. Grafting a pair of ovaries into ovariectomized rats with lesions of the median eminence did not alter the levels of plasma prolactin, but it did cause a rapid resumption of tumor growth. These results suggest that the prolactin stimulus to growth of mammary tumor is dependent on the presence of ovarian hormones.

0088 INDUCTION OF ARYL HYDROCARBON HYDROXYLASE IN MOUSE TISSUES FROM A HIGH AND LOW CANCER STRAIN AND THEIR F_1 HYBRIDS. *(E.)*

Bürki, K. (Med. Coll. Georgia, Augusta), A. G. Liebelt and E. Bresnick. *J Natl Cancer Inst* 50(2):369-380, 1973.

mong inbred mice from the high-leukemia strain KR and the low-leukemia strain Af, which in ddition represent extremes of resistance and susceptibility, resp., to polycyclic hydrocarbon-nduced skin tumorigenesis, marked cell- and issue-specific differences were apparent in he capability of aryl hydrocarbon hydroxylase ctivity to be induced in response to 3-methyl-holanthrene (MCA). In Af mice, aryl hydrocarbon ydroxylase was inducible by two-to several-fold n most tissues examined, except possibly some ymphoreticular organs, e.g., spleen. In AKR ice, this enzyme system in liver was relatively esistant to induction by MCA. In contrast, the xtrahepatic hydroxylase activity from AKR mice as slightly induced in tissues such as lung, pidermis, and probably lymph nodes. The F_1 ybrid mice from genetic crosses between these trains resembled Af mice in susceptibility to he effects of MCA as inducer of hydroxylase ctivity in most tissues tested, though not to the ame degree as the parent inbred strain. Further, hese hybrids lack MCA-inducible enzyme in corium utis under the experimental conditions. Thus he regulation of enzyme induction, in the genetic roups examined, seemed not to reflect simple endelian genetics. The control and inducible evels of hepatic hydroxylase activity from nbred and hybrid mice were higher in females han in males. The hepatic enzyme system erived from female mice was more active in etabolizing MCA as substrate and in catalyzing he formation of polycyclic hydrocarbon-hepatic ytosol protein complexes. However, a lack f correlation was noted between metabolism f MCA or binding of polycyclic hydrocarbon to ytosol macromolecules and the ability of epatic hydroxylase activity to be induced n response to MCA.

089 COMPARATIVE STUDIES OF NEOPLASTIC RESPONSE TO A SINGLE DOSE OF NITROSO COMPOUNDS: . THE EFFECT OF N-NITROSOHEXAMETHYLENEIMINE IN YRIAN GOLDEN HAMSTERS AND SWISS MICE. *(E.)*

lthoff, J. (Eppley Inst. Res. Cancer Allied Dis., maha, Nebr.), P. Pour, A. Cardesa and U. Mohr. *Krebsforsch* 78(1):78-81, 1972.

Syrian golden hamsters and Swiss mice (randomly bred, 8 wk old) were treated s.c. with a single dose of nitrosohexamethyleneimine (N-6-MI) dissolved in 0.9% NaCl. Controls were treated with the solvent. Male and female hamsters showed a LD50 of 142 mg/kg b.w. while in mice the acute toxicity (LD50) of N-6-MI was 65 mg/kg b.w. for females and 70 mg/kg b.w. for males. Control hamsters did not show tumors of the respiratory system. Papillary tumors were found in the larynx and multiple papillary neoplasms developed in the trachea. Females dosed under 32 mg/kg b.w. had no tracheal tumors. Mice did not develop neoplasms in the upper respiratory tract. Lung adenomas were found in 10% of the controls (females and males). In the experimentals the incidence averaged 20% for females and 31% for males. A few neoplasms originating in organs other than the respiratory system were found in the controls as well as in experimentals of both animals. It is concluded that a single dose of 4 mg/kg b.w. of N-6-MI is sufficient to induce tumors in the respiratory system of Syrian golden hamsters.

0090 NASAL AND OTHER TUMOURS IN RATS GIVEN 3,4,5-TRIMETHOXYCINNAMALDEHYDE, A DERIVATIVE OF SINAPALDEHYDE AND OF OTHER α,β-UNSATURATED ALDEHYDIC WOOD LIGNIN CONSTITUENTS.

(E.) Schoental, R. (Med. Res. Council Lab., Carshalton, Surrey, England) and S. Gibbard. *Br J Cancer* 26(6):504-505, 1972.

3,4,5-Trimethoxycinnamaldehyde (TMCA) was administered in two doses to six white male rats weighing 75-80 g (150 mg/kg body weight, as a suspension in 20% aqueous ethanol by i.p. injection, followed within one week by a second dose, 100 mg/kg in dimethylformamide injected s.c.). Four rats which survived longer than 17 months after this treatment developed tumors. These consisted of a sarcoma in the peritoneal cavity with metastatic nodules on the omentum in a rat that died after 20 months, a mesothelioma of the tunic albuginea of both testes in a rat killed after 25 months and two nasal squamous carcinomata in two rats killed after 20 and 24 months resp. These results may have some bearing on the high incidence of nasal tumors among wood workers. Though it is not known whether TMCA is present in wood lignins, it may be formed metabolically in the animal body.

0091 ISOGENIC TRANSPLANTATION OF ETHYLNITROSOUREA-INDUCED TUMORS OF THE CENTRAL AND PERIPHERAL NERVOUS SYSTEM IN TWO DIFFERENT INBRED RAT STRAINS. *(E.)*

Wechsler, W. (Max-Planck Inst., Köln, West Germany), M. A. Ramadan and A. Gieseler. *Naturwissenschaften* 59(10):474, 1972.

Transplantable tumors of the central and peripheral nervous system (CNS, PNS) were established in inbred rat strains. Primary tumors were induced transplacentally in the third period of gestation by single injections of 20 to 80 mg ethylnitrosourea (ENU)/kg in BDIX and CD Fisher rats. Fourteen primary tumors of the CNS and 35 tumors of the PNS were used in the study. The tumors were classified as malignant gliomas or neurinomas and were characterized by a heterogenous cell population with various degrees of differentiation and anaplasia. For isogenic transplantation, tumor tissue was innoculated s.c. either as minced tumorbrei or as small pieces (25-250 mg tissue/injection) into male and female rats of different age groups. CNS tumors had a lower rate of acceptance in the first passage than PNS tumors. The rate increased to about 100% after the second passage. Transplanted primary brain tumors were accepted more readily when injected into recipient hosts younger than one month, but age-dependent differences were less marked for PNS tumors. The mean growth rate for accepted CNS tumors in the first passage was generally slower than that for PNS tumors. Transplanted CNS tumors underwent marked sarcomatous changes, usually after the first passage, while PNS tumors maintained their primary morphological characteristics for several passages.

0092 INDUCTION OF BRAIN TUMOURS IN HIGH YIELD BY ADMINISTRATION OF N-ETHYL-N-NITROSOUREA TO NEWBORN RATS.

(E.) Searle, C. E. (U. Birmingham Med. Sch., England), E. L. Jones and W. T. Smith. *Experientia* 28(12):1452-1453, 1972.

Seventeen Wistar-derived albino rats and 21 Lister hooded rats were injected s.c. 24 hr after birth with 10 mg N-ethyl-N-nitrosourea (ENU) per kg body wt. Routine neurohistological examination of the 34 animals that survived to weaning revealed a total of 71 neural tumors, of which 53 (74%) were located in the brain. The preferred site was in the cerebral hemisphere (47 tumors). Histologically, the most common tumors were subependymal-plate gliomas (17) and oligodendrocytomas (10). There were nine tumors in the spinal cord and nine in the peripheral nervous system, of which seven were situated in the cranial nerves and their ganglia. Median tumor induction time was 392 days. The present study suggests that albino and hooded rats are more useful for studies of chemically induced brain tumors than is apparent from the existing literature.

0093 THE FAILURE OF INTERMEDIATES OF DNA SYNTHESIS TO INFLUENCE THE INITIATION BY URETHANE OF SKIN TUMOURS IN MICE.

(E.) Pound, A. W. (Dept. Path., U. Queensland, Brisbane, Australia). *Br J Cancer* 26(6):509-512, 1972.

Groups of 40 mice, random bred males of the strain Hall, 7-8 wk of age and weight 25-26 g were injected s.c. with 20 mg urethane in 0.25 ml saline, and at the same time one of the intermediates of RNA or DNA synthesis, also dissolved or suspended in 0.25 ml saline or (controls) an additional 0.25 ml saline. In Experiments V and VI the animals were given an application of 25% v/v acetic acid in acetone 18 hr before the injection of urethane. From the seventh day later the animals were given a standard promoting treatment with croton oil to the skin of the back once a week for 20 wk. In Experiment VI the promoting treatment was delayed for 12 wk. Skin tumors were counted 22 wk after commencement of the promoting treatment and the number of living adenomata was assessed by counting the characteristic nodules presenting on the surface at autopsy. No significant difference in the yield of skin tumors was found between the mice given urethane alone or those given urethane with any of the intermediates of DNA synthesis. The injection of early intermediates of pyrimidine synthesis did not influence the augmented tumor yield that follows a single treatment of the skin with acetic acid before injection with urethane, nor the rate at which the tumor yield declined when the promoting treatment was delayed. Similarly, none of the intermediates injected with urethane influenced the yield of lung tumors in the mice whether the animals were killed 22 or 34 wk later, even though the number of lung adenomata increased significantly with the longer time. Experiments V and VI show that although the preliminary treatment with the acetic acid increased the tumor yields in the skin, there was no significant alteration in the number of adenomata in the lungs.

0094 THE CARCINOGENICITY OF SOME 6-SUBSTITUTED BENZO(a)PYRENE DERIVATIVES IN MICE.

(E.) Dewhurst, F. (Sch. Biol., City of Leicester Polytechnic, England), D. A. Kitchen and G. Calcutt. *Br J Cancer* 26(6):506-508, 1972.

6-Substituted benzo(a)pyrene derivatives were tested for carcinogenicity in Balb/c mice after s.c. injection. Controls were injected with propylene glycol. No tumors appeared in controls. Tumors found in experimental animals all arose at or around the site of injection and all were fibrosarcomata. The 6-carboxaldehyde, the 6-methyl derivative and 6-hydroxymethyl compound were found to be carcinogenic, the 6-bromo compound was possibly a weak carcinogen while the 6-nitrile and 6-amide derivatives appeared non-carcinogenic. The formation of charge transfer complexes with iodine and acridine was examined. Compounds showing only weak acceptor or no complex forming properties proved non-carcinogenic. The results support the hypothesis that either donor or acceptor properties may be associated with carcinogenicity. Comparison of 6-substituted benzo(a)pyrenes with 7-substituted benz(a)anthracenes showed that carcinogenic activity runs parallel for s.c. injection in the two series.

0095 THE ULTRASTRUCTURE OF DMBA-INDUCED BREAST TUMORS IN SPRAGUE-DAWLEY RATS.

(E.) Murad, T. M. (Dept. Pathol., Ohio State U., Columbus) and E. von Haam. *Acta Cytol (Baltimore)* 16(5):447-453, 1972.

Breast tumors induced in 60 Sprague-Dawley rats by administration of 7,12-dimethylbenz(a)anthracene were studied by electron microscopy, radioautography, and histochemistry. Myoepithelial cells played a key role in the initiation of tumorigenesis and continued to proliferate with increasing size of the tumors. Positive reactions for alkaline phosphatase and adenosine triphosphatase were detected on the peripheral cells of tumor nodules and also on small cell groups within the tumors. The localization of lead precipitate was observed on the plasma membranes of the myoepithelial cells. However, unlike human myoepithelial carcinoma, the reaction for alkaline phosphatase remained strongly positive on the plasma membrane of myoepithelial cells. This observation and the absence of metastasis suggest that the experimental tumors could represent a benign proliferation of myoepithelial and epithelial cells, rather than true carcinomas.

096 ARYL HYDROCARBON HYDROXYLASE, EPOXIDE HYDRASE, AND 7,12-DIMETHYLBENZ(A)ANTHRA-ENE-PRODUCED SKIN TUMORIGENESIS IN THE MOUSE.

E.) Nebert, D. W. (Natl. Inst. Arthritis etabolic Dis., Bethesda, Md.), W. F. Benedict nd J. E. Gielen. *Mol Pharmacol* 8(2):374-379, 972.

ouse skin tumorigenesis initiated by a single opical application of 20 µg 7,12-dimethylbenz(a)-nthracene: (DMBA) and promoted by repeated pplications of 1 µg phorbol ester was unrelated o genetic differences in the extent of aryl ydrocarbon hydroxylase induction by polycyclic ydrocarbons in inbred or hybrid C57Bl/6N (B6) nd DBA/2N (D2) mice. DMBA did not produce skin umors in any inbred B6 mice but caused tumors in 9 of 21 inbred D2 mice. Hepatic aryl hydrocarbon ydroxylase activity was induced by 3-methylcho-anthrene (MC, 80 mg/kg body wt) about four- to ive-fold inbred B6 mice and was not inducible n inbred D2 mice. Skin aryl hydrocarbon ydroxylase activity was induced by MC (200 µg) bout eight-fold in the B6 mice and less than wo-fold in the D2 mice. Although aryl hydrocar-on hydroxylase activity was inducible in 34 D2 B6D2F$_1$ hybrids and not inducible in 25 B6D2F$_1$ ybrids, the percentage of tumors was about 60% or both groups. Hepatic epoxide hydrase activity as the same among inbred and hybrid B6 and D ice and, in contrast to the hydroxylase, was ot inducible by MC. Epoxide hydrase activity as not detected in the skin of these mice. f aryl metabolism of DMBA is a necessary step n the initiation of chemical carcinogenesis, ne constitutive level of skin hydroxylase ctivity may be sufficient.

097 AUTORADIOGRAPHIC STUDIES OF RNA METABOLISM DURING THE DEVELOPMENT OF DIBUTYLNITRO-AMINE-INDUCED TUMORS OF THE URINARY BLADDER IN RATS.

Ger.) Kunze, E. (Path. Inst. U. Munich, W. ermany), A. Schauer and J. Spielmann. *Z Krebsforsch* (4):236-248, 1971.

Seventy adult female Wistar rats received 20 mg/kg/day of N,N-butylnitrosamine in their drinking water over 50 to 270 day periods. Sharply-delineated urinary bladder mucosal segments missing alkaline phosphatase and with decreased NADH-diaphorase activities were observed within 11 months post-treatment. Preneoplastic enzyme deficiencies were followed by mucosal hyperplasia. Transition states through the development of papillomas and, later, of carcinomas could be seen. Autoradiographic studies of RNA metabolism, one hour after a single i.p. injection of ^{3}H-cytidine, revealed a 2.5 to 4-fold increase in cytidine incorporation occurring in the preneoplastic enzyme-deficient areas, as compared to the surrounding epithelium. Thirteen out of 27 papillomas revealed increased alterations (6 decreased, 8 exhibited no change) in ^{3}H-cytidine incorporation rates with respect to normal tissue. No correlation between enzyme histochemical, autoradiographic and histologic findings could be established. Highest labels were found, however, in papillomas with squamous epithelial metaplasia. Urinary bladder carcinomas exhibited heterogenous ^{3}H-cytidine incorporation rates, (as did the papillomas), which were independent of the degree of cell differentiation; no increased labels with respect to those found in papillomas were ascertained. Further studies are required to clarify the significance of enhanced RNA metabolism in the preneoplastic areas, occurring under deficient enzyme protein synthesis conditions, in urinary bladder carcinogenesis.

0098 LACK OF CYCLAMATE SYNCARCINOGENICITY IN BUTANOL-BUTYL-NITROSAMINE-INDUCED URINARY BLADDER CARCINOGENESIS IN RATS. *(Ger.)*

Schmähl, D. (German Cancer Res. Ctr., Heidelberg, W. Germany) and F. W. Krüger. *Arzneim Forsch* 22(6):999-1000, 1972.

One hundred and twenty 100 day-old male Sprague-Dawley rats were distributed into three groups to test the syncarcinogenicity of cyclamate in 4-hydroxybutanol-butylnitrosamine (BBN)-induced carcinogenesis. One group received 10 mg/kg/day of BBN in their drinking water, the other group received 2,500 mg/kg/day of cyclamate in their diet, in addition to a similar dose of BBN; the third group received no treatment. The feeding experiment lasted 400 days. All 40 rats receiving BBN alone died of squamous cell carcinoma of the bladder which developed after an average latency period of 400 ± 45 days. Twenty seven of the 40 rats which underwent combined treatment had typical urinary bladder carcinomas, and 2 had extensive bladder papillomatosis; 20 of these rats died 440 days after the beginning of the experiment. The surviving 11 rats exhibited hematuria and were suspect of bladder carcinoma. Regression curves for the two groups, plotted with the tumor incidence as ordinant and the latency period as abscissa, were parallel. The high dose of cyclamate produced no enhancement of BBN-induced carcinogenesis. No urinary bladder alterations occurred in the control group.

0099 CYTOMORPHOLOGIC AND MORPHOMETRIC STUDIES OF HEPATOCARCINOGENESIS. II. REVERSIBILITY OF NUCLEAR SIZE ALTERATIONS OCCURRING IN THE NITROSOMORPHOLINE-TREATED RAT LIVER. *(Ger.)*

Romen, W. (Inst. Path. U. Würzburg, Germany), W. Ross and P. Bannasch. *Z Krebsforsch* 73:134-140, 1972.

The morphometry of 73 male Sprague-Dawley rat hepatocyte nuclei was studied after a 7 wk treatment period with nitrosomorpholine (12 mg/100 ml drinking water/day) and 4, 52 and 87-97 weeks after the end of the feeding period. Attention was focused on 4 cell categories referred to as glycogen-poor, "clear" glycogen storage, hepatoma, and hepatocyte population remainder cells, referred to as X-cells. The nuclear area of non-treated rat liver cells reached peak values ranging from 45 to 50 μm^2. Nuclear enlargement to double the normal size was observed in the X-cells and most of the glycogen-storage cells, at the end of the 7 wk treatment period. More than 30% of the X-cells and 25% of the glycogen storage cells had triple nuclear volumes. The glycogen-poor cell nuclei were also enlarged, but to a lesser extent. Four weeks later, a minor regression of the nuclear enlargement was observed in the glycogen storage and some of the X cells, while total disappearance of the glycogen-poor cells occurred. Both glycogen storage and X-cell nuclei returned to normal size 52 wk after the end of carcinogen intake; scattered hepatoma cells and small hepatoma cell clusters were observed. Eighty seven wk after the end of experimental treatment period both storage and X cell nuclei remained unaltered with respect to the observations recorded 52 wk post treatment. Large numbers of hepatoma cells with 75 μm^2-100 μm^2 nuclei, the latter comparable in size to those seen at the end of the acute poisoning stage, were observed, 87 to 97 wk after the end of the carcinogen intake period. Nuclear enlargement occurring in hepatocytes after carcinogen intake should be viewed differently than that in hepatoma cells. The initial nuclear enlargement occurring as a result of toxic effects was found to be a reversible phenomenon and therefore cannot be considered as a precancerous cell reaction. Post treatment studies rather than long-lasting treatment experiments are recommended to clarify the karyologic events related to carcinogenesis.

0100 FOLATE DEFICIENCY AND FORMIMINOGLUTAMIC ACID EXCRETION DURING CHRONIC DIETHYLNITROSAMINE ADMINISTRATION TO RATS. *(E.)*

Poirier, L. A. (Montreal Cancer Inst., Quebec, Canada) and V. M. Whitehead. *Cancer Res* 33(2): 383-388, 1973.

Elevated levels of the histidine catabolite formiminoglutamic acid were excreted into the urine of male Wistar rats given both 0.01% diethylnitrosamine in their drinking water for 1-5 wk and an injection of a loading dose of histidine. Similar histidine loading of control rats that received no carcinogen did not produce an elevation in urinary formiminoglutamic acid excretion. The elevation in urinary formiminoglutamic acid excretion caused by chronic diethylnitrosamine administration was prevented by high dietary levels of the methyl donors methionine, betaine, and choline; high dietary levels of folate and vitamin B_{12}, either alone or in combination, had no significant effect on the elevated formiminoglutamic acid excretion caused by diethylnitrosamine. The elevated formiminoglutamic acid excretion caused by the administration of diethylnitrosamine for 3 wk was associated with a decrease in the hepatic levels of the enzymes formiminoglutamic acid transferase and urocanase, and with a decreased hepatic content of the higher conjugates of folate. Whereas dietary methionine administration completely prevented this decrease in the hepatic content of the higher conjugates of folate, dietary folate had no effect. Diets that contained elevated levels of methionine and choline also led to only a slight reversal of the decreased hepatic formiminoglutamic acid transferase activity caused by diethylnitrosamine. Thus the antagonistic effects on formiminoglutamic acid excretion by diethylnitrosamine and the methyl donors appeared to be mediated through the hepatic content of folic acid cofactors.

0101 INTERACTION OF 4-NITROQUINOLINE 1-OXIDE AND RELATED CARCINOGENS WITH HISTONE AND ALIPHATIC AMINO ACIDS. *(E.)*

Okano, T. (Pharmaceutical Inst., Tohoku U., Japan) and Y. Sato. *Gann* 63(6):713-724, 1972.

Examinations were made on the interaction between carcinogenic quinoline derivatives and calf thymus histone, and 13 of its constituent aliphatic amino acids. The interaction systems of histone and quinolines produced a difference spectrum in the visible region and intensity of this difference spectrum increased by heat denaturation of histone. The 13 aliphatic amino acids were classified into three kinds by the manner of their interaction with quinolines; glycine, alanine, leucine, isoleucine, valine, glutamic acid, and aspartic acid did not show any tendency to interact with the quinolines. Arginine, lysine, and proline, which have basic group with p*K*a larger than 10, produced a new absorption band in the visible spectral region which did not change with time. Methionine, serine, and threonine, which have a nucleophilic group in their molecule, produced a visible difference spectrum by interaction with the quinolines and the spectral intensity increased with time. The complexes formed between basic amino acids and 4-nitroquinoline 1-oxide were analyzed on the basis of Benesi-Hildebrand formula. It was concluded that an n-π charge transfer between quinolines and basic amino acid moiety of the macromolecule played an important part in the intermolecular interaction between the quinolines and histone.

02 DURATION OF SMOKING AND QUANTITY OF TOBACCO USED BY PATIENTS WITH GASTRIC
NCER. *(E.)* Zacho, A. (Dept. Surgery, Finsen
st., Copenhagen, Denmark), C. Cederqvist, J.
elsen and V. Larsen. *Acta Med Scand* 193(1-2):
-48, 1973.

o hundred twenty-one patients with gastric
ncer (g.c.), 174 of them current or ex-smokers,
re questioned about their smoking habits. Data
garding 157 patients (131 men and 26 women) who
d smoked more than a total of 20 kg were
alysed. While the women start smoking at
fferent ages, the men start early, at approxi-
tely the same age, the majority before 16. The
erage duration of smoking was 48 years for men
d 37 years for women. The average quantity of
bacco used was 378 kg for men and 176 kg for
men. Among the women there is a marked dispersion
respect to the duration of smoking as well as
e age on admission for g.c. Among the men there
s an accumulation of admissions around the age of
, i.e. after an average of 48 years of smoking.
us in men there is a definite concurrence between
e duration of smoking and the time of admission
r g.c. In men over 60 years there is a statis-
cally significant relation between the average
tal amount of tobacco used and the time of
mission for g.c., independent of the patient's
e. It is concluded that the use of tobacco may
one of the causes of the higher frequency of
. in men than in women.

03 METABOLIC CONTROLS IN PRECANCEROUS LIVER - II. LOSS OF FEEDBACK CONTROL OF CHO-
TEROL SYNTHESIS, MEASURED REPEATEDLY *IN VIVO,*
ING TREATMENT WITH THE CARCINOGENS N-2-FLUORENYL-
TAMIDE AND AFLATOXIN. *(E.)* Horton, B. J.
ite Agricultural Res. Inst., Australia), J. D.
ton and J. R. Sabine. *Eur J Cancer* 8(4):
-443, 1972.

dback control of cholesterogenesis in rat
er during the course of carcinogen treatment
examined using an assay for cholesterol
thesis which permits repeated tests on the
e animal. Male, hooded-Wistar rats were
ated with 10 μmole/kg AY-9944 (which inhibits
conversion of 7-dehydrocholesterol to
lesterol), and then 24 hr later blood levels of
ehydrocholesterol were measured. This gives
accurate estimation of liver sterol synthesis
ing that 24 hr period. By feeding 5% cholesterol
two days before each alternate assay, it was
sible to measure feedback control of cholesterol
thesis for each individual animal during the
cinogen treatment. Rats injected i.p. with a
gle LD$_{50}$ dose of aflatoxin showed a severe loss
control of cholesterogenesis 3 to 5 weeks
er. Most animals had recovered normal
trol 10 weeks after the aflatoxin treatment.
-fluorenylacetamide feeding caused a similar
ked loss of control within 1 to 2 weeks, and
e also most animals regained normal control
er. With both carcinogens the actual degree
of loss of control of cholesterol synthesis was extremely variable between individual animals. Only a few animals showed complete loss of control and there was a wide range of varying degrees of partial control. Control of cholesterogenesis, as measured by two entirely independent assay systems, has now been shown to be defective after treatment with two different carcinogens, thus confirming the importance in the carcinogenic process, at least in the liver, of this loss of control previously known to occur in all fully-developed hepatomas.

0104 ON THE BINDING OF AFLATOXIN B_1 AND ITS METABOLITES TO HEPATIC MICROSOMES.
(E.) Gurtoo, H. L. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Biochem Biophys Res Commun* 50(3):649-655, 1973.

Rat liver microsomes were incubated with aflatoxin B_1 (AFB$_1$), NADPH and/or SKF-525A, and AFB$_1$ metabolism was studied using the cytochrome P450-dependent mixed function oxidase method. Recording spectrophotometers were used to determine difference spectra of microsomes from the various mixtures. Microsomes pretreated with NADPH showed a peak at ∿412 nm; when SKF-525A was mixed with NADPH for pretreatment, or when microsomes were pretreated with SKF-525A alone, no such peak was seen. The peak at ∿412 nm was apparently due to an AFB$_1$ metabolite covalently bound to microsomes. The metabolite was thought to be related either to AFB$_{2a}$ or to the precursor of AFB$_{2a}$.

0105 EVALUATION OF THE CARCINOGENIC ACTIVITY OF THE HYDROXYMETHYL DERIVATIVES OF 7,12-DIMETHYLBENZ(A)ANTHRACENE BY AN *IN VITRO-IN VIVO* TECHNIQUE.
(E.) Flaks, A. (Sch. Med., Leeds, England), J. M. Hamilton, D. B. Clayson and P. Sims. *Int J Cancer* 10(3):548-551, 1972.

7,12-Dimethylbenz(a)anthracene and three of its metabolites were tested for carcinogenic activity by *in vitro* incubation with BALB/c pulmonary tissue for two days followed by s.c. implantation of the explants into isologous mice. 7,12-Dimethylbenz(a)anthracene and its 7- and 12-hydroxymethyl derivatives showed comparable levels of activity as carcinogens. 7,12-Dihydroxymethylbenz(a)anthracene was inactive in both *in vitro* and *in vivo* tests. Pulmonary tumors appeared in 48% (14/30) of mice injected with explants cultured in the parent compound (4 μg/ml); in 36% (10/28) of mice injected with explants treated with the 7-hydroxymethyl derivative (4 μg/ml); and in 35% (10/29) of mice injected with explants treated with the 12-hydroxymethyl derivative (4 μg/ml). These results suggest that some mechanism not involving hydroxylation of methyl groups is involved in the induction of tumors by 7,12-dimethylbenz(a)anthracene and its 7- and 12-hydroxymethyl metabolites. The possibility that K-region epoxides are involved in the production of tumors is discussed.

0106 BINDING OF TWO DIFFERENT AZODERIVATIVES OF DIFFERENT CARCINOGENICITY BY LIVER PROTEINS AND DNA. *(Fr.)* Chauveau, J. (Inst. Cancer Res., Villejuif, France) and A. Benoit. *C R Acad Sci [D] (Paris)* 276(2):235-238, 1973.

Fixation of ^{14}C-dimethylaminoazobenzene (^{14}C-DAB) and its non-carcinogenic homolog ^{14}C-2-methyl-DAB by total liver proteins and DNA *in vivo* was studied. Male Sprague-Dawley rats were fed a complete synthetic diet containing 22% proteins and 20 mg/kg riboflavin to which 0.06% ^{14}C-DAB or 0.063% ^{14}C-2-methyl-DAB were added. The average food intake amounted to 10 g/day over the experimental period. Rats were sacrificed one, two, or three weeks after the beginning of the experiment following a 15 hr fasting period. Magnesium cation containing sucrose solution liver homogenate aliquots were used for the measurement of protein - and DNA - bound radioactivity, following appropriate separation and purification procedures. No significant differences in both azoderivative metabolite fixation by proteins were observed regardless of compound carcinogenicity and duration of administration. Incorporation of DAB label- amounted to 151 mμ moles/g protein after both one and two weeks and to 188 mμ moles/g protein after three weeks of administration. Incorporation of 2-methyl-DAB amounted to 158, 191, and 248 mμ moles/g protein after one, two, and three weeks of treatment, resp. The label binding kinetics by DNA differed according to compound carcinogenicity. The 2-methyl-DAB level was constant throughout the three-week administration period whereas the DAB-derived label reached a maximum of DNA fixation after 14 days of administration. Incorporation of DAB-derived label *vs* 2-methyl-DAB-derived label by liver DNA presented a ratio equalling 3 after one week and 4 after two weeks of treatment, indicating the involvement of DNA as a target of carcinogen action. Mechanisms related to the carcinogen-DNA interaction are discussed and experiments to obtain some evidence related to the above are in progress.

0107 INDUCTION OF BLADDER CANCER BY *N*-NITROSO-DIBUTYLAMINE AND *N*-NITROSO-BUTANOL-(4)-BUTYLAMINE. *(E.)* Kolar, G. F. (German Cancer Res. Ctr., Heidelberg, W. Germany). *Br J Cancer* 26(6):515-516, 1972.

Bladder carcinogenesis by *N*-nitroso-dibutylamine and *N*-nitroso-butanol-(4)-butylamine could be the result of conversion to an active oxygenated metabolite, rather than alkylation. Assuming that enzymic dealkylation is an essential step in the activation of *N*-nitroso-butanol-(4)-butylamine *in vivo* oxidation of the alcohol group to a carboxyl, followed by enzymic hydroxylation and cleavage, would lead to the release of 4-butanal-1-carboxylic acid which would readily cyclize to its tautomeric lactol. The mechanistic feasibility of this pathway is supported by a recent finding that *N*-nitroso-butanol-(4)-butylamine is metabolized in the rat to *N*-nitroso-butanoic acid-(4)-butylamine in 20% yield; this compound was also shown to be a powerful carcinogen.

0108 ACUTE AND CHRONIC EFFECTS OF INTRAPERITONEAL INJECTION OF TWO TYPES OF ASBESTOS IN RATS WITH A STUDY OF THE HISTOPATHOGENESIS AND ULTRASTRUCTURE OF RESULTING MESOTHELIOMAS. *(E.)* Shin, M. L. (U. Maryland, Sch. Med., Baltimore) and H. I. Firminger. *Am J Pathol* 70(3):291-314, 1973.

Two hundred male Wistar rats weighing 150 to 180 g each were injected i.p. in the left lower quadrant of the abdomen with 50 mg of asbestos fibers. One half of the rats received chrysotile B and the other half crocidolite. Malignant mesotheliomas were induced in the rat peritoneum. The immediate toxicity of the fibers was noted in both groups of animals, producing approximately 40% mortality, within 8 days after the injection associated with acute peritonitis. Tissue reactions to these two types of asbestos were significantly different. Crocidolite fibers were easily seen by light microscopy, in the tissue sections throughout the period of study, and they produced foreign-body giant cell granulomas. However, giant cells were not seen in chrysotile granulomas, and the asbestos fibers were only seen by electron microscopic study. They appeared to be coated by a protein-like substance. During earlier stages of tumorigenesis, the epithelioid and/or mixed cell type mesotheliomas seemed to have no specific relationship to granulomas, but pure spindle cell tumors were seen to develop in close relationship to granulomas, and they appear to be fibrosarcomas. Electron microscopic and histochemical methods were used to define the morphologic characteristics of the tumor cells. The formation of hyaluronic acid was found in cells of the epithelioid type, contrasted with extracellular accumulation in the spindle cell tumors.

0109 A FAST METHOD OF LIVER TUMOR INDUCTION IN RATS. *(Rus.)* Zabezhinskiy, M. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Vopr Oncol* 18(12):85, 1972.

A quick procedure for liver tumor induction in rats is presented. Experiments were performed with two groups of white mongrel rats including 10 males and 10 females each. Rats of the first group received 2.0 mg diethylnitrosamine (DENA) in water intragastrically (probe) 5 days/wk for three months and 4.0 mg DENA thereafter for 1.5 months; the total amount of administered carcinogen reached 240 mg/rat. Rats of the second group were given DENA through their drinking water in 200 mg/l concentrations, the daily intake of the carcinogen ranging between 1/15 and 1/10 LD$_{50}$. Carcinogen administration to both groups lasted 4.5 months. The first laparotomy performed 3.5 and 2.5 months after the beginning of the

xperiment in rats of the first and second groups,
esp., revealed tumor nodules in the liver in 3
ut of 4 rats of the first and in 1 out of 4 rats
f the second group. The second laparotomy
erformed 6 and 5 months after the beginning of
arcinogen administration in rats of the two
roups, resp., revealed liver tumors in all the
ats. Ten rats (4 males and 6 females) of the
irst group and seven rats (5 males and 2 females)
f the second group died before the first
aparotomy of causes indicating no relationship
DENA toxicity. Most of the tumors were
patocellular carcinomas but some cholangiomas,
olangiocarcinomas, and hemangioendotheliomas
re observed also. Neoplastic alterations
veloped on a cirrhotic background in rats of
e first experimental group. Administration of
NA with the drinking water in 200 mg/l concentra-
ons is recommended to achieve liver tumor induc-
on within 2.5-5.0 months.

10 MALIGNANT TRANSFORMATION OF BHK_{21}
CLONE 13 CELLS *IN VITRO* BY NITROSAMINES--
CONDITIONAL STATE. *(E.)* Di Mayorca, G. (U.
linois Med. Ctr., Chicago), M. Greenblatt,
Trauthen, A. Soller and R. Giordano. *Proc Natl
ad Sci USA* 70(1):46-49, 1973.

K_{21} clone 13 hamster cells were cultured with
methylnitrosamine or nitrosomethylurea at 38.5
32 C and transformation was observed. Spontane-
sly transformed BHK_{21} cells, normal cells, and
lyoma virus-transformed cells were also observed
the two temperatures. The growth rate of
emically transformed cells was similar at 38.5
d 32 C; the growth rate of these cells resembled
at of normal and virus-transformed cells. How-
er, while normal and virus-transformed cells
hibited, resp., normal and transformed morphologies
both temperatures, spontaneously and chemically
ansformed cells showed a transformed morphology
38.5 and a normal morphology at 32 C. The
ansformed phenotype of these cells at 38.5 C re-
rted to normal when the temperature was lowered
32 C. It was hypothesized that the conditionally
ansformed state of these cells was due to the
action of nitrosamines with the cell genome to
oduce a temperature-sensitive mutation in a gene
ential for maintenance of the normal phenotype.
s mutation, at the higher temperature, would
press the function or synthesis of the gene
oduct responsible for the normal phenotype.

1 EXPERIMENTAL STUDIES ON TUMORIGENIC ACTIVI-
TY OF CIGARETTE SMOKE CONDENSATE ON
SE SKIN. IV. COMPARATIVE STUDIES OF CONDENSATES
M DIFFERENT RECONSTITUTED TOBACCO SHEETS, THE
ECT OF $NaNO_3$ AS ADDITIVE TO TOBACCO OR RECONSTI-
ED TOBACCO SHEETS, THE EFFECT OF VOLATILE CON-
TUENTS OF SMOKE, THE EFFECT OF INITIAL TREATMENT
H DMBA. *(Ger.)* Dontenwill, W. (Res. Inst.
arette Ind. Hamburg, W. Germany), H.-J. Chevalier,
P. Harke, H.-J. Klimish, U. Lafrenz and G.
kzeh. *Z Krebsforsch* 78(3):236-264, 1972.

The tumorigenic activity of condensates from reference cigarettes, cigarettes made from different reconstituted tobacco sheets as well as from an admixture of reference cigarettes and reconstituted tobacco sheets with 8% sodium nitrate as additive was examined in skin painting experiments with ICI mice. In preparing solutions of condensate by the method applied it was possible that also a portion of volatile smoke components was dissolved. The solution of the condensate from reference cigarettes prepared by this method was compared with a solution containing non-volatile smoke components only (dry condensate). A solution containing nothing but volatile smoke components (vapor phase condensate) was investigated as well. Moreover, different solutions of condensate were investigated on the mouse skin after an initial treatment with DMBA. Applications were made 3 times/week throughout the life span of the mice but were limited to a maximum of two years. No difference in the biological activity with regard to tumor induction was observed between the condensate solution including a portion of the volatile smoke components and the dry condensate.
The vapor phase condensate was virtually ineffective. The tumorigenic activity of condensates made from reconstituted tobacco sheets depended on the manufacturing process employed for preparing reconstituted tobacco sheets. Effects of condensates from reconstituted tobacco sheets made in five different processing techniques were comparable to the condensate of reference cigarettes. Adding 8% $NaNO_3$ to the tobacco of reference cigarettes or to reconstituted tobacco sheets contributed to a considerable reduction of the tumorigenic activity of the corresponding condensates. A distinct syn- or cocarcinogenic effect could be established by using DMBA as a single initiator followed by repeated applications of tobacco smoke condensate of reference cigarettes or of application of a fraction free from cyclic aromatic hydrocarbons as compared to applying either of these solutions alone. The tumor-inducing activity may not be explained by the benzpyrene content of the condensates. There may be, however, a correlation between the amount of fraction VI containing polycyclic aromatic hydrocarbons and the tumorigenic response.

0112 THE EFFECT OF PHYTOHAEMAGGLUTININ AND CYCLOPHOSPHAMIDE ON THE INCIDENCE OF MAMMARY TUMOURS IN RATS. *(E.)* Hunt, P. S. (Prince Henry's Hosp., Melbourne, Australia), E. D. Holyoke and R. Perlmutter. *Aust NZ J Surg* 42(1):96-98, 1972.

At the age of 70 days, the members of each of four groups of Sprague-Dawley rats were given 2 mg, 7,12-dimethylbenz(a)anthracene (DMBA) by intragastric feeding. One control group was given DMBA alone. The second group was given 15 mg cyclophosphamide per kg body wt i.p. on each of five days before and two days after DMBA. Animals in the third group were given 1 ml phytohemagglutin (PHA) i.p. on each of five days before and two days after DMBA. Those in the fourth group were given both PHA and cyclophosphamide. Cyclophosphamide increased the incidence of mammary tumors at one year, but not the number of animals with tumors. The mean number of tumors was 1.6 in the group given DMBA alone and 2.9 in the cyclophosphamide group. PHA significantly inhibited the carcinogenic effect of DMBA and DMBA with cyclophosphamide. No additive effect, reducing tumor incidence, of PHA and cyclophosphamide was noted. PHA administered parenterally in this dosage was toxic and probably caused the early deaths of 12% of rats so treated. The evidence that PHA acts by stimulating surveillance mechanisms to produce inhibition of carcinogenesis is equivocal and indirect. The present work suggests that PHA protects against the development of mammary tumors in rats, perhaps by immune means. This may be because it has an effect when given at a time which coincides with immune surveillance and elimination of neoplastic cells.

0113 CHRONIC MYELOGENOUS LEUKEMIA IN RATS FED 2,7-DIACETYLAMINOFLUORENE. *(E.)*

Takayama, S. (Tokyo Med. Dental U., Japan), T. Yamada and S. Kamata. *Acta Pathol Jap* 22(2): 309-319, 1972.

An unsuccessful attempt was made to induce gastric carcinoma in 327 female Donryu and Wistar rats by oral administration of 2,7-diacetylaminofluorene (0.025% of the basic diet). Of the various neoplasms that were induced, mammary carcinoma, leukemia, and hepatoma showed the highest incidence. Leukemia was detected in 31 rats, 29 which had chronic myelogenous leukemia. Morphologically, this leukemia resembled chronic myelogenous leukemia in man. There was intense proliferation of neutrophilic myeloid cells in the bone marrow, spleen and liver, infiltration of the muscle, kidney, and other organs, and atrophy of bony trabeculae, splenic follicules, and hepatic cell cords. The neutrophilic granulocytes showed every stage of maturation, and there were occasional areas of infiltration by erythroblasts, eosinophilic leukocytes, and megakaryocytes in the bone marrow. Rats affected by chronic myelogenous leukemia survived no longer than 2 wk after the appearance of leukemic cells in peripheral blood.

0114 TRANSPLACENTAL CARCINOGENESIS BY URETHAN IN MICE: TERATOGENESIS AND CARCINOGENESIS IN RELATION TO ORGANOGENESIS. *(E.)*

Nomura, T. (Osaka U. Med. Sch., Japan) and E. Okamoto. *Gann* 63(6):731-742, 1972.

Teratogenesis and carcinogenesis in relation to organogenesis was studied using the properties of short-action and high placental permeability of urethan. Pregnant mice were exposed to urethan on various days of gestation (day 5 to 19) by a "single" s.c. injection into the interscapular region; malformations and neoplasms were induced in their offspring. Lung and tail anomalies were induced in 70-80% of the day 9 urethan-treated group only, and this same group also developed a malignant teratoma. Pulmonary tumors were induced in high incidence in the groups exposed to urethan after day 13 of gestation. Embryological studies revealed that lung buds of mouse fetuses of this strain appeared on day 12. The same chronological relation was observed in induced hepatomas. Histologically, most of these pulmonary tumors were papillary adenomas. When the injection-parturition interval was short and late in term, pulmonary tumors were induced in higher incidence. Actual incidence of pulmonary tumors decreased in the day 19 urethan-treated group, when compared to that in the day 15 or 17 urethan-treated group. About one-third of mothers developed endometrial hyperplasia and hemangioma of uterine or placental origin. When urethan was injected to lactating mothers, pulmonary tumors were induced in more than 50% of sucklings. This indicates that sucklings were exposed to urethan through the mother's milk.

typic structure to be invaded by solid trabecular or tubular structures, the latter consisting mainly of prismatic cells. No infiltrative growth was observed. Nitrosomorpholine poisoning-induced cytoplasmic alterations were categorized into four groups: reversible cytotoxic alterations including loss of glycogen and chromatolysis; persistant toxic and post toxic alterations (occurring after the end of the carcinogen intake period), including increased glycogen storage, referred to as glycogenosis, and hypertrophy of the smooth endoplasic reticulum, occurring in GS, AcG and X cells; temporary posttoxic cytoplasmic alterations including fatty accumulation (FS cells), and progressive posttoxic alterations including a gradual liver parenchymal glycogen loss and ribosomal proliferation along with the developing hepatomas, constituted the other two types of cytoplasmic alterations.

0115 CYTOMORPHOLOGIC AND MORPHOMETRIC STUDIES OF HEPATOCARCINOGENESIS. I. REVERSIBLE AND IRREVERSIBLE ALTERATIONS OF THE LIVER PARENCHYMAL CELLS OCCURRING IN NITROSOMORPHOLINE-POISONED RATS. *(Ger.)*

Bannasch, P. (Inst. Path., U. Würzburg, W. Germany), J. Papenburg and W. Ross. *Z Krebsforsch* 77:108-133, 1972.

The morphology and morphometry of male Sprague-Dawley rat hepatocytes were studied following a 7 to 9 wk treatment period with nitrosomorpholine (12 mg/100 ml drinking water/day) and 4, 52, and 61 to 87 weeks later. Six types of pathologically altered hepatocytes were taken into consideration: basophilic low glycogen (BLG) cells, "clear" glycogen storage (GS) cells, acidophilic glycogen storage (AcG) cells, fat storage (FS) cells, hepatoma (H) cells and population remainder cells referred to as X-cells. Liver lobe sections preparations obtained at the end of the carcinogen intake period (a total of 101.5 to 129.6 mg/animal of nitrosomorpholine) featured 3.55% of BLG, 58.03% of GS, 1.43% of AcG, 0.01% of FS, and 36.51% of X cells. The proportion of BLG cells decreased to 0.19% by 4 wk and to zero at 52 wk post treatment. The GS cells decreased to 15.28% 61 to 87 wk after the end of the treatment period when the AcG cell population reached 2.94%. The fat storage cells reached a peak of 1.89% by 52 wk

ut decreased to 0.38% during the last observation
eriod. The first hepatoma cells were observed
wk after the end of the carcinogen feeding
eriod; the percentage of hepatoma cells increased
om 0.09 to 5.44% by 52 wk and reached 62.30%
uring the last observation period. The X cells
ecreased from 36.51 to 18.72% during this period.
tenfold decrease in necroses and a considerable
icrease in mitoses occurred from the first through
e last post treatment observation period. The
erage single cell area increased by 32% with
espect to intact liver cells at the end of the
eding period, reached its maximum size of 43.7%
ove normal 4 wk after the end of carcinogen
take, and decreased to a minimum (still 17%
ove normal) at the end of the observation
riod. The histologic features of the liver
eparations obtained 61-87 wk after the end
carcinogen intake presented extended hepatomas.
rge areas of the hepatic lobe lost their organo-

16 FAILURE OF TRANSPLACENTAL TUMOUR-INDUCTION
IN RATS BY 7,12-DIMETHYLBENZ(*a*)ANTHRACENE.
.) Kellen, J. A. (Dept. Clin. Biochem., Banting
st., Ontario, Canada). *Res Commun Chem Pathol
armacol* 4(1):135-140, 1972.

17 ANGIOGRAPHY IN DIBUTHYLNITROSAMINE-
INDUCED RAT BLADDER TUMOURS. *(E.)*
elund, L. (U. Hosp., Lund, Sweden), J. Göthlin
d H. Henrikson. *Acta Pathol Microbiol Scand*
80(5):691-693, 1972.

18 RECONSTITUTED LIVER MICROSOMAL ENZYME
SYSTEM THAT HYDROXYLATES DRUGS, OTHER
EIGN COMPOUNDS, AND ENDOGENOUS SUBSTRATES.
. DIFFERENT SUBSTRATE SPECIFICITIES OF THE
OCHROME P450 FRACTIONS FROM CONTROL AND
NOBARBITAL-TREATED RATS. *(E.)* Lu, A. Y. H.
pt. Biochem., Hoffmann-La Roche Inc., Nutley,
.), W. Levin, S. B. West, M. Jacobson, D. Ryan,
Kuntzman and A. H. Conney. *J Biol Chem* 248(2):
-460, 1973.

19 THE MOLECULAR BASIS OF MUTAGENESIS BY
METHYL AND ETHYL METHANESULFONATES.
) Rhaese, H.-J. (Goethe U., Molecular
etics Group, Frankfurt, West Germany), N. K.
tker. *Eur J Biochem* 32(1):166-172, 1973.

0 STILBESTROL AND UROGENITAL-TRACT CANCER
IN ADOLESCENTS AND YOUNG ADULTS. *(E.)*
derson, B. E. (Dept. Path., U. Southern
ifornia, Los Angeles), B. D. A. Benton,
T. Weaver, G. Linden and J. F. Nolan. *N
l J Med* 288(7):354, 1973.

1 CARCINOGENIC EFFECT OF *N*-NITROSOHEXA-
METHYLENIMINE IN SYRIAN GOLDEN HAMSTERS.
) Althoff, J. (U. Nebraska Med. Ctr., Omaha),

A. Cardesa, P. Pour and U. Mohr. *J Natl Cancer Inst* 50(2):323-329, 1973.

0122 OCCURRENCE OF A PRIMARY LIVER CARCINOMA IN A RHESUS MONKEY FED AFLATOXIN B_1. *(E.)* Adamson, R. H. (Natl. Cancer Inst., Bethesda, Md.), P. Correa and D. W. Dalgard. *J Natl Cancer Inst* 50(2):549-553, 1973.

0123 HORMONAL EFFECTS ON THYMIDINE KINASE AND THYMIDYLATE KINASE ACTIVITY OF ESTROGEN-DEPENDENT TUMORS IN THE RAT. *(E.)* Thomson, M. J. (Dept. Biochem., U. British Columbia, Vancouver, Canada), M. R. Garland and James F. Richards. *Cancer Res* 33(2):220-225, 1973.

0124 ACID HYDROLYSIS PRODUCTS OF DDD AND DDT PRECURSORS. *(E.)* Jensen, B. L. (Coll. Pharmacy, U. Michigan, Ann Arbor), and R. Counsell. *J Org Chem* 38(4):835-838, 1973.

0125 STIMULATION OF CELL DIVISION BY MEMBRANE-ACTIVE AGENTS. *(E.)* Ingram, L. O. (Biology Div., Oak Ridge Natl. Lab., Tenn.) and W. D. Fisher. *Biochem Biophys Res Comm* 50(2):200-210, 1973.

0126 INHIBITION OF MICROSOMAL ALDRIN EPOXIDATION BY DIQUAT AND SEVERAL RELATED BIPYRIDYLIUM COMPOUNDS. *(E.)* Krieger, R. I. (Dept. Environmental Toxicol., U. California, Davis), P. W. Lee, A. Black and T. R. Fukuto. *Bull Environ Contam Toxicol* 9(1):1-3, 1973.

0127 MUSCLE ACTIVITY, BODY FAT, AND INDUCED RAT MAMMARY TUMORS. *(E.)* Moore, C. (Louisville, Ky.) and P. W. Tittle. *Surgery* 73(3):329-332, 1973.

0128 HEPATOCARCINOGENICITY OF POLYCHLORINATED BIPHENYLS IN MICE. *(E.)* Nagasaki, H. (Nara Med. U., Japan), S. Tomii, T. Mega, M. Marugami and N. Ito. *Gann* 63(6):805, 1972.

0129 3-METHYLCHOLANTHRENE (MC) BINDING TO DNA, ITS RELATION TO DNA SYNTHESIS IN MOUSE MAMMARY NODULE. *(E.)* Banerjee, M. R. (Zoology Dept., U. Nebraska, Lincoln), D. Kinder and J. Wagner. *Proc Am Assoc Cancer Res* 13(March):123, 1972.

0130 INTERACTION OF AROMATIC AMINE DERIVATIVES WITH BACTERIAL AND PHAGE GENOMES. *(E.)* Tagashira, Y. (Temple U., Sch. Med., Philadelphia, Pa.), S. Fukuda and N. Yamamoto. *Proc Am Assoc Cancer Res* 13(March):62, 1972.

0131 EFFECT OF DMBA ON HUMAN PROSTATE IN ORGAN CULTURE. *(E.)* Noyes, W. F. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Proc Am Assoc Cancer Res* 13(March):81, 1972.

0132 EFFECT OF DOSE FRACTIONATION OF 2-FAA ON CELL SURVIVAL AND HYPERPLASTIC RESPONSE ON THE RAT LIVER. *(E.)* Albert, R. E. (New York U. Med. Ctr., N.Y.), L. Bilger and F. J. Burns. *Proc Am Assoc Cancer Res* 13(March):67, 1972.

0133 FAILURE OF ZINC TO NEGATE CADMIUM CARCINOGENESIS. *(E.)* Furst, A. (U. San Francisco, Calif.) and D. Cassetta. *Proc Am Assoc Cancer Res* 13(March):62, 1972.

0134 STUDIES ON THE MECHANISM OF SKIN TUMOR PROMOTION. THE ROLE OF CELL PROLIFERATION AND EPIDERMAL HYPERPLASIA. *(E.)* Raick, A. N. (Banting Inst., U. Toronto, Canada). *Proc Am Assoc Cancer Res* 13(March):39, 1972.

0135 INHIBITION OF THE CARCINOGENIC ACTIVITY OF BENZO(RST)PENTAPHENE BY ITS FLUORO DERIVATIVES. *(E.)* Boger, E. (Bio-Res. Inst., Cambridge, Mass.) and A. Treger. *Proc Am Assoc Cancer Res* 13(March):37, 1972.

0136 EHRLICH ASCITES CELL FERMENTATION AND WATER ELUTION TESTS OF CHARCOAL ADSORPTIVE CAPACITY AND DETERIORATION IN CIGARETTES. *(E.)* Burk, D. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 13(March):35, 1972.

0137 EFFECT OF P-HYDROXYACETANILIDE (PHAA) AND SODIUM SULFATE (SS) ON THE MURINE LEUKEMOGENICITY OF N-[4(5-NITRO-2-FURYL)-2-THIAZOLYL] ACETAMIDE (NFTA). *(E.)* Cohen, S. M. (U. Wisconsin Med. Sch., Madison), F. J. Ansfield and G. T. Bryan. *Proc Am Assoc Cancer Res* 13(March):35, 1972.

0138 COMPARATIVE STUDIES WITH HYDRAZINE DERIVATIVES: CARCINOGENICITY OF 1,1-DIMETHYLHYDRAZINE, UNSYMMETRICAL (1,1-DMH) IN THE BLOOD VESSELS, LUNGS, KIDNEYS AND LIVER OF SWISS MICE. *(E.)* Toth, B. (U. Nebraska Coll. Med., Omaha). *Proc Am Assoc Cancer Res* 13(March): 34, 1972.

0139 AFLATOXIN B_1 CARCINOGENICITY IN INFANT MICE. *(E.)* Vesselinovitch, S. D. (U. Chicago, Ill.), N. Mihailovich, L. S. Lombard and G. N. Wogan. *Proc Am Assoc Cancer Res* 13(March):49, 1972.

0140 RENAL CARCINOGENESIS BY BENZO(a) PYRENE. *(E.)* Lombard, L. S. (U. Chicago, Ill.) and S. D. Vesselinovitch. *Proc Am Assoc Cancer Res* 13(March):49, 1972.

0141 INCIDENCE OF FISH TUMORS FOUND IN A POLLUTED WATERSHED AS COMPARED TO NONPOLLUTED CANADIAN WATERS. *(E.)* Brown, E. R. (Chicago Med. Sch., Ill.), L. Keith, J. B. G. Kwapinski and J. Hazdra. *Proc Am Assoc Cancer Res* 13(March):45, 1972.

0142 RAT MAMMARY NEOPLASIA AFTER NEONATAL AND/OR ADULT 7,12-DIMETHYLBENZ(A)ANTHRACENE ADMINISTRATION. *(E.)* Brown, R. D. (Med. Dept., Brookhaven Natl. Lab., Upton, N.Y.), A. R. Rao and C. J. Shellabarger. *Proc Am Assoc Cancer Res* 13(March):44, 1972.

0143 STUDIES ON 2-ACETYLAMINOFLUORENE (AAF) CARCINOGENESIS IN ZYMBAL'S GLAND (Z.g.) OF THE RAT. *(E.)* Janss, D. H. (VA Hosp., Memphis, Tenn.) and C. C. Irving. *Proc Am Assoc Cancer Res* 13(March):44, 1972.

0144 MECHANISM OF INTRODUCTION OF FLUORENYLAMINE (FA) SUBSTITUENTS INTO NUCLEIC ACID BY RAT LIVER. *(E.)* King, C. M. (Michael Reese Hosp., Chicago, Ill.) and B. Phillips. *Proc Am Assoc Cancer Res* 13(March):43, 1972.

0145 EFFECT OF MICROSOMAL ENZYME-INDUCING CHEMICALS ON THE METABOLISM OF HEXAMETHYLMELAMINE (HMM, NSC-13875) IN RATS AND HAMSTERS. *(E.)* Worzalla, J. F. (U. Wisconsin Med. Sch., Madison), D. M. Lee, R. O. Johnson and G. T. Bryan. *Proc Am Assoc Cancer Res* 13(March):41, 1972.

0146 REMOVAL OF BOUND CARCINOGEN DURING DNA REPAIR IN NON-DIVIDING HUMAN LYMPHOCYTES. *(E.)* Lieberman, M. W. (Temple U. Med. Sch., Philadelphia, Pa.) and A. Dipple. *Proc Am Assoc Cancer Res* 13(March):40, 1972.

0147 POSSIBLE ROLE OF 6-HYDROXYMETHYLBENZO(A)-PYRENE IN TUMOR INDUCTION WITH BENZO(A)-PYRENE. *(E.)* Flesher, J. W. (Coll. Med., U. Kentucky, Lexington) and K. L. Snyder. *Proc Am Assoc Cancer Res* 13(March):55, 1972.

0148 THE PERSISTENCE AND PROLIFERATION OF PARENCHYMAL CELLS OF THE RAT LIVER IN RELATION TO HEPATOMA INDUCTION BY 2-FAA. *(E.)* Burns, F. J. (New York U. Med. Ctr., N.Y.), R. E. Albert and D. Gardner. *Proc Am Assoc Cancer Res* 13(March): 54, 1972.

0149 PRECOCIOUS APPEARANCE OF α-FETAL PROTEIN IN SERUM OF RATS GIVEN HEPATOCARCINOGENS. *(E.)* Kroes, R. (Natl. Cancer Inst., Bethesda, Md.) and J. H. Weisburger. *Proc Am Assoc Cancer Res* 13(March):53, 1972.

0150 MODIFICATION OF ARYL HYDROCARBON METABOLIS IN SYRIAN HAMSTER CELL CULTURES BY VITAMIN A. *(E.)* Rasmussen, R. E. (Cancer Res. Inst., U. California, San Francisco), I. Y. Wang and T. T. Crocker. *Proc Am Assoc Cancer Res* 13(March):53, 1972.

151 INDUCTION OF CARCINOMA BY *IN VITRO* EX-
POSURE OF EPITHELIAL-LIKE CELLS FROM
AT LIVER TO CHEMICAL CARCINOGENS. *(E.)*
illiams, G. M. (Natl. Cancer Inst., Bethesda,
d.), J. H. Weisburger, J. Elliott and E. K.
eisburger. *Proc Am Assoc Cancer Res* 13(March):53,
972.

52 THE INCORPORATION OF POLYCYCLIC HYDROCAR-
BONS INTO PREFORMED PROTEIN. *(E.)*
orrison, J. C. (U. Tennessee Med. Units, Memphis),
. D. Whybrew, C. M. Sobhy, T. C. Trass, W. C.
orrison, S. A. Fish and E. T. Bucovaz. *Proc Am*
ssoc Cancer Res 13(March):52, 1972.

53 ACETYLATION OF RNase BY THE CARCINOGEN N-
ACETOXY-2-FLUORENYLACETAMIDE (N-ACETOXY-
-FAA). *(E.)* Barry, E. J. (VA Hosp., Minneapolis,
inn.). *Proc Am Assoc Cancer Res* 13(March):52, 1972.

54 ISOLATION AND PARTIAL CHARACTERIZATION OF
A MOUSE-SKIN PROTEIN-BOUND DERIVATIVE OF
BENZ[a,h]ANTHRACENE. *(E.)* Selkirk, J. K.
cArdle Lab., U. Wisconsin, Madison) and C.
idelberger. *Proc Am Assoc Cancer Res* 13(March):59,
72.

55 METABOLIC EFFECTS OF STRONTIUM IN MARINE
HYDRA. *(E.)* Skoryna, S. C. (Gastroin-
estinal Res. Lab., McGill U., Montreal, Ottawa,
anada), K. C. Hong, V. Kratky, E. M. Scheithauer
d M. Fuska. *Proc Am Assoc Cancer Res* 13(March):
, 1972.

56 A MAMMALIAN DETECTION SYSTEM FOR CHEMICAL
MUTAGENS. *(E.)* Capizzi, R. L. (Biomed.
b., Edgewood Arsenal, Md.), W. J. Smith and B.
pirmeister. *Proc Am Assoc Cancer Res* 13
arch):58, 1972.

57 *IN VITRO* TRANSFORMATION OF FELINE EM-
BRYO CELLS BY A CHEMICAL CARCINOGEN.
.*)* Rhim, J. S. (Natl. Inst. Hlth., Bethesda,
.), W. Vass, R. J. Huebner and W. A. Nelson-Rees.
oc Am Assoc Cancer Res 13(March):58, 1972.

58 RESPIRATORY INFECTION, A CO-FACTOR IN
RESPIRATORY CARCINOGENESIS. *(E.)*
tteshein, P. (Biology Div., Oak Ridge Natl.
., Tenn.), C. Richter and W. Lijinsky. *Proc Am*
soc Cancer Res 13(March):57, 1972.

59 CORRELATION BETWEEN THE CARCINOGENICITY OF
ISOMERIC N-HYDROXY-ACETYLAMINOFLUORENES
THEIR *IN VITRO* EFFECT ON MITOCHONDRIA. *(E.)*
iler, H. I. (Chem., Biochem., Southern Illinois
U., Carbondale), B. G. Daniel and H. R. Gutmann. *Proc Am Assoc Cancer Res* 13(March):56, 1972.

0160 ULTRASTRUCTURAL STUDIES DURING RESPIRATORY CARCINOGENESIS. *(E.)* Harris, C. C. (Natl. Cancer Inst., Bethesda, Md.), F. E. Jackson, J. M. Smith, M. B. Sporn and U. Saffiotti. *Proc Am Assoc Res* 13(March):55, 1972.

0161 EFFECTS OF FRESH SMOKE FROM MARIJUANA AND KENTUCKY STANDARD CIGARETTES ON FIBROBLASTIC CELLS OF ADULT HUMAN LUNG EXPLANTS. *(E.)* Leuchtenberger, C. (Swiss Inst. Exp. Cancer Res., Lausanne, Switzerland), R. Leuchtenberger and A. Schneider. *Proc Am Assoc Cancer Res* 13(March):9, 1972.

0162 ENZYMIC HYDROLYSIS OF METHYLAZOXYMETHANOL ACETATE (MAMAc) BY SERUM. *(E.)* Ball, C. R. (Dept. Exp. Path., Cancer Res., U. Leeds, England) and H. W. van den Berg. *Proc Am Assoc Cancer Res* 13(March):9, 1972.

0163 EFFECTS OF HYPOPHYSECTOMY ON LIVER CARCINOGENESIS IN RATS GIVEN DL-ETHIONINE OR α-NAPHTHYLISOTHIOCYANATE. *(E.)* Ito, N. (Dept. Path., Nara Med. U., Japan), Y. Hiasa, Y. Yokota, S. Makiura, Y. Kamamoto and Y. Konishi. *Proc Am Assoc Cancer Res* 13(March):9, 1972.

0164 INDUCTION OF URINARY BLADDER TUMORS IN HAMSTERS WITH 3,2'-DIMETHYL-4-AMINODIPHENYL. *(E.)* So, B. T. (American Hlth. Fdn., New York, N.Y.) and E. L. Wynder. *Proc Am Assoc Cancer Res* 13(March):6, 1972.

0165 ESTRIOL BEST ESTROGENIC INHIBITOR OF DMBA INDUCED RAT BREAST TUMORS. *(E.)* Lemon, H. M. (U. Nebraska Med. Ctr., Omaha). *Proc Am Assoc Cancer Res* 13(March):4, 1972.

0166 INHIBITION OF CARCINOGENIC AND TOXIC EFFECTS OF POLYCYCLIC HYDROCARBONS BY PHENOLIC ANTIOXIDANTS AND ETHOXYQUIN. *(E.)* Wattenberg, L. W. (Dept. Path., U. Minnesota, Minneapolis). *Proc Am Assoc Cancer Res* 13(March):3, 1972.

0167 URINARY BLADDER CARCINOMA AND OTHER PATHOLOGICAL ALTERATIONS IN RATS FED CYCLAMATES. *(E.)* Richardson, H. I. (Food Drug Adm., U. S. Dept. HEW, Washington, D.C.), M. E. Richardson, H. L. Stewart, E. J. Lethco and W. C. Wallace. *Proc Am Assoc Cancer Res* 13(March):2, 1972.

0168 ENHANCEMENT OF TUMORIGENESIS IN THE URINARY BLADDER OF HAMSTERS BY COADMINISTRATION OF 2-ACETYLAMINOFLUORENE (AAF) AND INDOLE.

(E.) Oyasu, R. (Northwestern U. Med. Sch., Chicago, Ill.), T. Kitajima, H. Sumie and M. Hopp. *Proc Am Assoc Cancer Res* 13(March):13, 1972.

0169 THE CARCINOGENICITY OF INTRAVENOUS NICKEL CARBONYL IN RATS. *(E.)* Lau, T. (U. Connecticut Sch. Med., Newington), R. L. Hackett and F. W. Sunderman, Jr. *Proc Am Assoc Cancer Res* 13(March):13, 1972.

0170 ALTERATION OF RNA TRANSPORT IN RAT LIVER DURING CARCINOGENESIS. *(E.)* Shearer, R. W. (Pacific Northwest Res. Fdn., Seattle, Wash.) and E. A. Smuckler. *Proc Am Assoc Cancer Res* 13(March):12, 1972.

0171 1'-HYDROXYSAFROLE: A PROXIMATE CARCINOGENIC METABOLITE OF SAFROLE. *(E.)* Wislocki, P. G. (U. Wisconsin Med. Ctr., Madison), P. Borchert, E. C. Miller and J. A. Miller. *Proc Am Assoc Cancer Res* 13(March):12, 1972.

0172 ACTIVATION OF CARCINOGENIC AROMATIC HYDROXYLAMINES BY ENZYMATIC O-ACETYLATION. *(E.)* Bartsch, H. (U. Wisconsin Med. Ctr., Madison), J. A. Miller and E. C. Miller. *Proc Am Assoc Cancer Res* 13(March):12, 1972.

0173 POST LYSOSOMAL TOTAL METABOLITES OF BENZO(A)PYRENE. *(E.)* Raha, C. R. (Eppley Inst., U. Nebraska, Omaha), C. H. Gallagher and C. Grandjean. *Proc Am Assoc Cancer Res* 13(March):11, 1972.

0174 THE RELATIONSHIP OF ESTROGEN AND PROLACTIN IN MAMMARY TUMORIGENESIS IN RATS. *(E.)* Sinha, D. (Roswell Park Mem. Inst., Buffalo, N.Y.), D. Cooper and T. L. Dao. *Proc Am Assoc Cancer Res* 13(March):10, 1972.

0175 THE BINDING OF 9,10-DIMETHYL-1,2-BENZANTHRACENE TO MOUSE EPIDERMAL SATELLITE DNA *IN VIVO*. *(E.)* Zeiger, R. S. (Natl. Inst. Hlth., Bethesda, Md.), R. Salomon, N. Kinoshita and A. C. Peacock. *Proc Am Assoc Cancer Res* 13(March): 24, 1972.

0176 ON THE MECHANISM OF HORMONE DEPENDENT MAMMARY TUMOR REGRESSION. *(E.)* Gullino, P. M. (Natl. Inst. Hlth., Bethesda, Md.) and Y. S. Cho-Chung. *Proc Am Assoc Cancer Res* 13(March):23, 1972.

0177 INDUCTION OF SQUAMOUS CELL NEOPLASIA IN RATS WITH A BENZO(a)PYRENE-FERRIC OXIDE MIXTURE. *(E.)* Blair, W. H. (Mercy Hosp./Med. Ctr., Chicago, Ill.), K.-C. Chen and H. K. Basavaraj. *Proc Am Assoc Cancer Res* 13(March):19, 1972.

0178 SEPARATION OF REGENERATION AND NEOPLASTIC HYPERPLASIA IN LIVER OF RATS FED 3' METHYLDIMETHYLAMINOAZOBENZENE. *(E.)* Yager, J. D., Jr. (McArdle Lab., U. Wisconsin, Madison) and V. R. Potter. *Proc Am Assoc Cancer Res* 13(March): 19, 1972.

0179 MOLECULAR BASIS OF ALKYLATION OF DNA BY CARCINOGENIC EPOXIDES. *(E.)* Paul, J. S. (U. Texas, Dallas) and P. O'B. Montgomery. *Proc Am Assoc Cancer Res* 13(March):16, 1972.

0180 ONCOGENIC PURINE DERIVATIVES: SELECTIVE BLOCKING OF REACTIVITIES BY METHYL SUBSTITUENTS. *(E.)* Brown, G. B. (Sloan-Kettering Inst., New York, N.Y.), M. N. Teller, N. J. M. Birdsall, T. C. Lee and J. C. Parham. *Proc Am Assoc Cancer Res* 13(March):14, 1972.

0181 ALTERATION OF THE CARCINOGENIC ACTIVITY OF 7,12-DIMETHYLBENZ(a)ANTHRACENE (DMBA) BY LIGHT. *(E.)* Davies, R. E. (Temple U. Med. Sch., Philadelphia, Pa.), H. A. Dodge and L. H. DeShields. *Proc Am Assoc Cancer Res* 13(March):14, 1972.

0182 CELL PROLIFERATION PATTERNS IN MCA-INDUCED SARCOMA. *(E.)* Post, J. (Goldwater Mem. Hosp., Welfare Island, New York, N.Y.), R. J. Sklarew and J. Hoffman. *Proc Am Assoc Cancer Res* 13(March):34, 1972.

0183 MALIGNANT TRANSFORMATION OF RAT EMBRYO FIBROBLASTS BY FLUORENYLHYDROXAMIC ACIDS. *(E.)* Gutmann, H. R. (VA Hosp., Minneapolis, Minn.), L. I. Sekely and D. Malejka-Giganti. *Proc Am Assoc Cancer Res* 13(March):32, 1972.

0184 INFLUENCE OF 1,10-PHENANTHROLINE (1,10-PHE) ON ETHIONINE CARCINOGENESIS IN RAT LIVER. *(E.)* Brada, Z. (Papanicolaou Cancer Res. Inst., Miami, Fla.), S. Bulba and F. Montes de Oca. *Proc Am Assoc Cancer Res* 13(March):32, 1972.

0185 THE EFFECT OF OVARIECTOMY ON EXPERIMENTAL MAMMARY CARCINOMA IN THE RAT WITH PORTACAVAL SHUNT. *(E.)* Reichle, F. A. (Temple U. Hlth. Sci. Ctr., Philadelphia, Pa.), J. F. O'Grady, R. M. Reichle, M. Gruenstein and G. P. Rosemond. *Proc Am Assoc Cancer Res* 13(March):31, 1972.

0186 REACTION OF *N*-HYDROXY-2-ACETYLAMINOFLUORENE (*N*-HO-AAF) WITH PROTEINS AND NUCLEIC ACIDS IN PRESENCE OF CARBAMYL PHOSPHATE (CP) *(E.)* Lotlikar, P. D. (Temple U. Med. Sch., Philadelphia, Pa.) and M. B. Wasserman. *Proc Am Assoc Cancer Res* 13(March):28, 1972.

0187 EFFECT OF SPLENECTOMY ON MAMMARY TUMOR AND LYMPHOMAS PRODUCED BY 7,12-DIMETHYL-

ENZ(a)ANTHRACENE (DMBA) IN MICE. *(E.)* Akamatsu, . (Med. Coll. Georgia, Augusta) and B. P. arton. *Proc Am Assoc Cancer Res* 13(March):27, 972.

0188 METABOLIC FORMATION OF A K-REGION EPOXIDE OF BENZO(a)PYRENE. *(E.)* Grover, P. L. (Inst. Cancer Res., London, England) and P. Sims. *Proc Am Assoc Cancer Res* 13(March):25, 1972.

0189 FORMATION OF NITROSAMINES FROM TERTIARY AMINES AND RELATED COMPOUNDS. *(E.)* ijinsky, W. (Biol. Div., Oak Ridge Natl. Lab., enn.). *Proc Am Assoc Cancer Res* 13(March):68, 972.

0190 CARCINOGENICITY AND METABOLIC DISPOSITION OF 2-AMINO-4-(5-NITRO-2-FURYL)THIAZOLE ANFT) IN SWISS MICE. *(E.)* Lower, G. M., Jr. U. Wisconsin Med. Sch., Madison) and G. T. Bryan. *roc Am Assoc Cancer Res* 13(March):98, 1972.

0191 ETHYLATION OF DNA *VERSUS* CANCER INDUCTION WITH ETHIONINE. *(E.)* Cox, R. (Fels es. Inst., Temple U., Philadelphia, Pa.) and E. arber. *Proc Am Assoc Cancer Res* 13(March):97, 1972.

0192 THE REACTION OF THE CARCINOGEN PROPANE SULTONE WITH GUANOSINE, ADENOSINE AND DNA. *E.)* Goldschmidt, B. M. (New York U. Med. Ctr., .Y.), K. Frenkel and B. L. Van Duuren. *Proc Am ssoc Cancer Res* 13(March):94, 1972.

0193 *IN VIVO* AND *IN VITRO* EFFECTS OF β-PROPIOLACTONE ON MOUSE SKIN CHROMATIN ND DNA. *(E.)* Segal, A. (New York U. Med. Ctr., .Y.), M. Schroeder and B. L. Van Duuren. *Proc m Assoc Cancer Res* 13(March):94, 1972.

0194 ETHYL NITROSOUREA CARCINOGENESIS IN THE NEONATAL MARSUPIAL (*DIDELPHYS MARSUPIALIS IRGINIANNA KERR*) - A NEW ANIMAL MODEL FOR ONCOGENESIS. *(E.)* Jurgelski, W. (Natl. Inst. Environmental Hlth. Sci., Research Triangle Park, N.C.), . M. Hudson, H. L. Falk and P. Kotin. *Proc Am ssoc Cancer Res* 13(March):93, 1972.

0195 ARYL HYDROCARBON HYDROXYLASE REGULATION IN MAMMALIAN CELL HYBRIDS. *(E.)* Wiebel, . J. (Natl. Cancer Inst., Bethesda, Md.), H. G. oon and H. V. Gelboin. *Proc Am Assoc Cancer Res* 3(March):82, 1972.

0196 A STUDY ON THE EFFECT OF CARCINOGENIC AND NON-CARCINOGENIC POLYCYCLIC HYDROCARBONS N THE POLAROGRAPHIC MAXIMUM OF COPPER. *(E.)* odany, V. (Cancer Res. Inst., Slovak Acad. Sci., ratislava, Czechoslovakia) and L. Bahna. *Neoplasma* 9(5):483-488, 1972.

0197 COCARCINOGENICITY OF LOW TOXICITY FACTORS. *(Rus.)* Kurlyandskiy, B. A. (Moscow City Sanitary and Epidemiol. Station, USSR.), A. G. Medvedovskiy and F. D. Mashbits. *Gig Sanit* (10): 83-85, 1972.

0198 SQUAMOUS CARCINOMA OF THE STOMACH FOLLOWING CORROSIVE ACID BURNS. *(E.)* Eaton, H. (Dept. Surg., Path., U. Ceylon, Peradeniya) and G. E. Tennekoon. *Br J Surg* 59(5):382-387, 1972.

0199 SHOCK STRESS AND DMBA-INDUCED MAMMARY TUMORS. *(E.)* Newberry, B. H. (Dept. Psychology, U. Wisconsin, Madison), G. Frankie, P. A. Beatty, B. D. Maloney and J. C. Gilchrist. *Psychosom Med* 34(4):295-303, 1972.

0200 TEMPERATURE-DEPENDENT KINETICS OF THE FORMATION OF PARAMAGNETIC COMPLEX CENTERS RESULTING FROM THE INTERACTION BETWEEN CARCINOGENIC HYDROCARBONS AND ELECTRON ACCEPTORS (HALOGENS). *(Rus.)* Petyaev, M. M. (Rostov Oncological Inst., USSR), V. S. Skotnikov and V. V. Denisov. *Buil Eksp Biol Med* 74(8):83-85, 1972.

0201 HYDROCARBONS IN HUMAN ENVIRONMENT. PART IX. CONTENT OF POLYCYCLIC HYDROCARBONS IN ICELAND SOIL. *(Ger.)* Grimmer, G. (Biochem. Inst. Environm. Carcinogens, Hamburg, W. Germany), J. Jacob and A. Hildebrandt. *Z Krebsforsch* 78(1):65-72, 1972.

See also:

* (Rev): 0009
* (Phys): 0207, 0216
* (Viral): 0225, 0235, 0248, 0289, 0329
* (Immun): 0352, 0378, 0384, 0388, 0389, 0392, 0394, 0400, 0409, 0458, 0461, 0494, 0504, 0518
* (Path): 0557
* (Epid-Biom): 0562, 0564, 0565, 0570, 0576

0202 CARCINOMA OF LARGE INTESTINE IN PATIENTS IRRADIATED FOR CARCINOMA OF CERVIX AND UTERUS. *(E.)* Castro, E. B. (Mem. Hosp. Cancer Allied Dis., New York, N.Y.), P. P. Rosen and S. H. Q. Quan. *Cancer* 31(1):45-52, 1973.

Twenty-six women patients with tumors of the colon rectum, or anus following radiation therapy for cancer of the cervix or uterus were analyzed. Nearly two thirds of the patients were more than 60 years old. The interval between irradiation and diagnosis of the second tumor varied from 5 to 30 years. Despite the lack of detailed information on radiation factors in half of the patients, circumstantial evidence from this study suggests that radiation therapy may have contributed to the development of colorectal adenocarcinoma in some of the patients. Among the findings supporting this conclusion in this series are: 1. localization of second tumor at site of radiotherapy; 2. 58% of the patients had symptomatic and clinically documented chronic radiation proctocolitis at sites of subsequent tumor; 3. 69% of the patients had post-irradiation interval of more than ten years; 4. 58% of the patients had histologic proof of radiation-related alterations in involved bowel segment; 5. occurrence of a much higher than expected percentage (58%) of mucinous (colloid) adenocarcinoma in involved region, of which 92% had clinical and/or histologic evidence of radiation proctocolitis. The overall and determinate 5-year survival in this series was 20% and 22%, resp.

0203 CANCERS INDUCED IN THE BABOON MONKEY *(PAPIO PAPIO)* BY INHALATIONAL EXPOSURE TO PuO_2. *(Fr.)* Metivier, H. (Atomic Energy Comm., Dept. Exp. Toxicol. Path., Montrouge, France), D. Nolibe, R. Masse and J. Lafuma. *C R Acad Sci* [*D*] *(Paris)* 275(25):3069-3071, 1972.

The carcinogenicity of radioactive plutonium dioxide ($^{239}PuO_2$) was studied in 19 young baboon monkeys weighing 3 to 4 kg. The 2-3-yr-old monkeys were exposed to inhalation of radioactive PuO_2 aerosol of 0.5 μ particle size, under experimental conditions, maintained on a fruit diet in individual cages, and sacrificed at 30 to 180 days after exposure. Two monkeys sacrificed 30 days after exposure and having 0.01 to 0.03 μCi/g fresh lung tissue activities elicited no pretumoral lesions. Ten monkeys, sacrificed 30 to 50 days post exposure and having 0.1 μCi/g fresh lung tissue activity, had lung alveolar canal malpighian metaplasia foci. Two monkeys had epidermoid lung carcinomas, and one presented bronchial metaplasia when sacrificed 80 and 130 days post PuO_2 inhalation. Of the two monkeys, sacrificed 180 days post-exposure, one presented lung atelectasia and the other had two mucus-secreting adenocarcinomas of the lung. Two monkeys with an initial lung radioactivity of 0.02 and 0.03 μCi/g were still alive after 90 and 780 days, resp. Lung tumors induced by ^{239}Pu developed much sooner in monkeys than in dogs, rats or mice. Baboon monkeys constitute an excellent model for radioactivity-induced carcinogenesis studies.

0204 IRRADIATION-INDUCED THYROID NEOPLASMS. CASE REPORTS AND REVIEW. *(E.)* Frankel, K. A. (Ohio State U. Coll. Med., Columbus), N. R. Thomford and W. J. Holaday. *Ohio State Med J* 68(4):347-349, 1972.

The association of external irradiation of the head and neck and thyroid cancer is explored in four case reports in which thyroid carcinoma followed head and neck irradiation in two men and two women aged 14-36 yrs. A brief review of the literature is also presented. External head and neck irradiation is associated with benign as well as malignant thyroid lesions. A linear relationship appears to exist between radiation dose and thyroid nodule development. It is speculated that radiation causes thyroid hypofunction, as a response to which the pituitary releases excessive amounts of thyroid stimulating hormone, which plays a part in the development of thyroid hyperplasia, adenomas and carcinomas.

0205 RADIATION DOSE-TIME RELATIONS FOR INDUCTION OF OSTEOSARCOMA IN MICE AND DOGS AND THEIR BEARING ON MAXIMAL PERMISSIBLE BURDEN OF ^{90}Sr IN MAN. *(E.)* Blair, H. A. (U. Rochester Sch. Med. Dent., N.Y.). *Health Phys* 23(6):759-765, 1972.

A method is discussed for deriving latent periods when survival times and quantities of injected carcinogenic nuclides (^{239}Pu, ^{226}Ra, ^{90}Sr) are the only data available. It is suggested that the toxicities with respect to bone cancer production by the three nuclides are related better on the basis of either radiation dose or injected dose causing cancer in equal times than on total tumor incidence over an indefinite time. The toxicity of ^{239}Pu was found to be 15.7 times that of ^{90}Sr and the toxicity of ^{226}Ra to be 2.5 times that of ^{90}Sr, on the basis of initiating dose. Whereas it had been thought that the toxicity of ^{226}Ra/injected dose was 200 times that of ^{90}Sr in dogs and mice, the present study indicated that the toxicity of ^{226}Ra/injected dose was no more than 40 times that of ^{90}Sr. This finding necessitated a lowering of the maximum permissible dose for man value for ^{90}Sr.

0206 LEUKEMIA FROM LOW-LEVEL RADIATION: IDENTIFICATION OF SUSCEPTIBLE CHILDREN. *(E.)* Bross, I. J. (Park Mem. Inst., Buffalo, N. Y.) and N. Natarajan. *N Engl J Med* 287(3):107-110, 1972.

A study of 295 leukemic children and 813 controls was performed in which the effect of intrauterine X-irradiation on childhood leukemia risk was investigated. Given the common assumption that children xposed to intrauterine radiation were similar with espect to leukemia susceptibility, radiation did ot appear significantly to affect leukemia risk. t was suggested, however, that the children studied ere in fact not similar with respect to leukemia isk. Rather, it was thought, children were ither "susceptible" or "nonsusceptible" to leukemia, nd their susceptibility influenced the hazard of adiation for them. Children were considered susceptible" or "nonsusceptible" on the basis of edical history of bacterial, allergic and viral iseases. It was found that the risk of leukemia or "susceptible" children was as much as ten imes greater than the risk for "nonsusceptible" hildren, and that "nonsusceptible" children were naffected by radiation doses which were orders of agnitude greater than doses which affected "suscepible" children.

207 A MODEL SYSTEM FOR THE EVALUATION OF THE ROLE OF CHOLESTEROL OXIDE IN ULTRAVIOLET ARCINOGENESIS. *(E.)* Black, H. S. (Baylor Coll. ed., Houston, Tex.) and D. R. Douglas. *Cancer es* 32(12):2630-2632, 1972.

kin specimens from albino hairless mice were ncubated in radiolabeled cholesterol and then rradiated for 15, 30, or 60 min with a mercury rc lamp (1.43 X 10^5 ergs/sq cm/sec of exposure). he total lipid fraction was applied to silica el-coated thin-layer chromatographic plates and eveloped with 1,2-dichloroethane. Polar material emaining at the origin of the plates was eluted nd rechromatographed. A band corresponding to he R_F of reference cholesterol α-oxide (cholestan-,6α-epoxy-3β-01) was eluted and prepared for adio-gas-liquid chromatographic analysis. A omparison of eluted radioactivity profiles learly indicated the presence of cholesterol -oxide as a component of the photooxidized onstituents derived from the radiolabeled holesterol taken up by mouse skin. The levels f the compound formed in mouse skin were dose ependent. Three criteria are suggested as the inimum required to establish a causal involveent of cholesterol α-oxide in the etiology of / carcinogenesis: comparable effects by both he primary incitant (UV light) and cholesterol -oxide, the same predisposing specificity, and direct relationship between the ability of the rimary incitant to form tumors and produce holesterol α-oxide.

208 THE MORPHOLOGY AND ENZYME HISTOCHEMISTRY OF ^{224}Ra-INDUCED OSTEOGENIC TUMORS OF THE OUSE. *(Ger.)* Gössner, W. (Inst. Biol., Munich, Germany), B. Hindringer, A. Luz and M. Schwabe. *Krebsforsch* 78(3):225-235, 1972.

o thousand 3-4 wk-old male and female NMRI mice ere injected i.p. with single doses of ^{224}Ra s chloride) ranging from 5 to 100 μCi/kg body wt. ndibular osteoblastic sarcomas occurred in 13 male and 10 female mice and sarcomas of the skull located elsewhere developed in 14 mice with an equal sex distribution. Sarcomas of the spinal cord, pelvis, and limb bones developed in 19, 15, and 14 female, and in 4, 2, and 4 male mice, resp., after a latency period ranging from 6 to 31 months. None of the 500 control mice developed osteogenic tumors. The structure of the tumors could not be related to sex or dose of radioactive agent. According to their morphology two major groups of tumors were distinguished. Tumors with no specific osteoblastic cells were referred to as heterologous and tumors including osteoblastic cells were referred to as homologous. The heterologous tumors were polymorphous cell sarcomas with prevalent spindle cell structures, and scattered bone particles. The homologous tumor preparations presented a regular large osteoblast-like cell distribution intermingled with spindle cells and osteoclast-like giant cells. Most of the sarcomas featured infiltrating growth, while others manifested malignancy through their polymorphism and numerous mitoses concentrated in the osteoblast-like cells. A certain site and structure relationship was observed in that most of the skull tumors were heterologous and most of the limb bone tumors were of the homologous type. Alkaline phosphatase activity featured a wide range of distribution with a highest activity in the large osteoblasts. Acid phosphatase levels were highest in the giant cells, and much lower in skull tumors than in sarcomas located elsewhere.

0209 RADIATION PROTECTIVE EFFECT OF A MIXTURE OF ATP, AET, AND SEROTONIN ON YIELDS OF 600-R X-RAY-INDUCED CHROMOSOME ABERRATIONS IN THE RAT. *(E.)* Baev, I. (Res. Inst. Radiology, Radiation Hygiene, Sofia, Bulgaria) and D. Benova. *Dokl Bulg Akad Nauk* 25(8):1129-1131, 1972.

0210 RADIATION-RELATED LEUKAEMIA AND LYMPHOMA. *(E.)* Black, P. J. (Guy's Hosp., London, England). *Lancet* (7762):1236, 1972.

0211 CARCINOMA ARISING IN POST-BURN SCAR. *(Fin.)* Rintala, A. (No affiliation) and L. Tuominen. *Duodecim* 88(9):639-645, 1972.

0212 THE EFFECTS OF ^{144}Ce-^{144}Pr ON THE METAPHASE CHROMOSOMES OF THE CHINESE HAMSTER LIVER CELLS *IN VIVO*. *(E.)* Brooks, A. L. (Lovelace Fdn. Med. Educ. Res., Albuquerque, New Mexico), R. O. McClellan and S. A. Benjamin. *Radiat Res* 52(3):481-498, 1972.

0213 BREAST TUMORS AND RADIATION IN MAN AND RODENTS. *(E.)* Warren, S. (Cancer Res. Inst., Bethesda, Md.), O. Gates and R. N. Chute. *Proc Am Assoc Cancer Res* 13(March):26, 1972.

0214 THYROID NEOPLASMS FOLLOWING THERAPEUTIC IRRADIATION OF THE NECK IN ADULTS. *(E.)* Seydel, H. G. (American Oncologic Hosp., Philadelphia, Pa.). *Proc Am Assoc Cancer Res* 13(March):69, 1972.

0215 ENHANCEMENT OF RADIATION CARCINOGENESIS IN MICE BY CYCLIC IMMUNOSUPPRESSION WITH CORTISONE ACETATE. *(E.)* Pomeroy, T. C. (Natl. Inst. Hlth., Bethesda, Md.) and W. G. Hardy. *Proc Am Assoc Cancer Res* 13(March):25, 1972.

0216 STUDIES ON ONCOGENESIS IN MICE OF A TUMOR-RESISTANT STRAIN X/Gf. *(E.)* Goldfeder, A. (Dept. Hlth. Hospitals, New York U., New York City). *Proc Am Assoc Cancer Res* 13(March):20, 1972.

0217 LEUKEMOGENIC VIRUS RELEASE IN MICE BY IRRADIATION. *(E.)* Haran-Ghera N. (Dept. Exp. Biol., Weizmann Inst. Sci., Rehovot, Israel) and A. Peled. *Proc Am Assoc Cancer Res* 13(March):10, 1972.

0218 LOW-LEVEL RADIATION AND LEUKEMIA: IDENTIFICATION OF SUSCEPTIBLE CHILDREN. *(E.)* Bross, I. D. J. (Roswell Park Mem. Inst., Buffalo, N.Y.) and N. Natarajan. *Proc Am Assoc Cancer Res* 13(March):2, 1972.

See also:

* (Rev): 0010
* (Viral): 0250, 0259, 0289
* (Immun): 0396
* (Path): 0558, 0559
* (Epid-Biom): 0561

0219 THE ONCOGENECITY OF MONKEY ADENOVIRUS SA7(C8) IN MICE AND RATS. *(Rus.)* Tsetlin, E. M. (L. A. Tarasevich State Control Inst. Med. Biol. Prep., Moscow, USSR.), I. S. Levenbuk, and A. D. Al'tshtein. *Vopr Onkol* 18(9):37-42, 1972.

The oncogenecity of monkey adenovirus SA7(C8) in ice and rats was studied. Marmoset adenovirus SA7(C8)-containing material was injected s.c. (0.2 l/animal) to mice of the lines CBA, CC57Br, AKR, C3HA, C57Bl/6, and A/He, and August line rats during the first 24 hr after birth. Animals eveloping tumors were killed to study the tumors nd internal organs. To test the transplantability f the tumors, 10% tumor tissue suspension in a edium with 10% bovine serum was prepared and inoculated s.c. to other animals. Complement fixation ests were used for a serologic study, using 10% umor extract in a physiological saline as antigens, era of the animals with tumors as immune sera, and amster sera with T-antigen antibodies to SA7(C8) and V-40 viruses. The adenovirus SA7(C8) was oncogenic n mice of the lines of CBA (in all 4 mice), AKR in 2/3), and CC57Br (in 1/2), developing tumors in 2-94 days. The virus did not induce tumors in ice of the lines C3HA, C57Bl/6, and A/He during n 8 months observation period. The virus induced umors in rats upon s.c. (17/23 rats) and i.p. 3/6) inoculation. Macroscopically, both mouse nd rat tumors did not involve surrounding tissue. he tumors of the rats showed denser constituency nd less necrotic changes than those observed in ice. Microscopically, the rat tumors were poorly ifferentiated hemangiopericytomas and the mouse umors were rhabdomyosarcomas. The tumors induced n mice by SA7(C8) virus were transplantable only o syngeneic adult mice (CBA and CC57Br). The at tumors were not transplantable. The transplanted mouse tumors differed from the primary tumors y their intense anaplasia. The primary tumor cells f mice of the lines CBA and AKR were successfuly ultured in a medium containing 10% bovine serum. -antigens to SA7(C8) virus were found in two out f four mouse and three out of 12 rat tumors by omplement fixation tests using hamster sera. ne of 13 sera of the rats with tumors induced by A7(C8) virus contained SA7(C8) virus T-antigen ntibodies.

220 TOWARD CONTROL OF TUMOUR AND VIRUS GROWTH BY HELICAL RNA. II. DIFFERENCE IN THE OST-INTEGRATIONAL CONTROL OF VIRUS SYNTHESIS BY OLY I:C IN ACUTELY AND CHRONICALLY B77 VIRUS INFECTED ELLS. *(E.)* Svec, J. (Cancer Res. Inst., Bratislava, echoslovakia), M. Grofova and V. Thurzo. *eoplasma* 19(6):621-629, 1972.

ick embryo fibroblast cultures were treated with µg/ml polyriboinosinic:polyribocytidylic acid oly I:C) 16 hr or one hr before infection with atislava Rous sarcoma virus (B77). Cultures re treated with ^{3}H-5-uridine and radioactivity labeled virus particles was studied to elucidate e effect of Poly I:C on early virus replication in infected cells. Poly I:C 16 hr before B77 infection markedly suppressed B77 release into culture fluid and also suppressed virus group-specific (GS) antigen production. Poly I:C started one hour before infection did not affect B77 replication or GS antigen production. Poly I:C did not influence the penetration of labeled B77 into cells. Nor did Poly I:C suppress virus replication in chick embryo cells infected with the following viruses: BH-RSV, BH-RSV(RAV_1), BH-RSV(RAV_2) and SR-RSV. These viruses showed full genome expression by ten days after infection in Poly I:C treated cells. Poly I:C did not affect ^{3}H-6-thymidine uptake by infected cells. Treatment of B77-producing rat 17RB77/K_2S cells and RBH_{tc} hamster cells with Poly I:C did not affect virus release.

0221 A DNA FRAGMENT OF HERPES SIMPLEX 2 AND ITS TRANSCRIPTION IN HUMAN CERVICAL CANCER TISSUE. *(E.)* Frenkel, N. (Dept. Microbiol., U. Chicago, Ill.), B. Roizman, E. Cassai and A. Nahmias. *Proc Natl Acad Sci USA* 69(12):3784-3789, 1972.

A cervical tumor removed from a 59-yr-old woman was found to lack all sign of the presence of herpes simplex virus (HSV), types 1 or 2. Nevertheless, attempts were made to hybridize DNA from the tumor cells with HSV type 2 DNA, and it was found that the tumor contained a unique DNA fragment comprising about 39% (i.e., 39 x 10^6 daltons) of the HSV type 2 DNA molecule. Renaturation kinetics indicated that 1-3.5 DNA fragments of HSV were present in each tumor cell, the number of fragments present depending on the ploidy of the cells. Viral DNA sequences were linked to certain highly repetitive tumor cell DNA sequences which reassociated under conditions designed to exclude the reassociation of viral DNA. The tumor also contained RNA transcripts complementary to 5% of the viral DNA. About 5% of the viral DNA was apparently transcribed in the tumor cells, an amount much less than the 50% of viral DNA transcribed in productively infected cells.

0222 DENSITY HETEROGENEITY OF SIMIAN VIRUS 40 RIBONUCLEIC ACID LATE AFTER INFECTION OF PERMISSIVE CELLS. *(E.)* Fried, A. H. (German Cancer Res. Ctr., Heidelberg). *J Virol* 10(6):1236-1239, 1972.

Buoyant density analyses were performed on SV40 RNA molecules isolated from permissive monkey CV-1 cells 44 hr after infection with SV40. The density distribution of this RNA was compared to that of single-stranded and double-stranded RNA produced using SV40 DNA and *E. coli* RNA polymerase. It was found that a population of SV40 RNA molecules, comprising about 20% of SV40 RNA in infected cells, was present, and that this RNA had a density less than that of the single-stranded molecules. These were evidently partially double-stranded molecules.

0223 CLONAL ANALYSIS OF RBI RAT TUMOUR CELLS PRODUCING CHICKEN SARCOMA VIRUS. *(E.)* Altaner, C. (Slovak Acad. Sci., Bratislava, Czechoslovakia), E. Hlavayova, M. Hladka and F. Svec. *Neoplasma* 19(5):385-395, 1972.

Twenty single cell clones were established from cultures of the RBI rat tumor, an infectious virus-producing tumor induced by Bratislava Rous chicken sarcoma virus (B77). Infectious virus was produced, at a low level, by each clone. All clones contained the B77 viral genome. When cells were irradiated and fused with inactivated Sendai virus, fused cells produced foci on chicken cells, as did unfused clonal cells. The number of clonal cells, fused or unfused, required to induce foci on chicken cells did not differ greatly from clone to clone. There was no clear correlation between clonal cell morphology and ability to induce foci. Most clones were hyperdiploid, some were hypertetraploid. Cells from some clones induced tumors in rats when as few as 1,000 cells were injected; other clones needed a million cells or more to induce tumors on rats. Plating efficiencies of viruses rescued from clones were similar.

0224 COMPARATIVE STUDIES ON LIPID CONTENT AND LIPID METABOLISM OF NORMAL AND TRANSFORMED MOUSE CELLS. *(E.)* Kulas, H.-P. (Max-Planck Inst. Virus Res., Tübingen, Germany), W.-D. Marggraf, M. A. Koch and H. Diringer. *Hoppe-Seyler Z Physiol Chem* 353(11):1755-1760, 1972.

Lipids were examined in cultured normal and SV40-transformed mouse cells, mainly cells of the strain 3T3. Lipids were examined in cultures of different cell density to see how lipid composition correlated with phases of cell growth. Neutral and polar lipids did not differ in amounts in different cell lines. Among neutral lipids, however, triglyceride was increased in content in SV40-transformed cells relative to normal cells. Triglyceride did not increase during the exponential phase of cell growth, it was found, but did increase when cell numbers remained constant in the stationary phase of growth. Total phospholipid contents and cholesterol contents increased in parallel with the cell number. Pulse labeling experiments using ^{32}P-orthophosphate indicated that phospholipids incorporated radioactivity differently in sparse and dense cell cultures.

0225 CHARACTERIZATION OF CHICKEN SARCOMA VIRUSES INDUCED WITH CHEMICAL CARCINOGENS AND MUTAGENS IN TRANSFORMED HAMSTER CELLS. *(E.)* Altanerova, V. (Slovak Acad. Sci., Bratislava, Czechoslovakia) and C. Altaner. *Neoplasma* 19(5):405-412, 1972.

Cells of a hamster line transformed by Schmidt-Ruppin Rous sarcoma virus were treated with 5-azacytidine, 3-methylcholanthrene, 4-nitroquinoline-1-oxide or benzo(a)pyrene. Treated cells produced viruses which were assayed for DNA- and RNA-dependent DNA polymerase. All induced viruses contained both enzymes. When hamsters were given injections of preparations of chemically-induced viruses, it was found that viruses induced by 5-azacytidine were highly tumorigenic; viruses induced by the other agents were less tumorigenic. 5-Azacytidine-induced viruses showed increased RNA-dependent DNA polymerase relative to the other viruses, and were more efficient in transforming duck cells than were the other viruses.

0226 CHARACTERISTICS OF TWO STRAINS OF AVIAN SARCOMA VIRUS B77. *(E.)* Simkovic, D. (Internal. Agency Res. Cancer, Lyon, France). *Neoplasma* 19(5):365-370, 1972.

Two derivatives of B77 avian sarcoma virus, designated strain 22-B77V and strain 55-B77V, were subjected to testing for host range, virus-induced interference and envelope antigenic properties. Results were compared with the same parameters in avian leukosis virus of subgroups A, B, C and D. In tests for host range on different types of chick embryo fibroblasts (CEF), the 22-B77V virus behaved as a virus of subgroup B or D. Both 22- and 55-B77V showed significant decreases of infectivity (i.e., interference) when plated on Rous-associated virus-infected CEF. Tests of the antigenicity patterns of their viral envelopes, confirmed 22-B77V as a subgroup B or D virus, and suggested that 55-B77V belonged to subgroup C.

0227 DIFFERENT TUMORIGENICITY OF AVIAN RNA SARCOMA VIRUSES FOR SOME MAMMALIAN SPECIES. *(E.)* Smida, J. (Slovak Acad. Sci., Bratislava, Czechoslovakia) and V. Smidova. *Neoplasma* 19(5):397-404, 1972.

Newborn rats, hamsters and guinea pigs were inoculated with virus preparations from chick embryo cells transformed by either of the following eight viruses: Bratislava sarcoma virus (B77); Schmidt-Ruppin Rous sarcoma virus (SR RSV); Fujinami sarcoma virus; Mill Hill 2 endothelioma virus; Bryan standard strain of Rous virus; and Rous-associated viruses strains 1, 2 and 7. Rats and hamsters were inoculated s.c. with 10^6 transformed chick cells, while guinea pigs were given 5×10^6 cells. SR RSV caused tumors in five of seven rats, in all of eight hamsters and in all of three guinea pigs. B77 virus caused tumors in two of ten rats, in two of eight hamsters and in two of six guinea pigs. None of the other viruses caused tumors in any of the animals treated.

0228 TOWARD CONTROL OF TUMOUR AND VIRUS GROWTH BY HELICAL RNA: I. EFFECT OF POLY I:C ON TUMOUR TRANSPLANTATION RESISTANCE IN ANIMALS BEARING TUMOURS OF VIRAL AND NON-VIRAL ORIGIN. *(E.)*

vec, J. (Slovak Acad. Sci., Bratislava, Czecho-
lovakia), L. Novotna and V. Thurzo. *Neoplasma*
9(5):447-452, 1972.

ats were injected i.p. with polyinosinic-polycytidy-
ic acid (Poly I:C, 100 µg) one day before, and
gain on two occasions after, i.p. implantation of
RBI rat ascites sarcoma (induced originally by
7 chicken sarcoma virus). Poly I:C administered
.p. fully protected inoculated rats against the
I tumor, even when tumor inocula reached 8×10^6
lls. However, Poly I:C i.p. did not protect
ainst i.m. transplantation of RBI cells. Poly
C, given to tumor-bearing hamsters, did not affect
ther spleen wt or *in vitro* cytotoxicity of
mphocytes for syngeneic tumor cells. Nor did
ly I:C affect the *in vitro* growth rate of rat
d hamster tumors.

29 COMPUTER ASSISTED ANALYSIS OF A CARRIER CULTURE INFECTED WITH MOLONEY LEUKEMIA IRUS.

(E.) ter Meulen, V. (U. Clin., Göttengen,
rmany), P. H. Bartels, G. F. Bahr, M. Bibbo,
Cremer, E. H. Lennette and G. L. Wied. *Acta
tol* 16(5):454-463, 1972.

t thymus cells in tissue culture were infected with
loney leukemia virus (MLV) and stained with fluores-
n-labeled rat anti-MLV serum. To distinguish in-
cted cells from uninfected cells the fluorescence
tterns in cultures were scanned by a recording
crospectrophotometer and data on distribution of
uorescence were subjected to computer analysis
r intracellular patterns of scanned fluorescence.
sults showed statistically significant differences
tween uninfected and infected cells. The average
kelihood for correct recognition of infection
a single cell from an infected culture was 3.3:1.

30 THE CARBOHYDRATE CONTENT OF CONTROL AND VIRUS-TRANSFORMED CELLS.

(E.)
rtmann, J. F. (Wistar Inst., Philadelphia, Pa.),
A. Buck, V. Defendi, M. C. Glick and L. Warren.
Cell Physiol 80(2):159-166, 1972.

lls from various sources were infected with Bryan
Schmidt-Ruppin Rous sarcoma virus, polyoma virus,
SV40 and carbohydrate contents in transformed
lls and in the cell wash medium were measured
gas-liquid chromatography. In SV40- and polyoma-
ansformed hamster cells, a decrease in sugar con-
ntrations was seen in cells. However, in the case
D-mannose and D-galactose, levels in the wash
re high, making the whole system (i.e., cells
us wash) nearly equal to untransformed cells in
ncentrations of these compounds. L-fucose was
creased in transformed cells, even when the cell
sh was taken into account. The sialic acid content
polyoma-transformed cells was equal to that in
transformed cells, while SV40-transformed cells
owed lowered sialic acid contents. In Rous
rus-transformed chicken cells, L-fucose levels
re markedly reduced, while D-mannose and the N-
etylhexosamines were scarcely affected.

0231 KINETICS OF INCORPORATION OF (^{32}P) PHOSPHATE INTO PHOSPHOLIPIDS OF A SV 40 TRANSFORMED MOUSE CELL DURING LOGARITHMIC GROWTH.

(E.) Marggraf, W.-D. (Max-Planck Inst. Virus Res., Tübingen, Germany), H. Diringer, M. A. Koch and F. A. Anderer. *Hoppe-Seyler Z Physiol Chem* 353(11):1761-1768, 1972.

STU-51A/232B mouse fibroblasts, from an SV40-transformed line, were grown logarithmically in the presence of (^{32}P)phosphate. Cells were given a 12 hr labeling period followed by a 48 hr chase; after the chase phospholipids were isolated and separated by chromatography and their kinetics of ^{32}P incorporation were studied. Maximal amounts of ^{32}P in phosphatidic acid and in phosphatidylinositol were seen at the time of the chase, while cardiolipin, phosphatidylserine, sphingomyelin and phosphatidylethanolamine had their maxima of ^{32}P uptake more than one cell cycle after the chase. Phosphatidic acid, phosphatidylinositol, cardiolipin, lecithin and phosphatidylethanolamine achieved a constant rate of ^{32}P uptake during the first hours after ^{32}P labeling. Sphingomyelin and phosphatidylserine uptake of ^{32}P reached constant rates only after a 6 hr lag period. It was thought that the formation of the latter two compounds proceeded via a pathway which has not yet been investigated.

0232 GLYCOLIPIDS OF MOUSE FIBROBLASTS AND VIRUS TRANSFORMED MOUSE CELL LINES.

(E.) Diringer, H. (Max-Planck Inst. Virus Res., Tübingen, Germany), G. Ströbel and M. A. Koch. *Hoppe-Seyler Z Physiol Chem* 353(11):1769-1774, 1972.

Normal and SV40-transformed 3T3 mouse cells, and normal and Friend virus-transformed STU mouse cells, were examined for glycolipid content. Hematoside (G_{M3}), a precursor in the biosynthesis of the higher ganglioside G_{D1a}, was found to be drastically decreased shortly after transformation of 3T3 cells by SV40. G_{D1a} itself remained normal just after SV40 transformation, but decreased with successive tissue culture passages after transformation. G_{M3} content depended on cell population density in normal and in SV40-transformed cells. In Friend-transformed cells, G_{M3} was increased twofold above its level in normal cells. Neutral glycolipids. like G_{M3}, were decreased in SV40-transformed cells and increased in Friend-transformed cells.

0233 DETECTION OF SOME MURINE LEUKEMIA VIRUS ANTIGENS IN VIRUS PARTICLES DERIVED FROM 3T3 CELLS TRANSFORMED ONLY BY MURINE SARCOMA VIRUS.

(E.) Fischinger, P. J. (Natl. Cancer Inst., Bethesda, Md.), W. Schäfer and E. Seifert. *Virology* 47(1):229-235, 1972.

Mouse 3T3 cells were transformed by murine sarcoma virus and isolated as a soft agar colony; these were called "sarcoma positive, leukemia negative (S^+L^-)" cells. A report on the detection of several antigens in S^+L^- cells and virus is presented. Murine leukemia virus (MuLV) antigens were first found in S^+L^- cell extracts and supernates using complement fixation (CF) tests with R-gs serum after a high concentration of virus-containing fluids. Immunoprecipitations tests were used to identify the antigens of the S^+L^- particle which reacted in the CF tests. The presence of a MuLV "gs-spec." antigen (IV) and a "gs-interspec." antigen (V) in S^+L^- cells and virus was confirmed when the purified gs-antigen preparation was reacted with S^+L^- virus and R-gs and G sera. Because S^+L^- virus activity could be measured by hemagglutination of sheep red blood cells, an indirect means of determining S^+L^- virus envelope specificity could be used. Ether-inactivated S^+L^- virus could not absorb anti-GLV or anti-FLV-RLV neutralizing antibodies. It is suggested that the envelope antigenicity of S^+L^- virus is quite different from that of standard MuLV strains.

0234 CELL LINE DERIVED FROM BALB/3T3 THAT IS TRANSFORMED BY MURINE LEUKEMIA VIRUS: A FOCUS ASSAY FOR LEUKAEMIA VIRUS.

(E.) Hackett, A. J. (Sch. Publ. Hlth., U. California, Berkeley) and S. S. Sylvester. *Nature New Biol* 239(93):164-166, 1972.

A new cell line, designated UC1-B, has been derived from Balb/3T3 cells. The line consisted of polygonal cells which grow as flat, smooth monolayers and which are contact inhibited. Unlike the standard Balb/3T3 line, UC1-B cells could be morphologically transformed by murine leukemia virus (MuLV). The UC1-B cells yielded MuLV after infection but did not produce sarcoma virus. Transformed cells lost contact inhibition and foci of piled-up cells developed on the monolayer. Three different stock pools of MuLV and one of Rauscher leukemia virus produced morphologically identical transformed foci. Infected UC1-B cells yielded typical type-C virus as detected by ^{3}H-uridine labeling, RNA-directed DNA polymerase assay, electron microscopy, and infectivity assays. In addition to typical C-type budding virus, bizarre forms were also observed in separate vacuoles within the same cell. Whether this represented defective MuLV replication or defective rescue of a latent sarcoma virus genome was unclear. The production of transformed foci on UC1-B monolayers was more rapid than, and as efficient as, XC-plaques on Balb/3T3.

0235 THE MECHANISM OF TRANSFORMATION OF HUMAN DIPLOID CELLS BY A LEUKEMIA-LIKE VIRUS. INCREASING THE TRANSFORMING ACTIVITY OF THE VIRUS BY 2,4-DINITROPHENOL.

(Rus.) Andzhaparidze, O. G. (Moscow Res. Inst. Viral Prep. USSR.), G. D. Zasukhina, L. G. Stepanova and L. L. Matusevich. *Vopr Virusol* 17(4):435-436, 1972.

Human diploid cells were infected with a leukemia-like virus isolated from a patient with hemocytoblastosis. Dinitrophenol 10^{-5} M, a DNA repair inhibitor, was added to an Eagle's medium, which was then added to the infected cells. Enzyme activities of the cultured cells were evaluated by the capacity of the cells to separate thymine dimers, formed by UV irradiation, in postirradiation conditions of cell cultivation. Uninfected control cells were capable of separating almost 50% of the formed thymine dimers 24 hr after irradiation, while the leukemia-like virus-infected cells lost the capacity to eliminate thymine dimers 12 hr post infection. The infected human diploid cells were transformed in the presence of 2,4-dinitrophenol in earlier periods than without the DNA repair inhibitor. The decrease in repair activity in the cells in the presence of an inhibitor was accompanied by intensified transformation of the cells due to the presence of RNA and DNA-containing oncogenic viruses. It is assumed that compounds promoting the activity of repair enzymes could exert an inhibition of cell transformation.

0236 PROTECTIVE EFFECT OF IMMUNE SERA AGAINST TRANSPLANTABLE MOLONEY VIRUS-INDUCED SARCOMA AND LYMPHOMA.

(E.) Pearson, G. R. (Natl. Cancer Inst., Bethesda, Md.), L. W. Redmon and L. R. Bass. *Cancer Res* 33(1):171-178, 1973.

Sera from mice immunized with Moloney sarcoma virus (MSV)-induced antigens were quantitated for antibodies by indirect membrane immunofluorescence (MF), cytotoxicity, and neutralization. These sera were then tested *in vivo* to determine whether antibody titers measured by these three methods would correlate with the effectiveness of the serum in preventing the growth of a transplantable Moloney virus-induced sarcoma (MSC) or lymphoma (LSTRA). The sources of sera were (a) BALB/c mice with progressively growing MSV-induced tumors (progressor serum); (b) BALB/c mice in which MSV-induced tumors regressed (regressor serum); and (c) BALB/c X DBA F_1 mice immunized with multiple inoculations of MSV-induced allogeneic tumor cells (C57BL origin; allogeneic serum). The pooled progressor sera showed low antibody titers in all three tests. In contrast, the pooled regressor and allogeneic sera were positive in the MF and neutralization tests, although neither showed any cytotoxic activity. The MF and neutralization titers with the allogeneic serum were 2 to 4 times higher than with the regressor serum. Pretreatment of recipient mice with regressor or allogeneic sera as early as 3 days before challenge with MSC tumors resulted in (resp.) 60 and 84% survivors free of disease when the experiments were terminated at 75 days. In contrast, resistance was not noted following pretreatment with the progressor sera. The allogeneic serum was also effective against the rapidly growing transplantable Moloney leukemia virus-induced lymphoma designated LSTRA. Single injections of this serum in mice either 3 or 1 day before LSTRA challenge or 2 days after challenge resulted in significant numbers of survivors during the period of observation. Multiple injections of this serum during the latent period for tumor development

esulted in up to 90% survivors, free of apparent
isease, when the experiments were terminated at
) days. These results demonstrate that the
ffectiveness of a serum in these systems in pre-
enting tumor growth correlates closely with its
F and neutralization titers and suggests that
easurement of antibody titers by conventional
ethods, particularly MF, might provide useful
nformation on the potential therapeutic value of
erum in the control of cancer.

237 BRAIN TUMORS INDUCED BY ROUS SARCOMA VIRUS,
SCHMIDT-RUPPIN STRAIN. III. MORPHOLOGY
F BRAIN TUMORS INDUCED IN ADULT MICE. *(E.)*
umanishi, T. (Brain Res. Inst., Niigata U.,
apan), F. Ikuta and T. Yamamoto. *J Natl Cancer
ıst* 50(1):95-109, 1973.

orphologic examination was made on 110 brain tumors
nduced in adult mice by intracerebral inoculation
ith chicken sarcoma cells producing Schmidt-Ruppin
rain of Rous sarcoma virus. It was found that the
lioma analogous to the human ordinary gliomas (glio-
lastoma multiforme and astrocytoma) developed in
dult mice after a long latency. Frequency was 26%
 the 110 tumors and was about 50% of the tumors
hat developed more than 90 days after inoculation.
he tumors composed predominantly of huge round cells
ere most common throughout the observation period of
)0 days and were tentatively designated "huge round-
elled tumors." Frequency was about 70% of the 110
umors and about 50% of the tumors developing after
) days. The glial origin of the huge round-celled
umor was suggested from its light and electron-micro-
copical characteristics. Either coexisting with these
o characteristic tumors or forming independent or
iscrete tumors, large elongated, neurinoma-like
or spongioblastic), and small spindle cells were
lso noted. Emphasis was given to the successful
nduction of the typical glioma in adult mice as
ell as to the frequent development of a characteris-
ic huge round-celled tumor that was composed
redominantly of huge round cells and resembled
uman giant-celled glioblastoma. Biochemical
nd biologic evidence for the mouse, not chicken,
rigin of each brain tumor is also discussed.

238 *IN VITRO* STIMULATION OF HUMAN LYMPHOCYTES
BY EPSTEIN-BARR VIRUS. *(E.)* Gerber, P.
Natl. Inst. Hlth. Bethesda, Md.) and S. J. Lucas.
ell Immunol 5(2):318-324, 1972.

he ability of Epstein-Barr virus (EBV) to stimulate
uman peripheral blood lymphocytes was investi-
ated. Lymphocytes from five EBV-seronegative donors
howed marked stimulation of DNA synthesis, as
easured by the rate of ^{3}H-thymidine incorporation,
ollowing *in vitro* infection with infective EBV,
ut showed no response to UV-irradiated, noninfec-
ious EBV. However, lymphocytes from ten EBV-
eropositive donors were stimulated equally with
oth infective and UV-inactivated EBV. The antigenic
stimulation was immunologically specific. Antibody that was demonstrably effective in neutralizing EB virus infectivity did not block antigenic stimulation. The pattern of the stimulatory response differed between sensitized, unsensitized, and EBV-infected lymphocytes. Whereas sensitized lymphocytes first showed stimulation on day 4, reached a peak at about 6-8 days, and thereafter declined, unsensitized lymphocytes showed a stepwise increase in ^{3}H-thymidine incorporation during the 12 days of observation. EBV-infected lymphocytes became permanently transformed, showing a high degree of DNA synthesis.

0239 EARLY INCREASE IN NUCLEAR ACIDIC PROTEIN SYNTHESIS AFTER SV40 INFECTION. *(E.)* Rovera, G. (Temple U. Sch. Med., Philadelphia, Pa.), R. Baserga and V. Defendi. *Nature New Biol* 237(77):240-241, 1972.

The synthesis of total cellular proteins and nuclear acidic proteins was studied in permissive green monkey kidney (CV1) and nonpermissive Chinese hamster embryo (ChH) cells during the first hours after infection, with SV40. Within 48 hr after SV40 infection, essentially all ChH cells were positive for T antigen and the percentage of cells synthesizing DNA as determined by autoradiography was greater than that in fresh medium-stimulated non-infected ChH cells. In both SV40 infected ChH and CV1 and in mock-infected, stimulated cells, ^{3}H-leucine incorporation into total acid insoluble material was decreased compared to mock-infected, non-stimulated cells. However, incorporation into the residual acidic protein fractions of ChH and CV1 cells was stimulated to the same levels as those in mock-infected fresh medium-stimulated cells. The analogy between virus-induced changes and serum-induced changes in nuclear acidic protein synthesis suggested that these two stimuli share a common mechanism in stimulating DNA synthesis.

0240 A POSSIBLE MECHANISM OF RNA REPLICATION IN ROUS SARCOMA VIRUS. *(E.)* Ogura, H. (Okayama U. Med. Sch., Japan) and T. Oda. *Acta Med Okayama* 25(5):567-571, 1971.

A possible mechanism for the replication of Rous sarcoma virus (RSV) RNA has been proposed based upon electron microscopic observations of purified RSV RNA and RSV-transformed chick cells. Partially separated double strands of RSV RNA were observed in viral RNA preparations purified on sucrose density gradients. In addition, RSV with two nucleoids which were elongated and ovoid in shape could occasionally be seen in the extracellular spaces of RSV-transformed chick cells, and separated RSV particles frequently occurred in pairs. Based upon these observations, a mechanism of RSV genome replication was suggested which involves the transcription of a DNA intermediate (minus strand) from the infecting RSV RNA genome (plus strand) followed by transcription of double stranded RSV-specific RNA from the DNA intermediate.

0241 ONCORNAVIRUS TYPE B ISOLATED FROM HUMAN LARYNGEAL CARCINOMA CELLS. *(Rus.)*

Il'in, K. V. (N. F. Gamaleya Inst. Epidemiol. Microbiol., Acad. Med. Sci. USSR, Moscow), A. F. Bykovskiy and V. M. Zhdanov. *Vopr Virusol* 17(4):494-499, 1972.

A virus isolated from a transplantable cell line, referred to as HE_p-2, of a human laryngeal carcinoma was subjected to immunologic, electron microscopic, and biochemical studies. Three hundred metaphase plates prepared from these HE_p-2 cell cultures revealed the human chromosome characteristics. Mice (25) C3H/Sn receiving 10 million HE_p-2 cells s.c. did not develop tumors for the four months observed. Newborn hamsters (16) receiving 5 million HE_p-2 cells did not show tumors for the 8 months studied. Tumors did not occur in BALB/c mice 16 months after infection with 0.1 ml virus suspension injected s.c.. The isolated virus had a flotation density of 1.16 g/ml in sucrose gradients, corresponding to the density of oncorna viruses. The virus was not observed in cultures after the cultures had been treated with actinomycin D (1 mkg/ml). In an electron microscopic study, the virus population was found to consist of two virion types A and B. The purified virions showed a reverse transcriptase activity, with the reaction products of DNA-RNA hybrids and double helical RNA. In an immunologic study, antisera to the virus antigen did not react with the mouse mammary gland carcinoma virus antigens. The isolated virus could be considered as a possible pathogenic factor in human tumors.

0242 RESTRICTED HOST RANGE OF A FELINE LEUKAEMIA VIRUS. *(E.)*

Jarrett, O. (Animal Leukaemia Res. Unit, Glasgow, Scotland), H. M. Laird and D. Hay. *Nature* 238(5361):220-221, 1972.

Feline leukemia virus (FeLV) types 1 and 5 were isolated from cats with alimentary lymphosarcomas and used to infect feline embryonic cells (FEA) and seven cultures of human embryonic lung cell (HEL). Leukemia virus particles were seen in FEA cells inoculated with FeLV-1 or FeLV-5 and in the seven HEL cell lines inoculated with FeLV-5; no virus was seen in any HEL cell lines inoculated with FeLV-1. Pseudotypes of Moloney murine sarcoma virus (MSV) with FeLV-1 or FeLV-5 envelopes MSV(FeLV-1) or MSV(FeLV-5) were produced and inoculated on FEA or HEL cells. MSV(FeLV-1) and MSV(FeLV-5) transformed FEA cells but only MSV(FeLV-5) transformed HEL cells. These results suggest that it is probably the viral envelope which mediates the ability of FeLV to infect human cells.

0243 EVIDENCE FOR A TRANSCRIPTION-CONTROL REGION OF SIMIAN VIRUS 40 IN THE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID, $Ad2^+ND_1$. *(E.)*

Patch, C. T. (Natl. Cancer Inst., Bethesda, Md.), A. M. Lewis, Jr. and A. S. Levine. *Proc Nat Acad Sci USA* 69(11):3375-3379, 1972.

DNA from the $Ad2^+ND_1$ hybrid virus of SV40 and adenovirus 2 (Ad2) was analyzed on a CsCl gradient. The complementary DNA strands were found to separate. The separated strands were used in DNA-RNA hybridization studies with RNA from Ad2- or SV40-infected monkey cells. The light strand of $Ad2^+ND_1$ DNA hybridized five times as much Ad2 RNA as did the heavy strand. Complementary RNA and SV40 RNA synthesized before viral DNA replication showed significant homology only with the light strands of $Ad2^+ND_1$ DNA. The light strand, however, hybridized only half as much of a preparation of RNA synthesized before and after DNA replication as did the heavy strand. Results suggested that the SV40 moiety in $Ad2^+ND_1$ contained a template for late RNA as well as for early RNA; therefore, the SV40 segment in $Ad2^+ND_1$ must contain a transcription-control region.

0244 SPECIFIC INITIATION SITE FOR SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID REPLICATION. *(E.)*

Thoren, M. M. (Natl. Inst. Allergy Infec. Dis., Bethesda, Md.), E. D. Sebring and N. P. Salzman. *J. Virol* 10(3):462-468, 1972.

Low molecular wt (LMW) SV40 DNA from infected monkey cells was isolcated and labeled with ^3H-thymidine so as to produce uniformly labeled newly synthesized DNA in the replicative intermediate molecules. The labeled LMW DNA fraction was layered on alkaline sucrose gradients and centrifuged. By this means, single-stranded DNA was isolated which ranged in molecular wt from 157,000 daltons (i.e., molecules 10% replicated) to 1,360,000 daltons (i.e., 85% replicated molecules). Newly synthesized single stranded DNA isolated as above was reannealed. It was found that the time required to effect 50% reassociation of the DNA increased at a rate proportional to the increasing size of the DNA. Results were taken to indicate that DNA replication was not initiated randomly, but that there was a single specific DNA replication initiation site.

0245 TRANSFORMATION OF SPLEEN CELLS THREE HOURS AFTER INFECTION *IN VIVO* WITH FRIEND LEUKEMIA VIRUS. *(E.)*

Rossi, G. B. (Sch. Med., State U. New York, Buffalo), G. Cudkowicz and C. Friend. *J Natl Cancer Inst* 50(1):249-254, 1973.

Three hours after DBA/2 mice were infected with Friend leukemia virus (FLV), hemopoietic cells in the spleen were transformed into neoplastic cells as judged by two parameters: 1) the acquisition of autonomous growth potential on intravenous transplantation into nonirradiated histocompatible mice; 2) the serial transplantability in subcutaneous as well as hemopoietic sites. Both criteria sharply distinguished transformed cells from normal hemopoietic cells which do not grow in nonirradiated mice and in the subcutaneous tissue. Transformation occurred in the initially infected hosts and not after spleen cell transplantation by way of virus carried in the inoculum. Infected ells killed by sonic disruption or freezing and hawing failed to induce transformation; cells arvested after < 3 hours of exposure to FLV were ot transformed; the pattern of hybrid resistance f (C57BL/6 X DBA/2)F_1 and (BALB/c X DBA/2)F_1 mice gainst the DBA/2 cells harvested three or more ours after infection was similar to that displayed gainst normal hemopoietic and leukemic DBA/2 cells; nd finally, the Fv-1 and Fv-2 genotype of the ybrids, which control susceptibility to infection, as not consistent with the possibility that the ransformed cells were of host (F_1) rather than donor DBA/2) origin. The acquisition of autonomous rowth potential and increased susceptibility to ybrid resistance (i.e., altered expression of hybrid-istocompatibility genes) are both early events in riend virus-induced leukemogenesis, and the asso-iation might be significant.

246 ENHANCEMENT OF MURINE LEUKEMIA VIRUS REPLICATION IN RAT TUMOR CELLS CONTAINING AT C-TYPE VIRUS. *(E.)* Turner, W. (Natl. Cancer nst., Bethesda, Md.), G. Pearson and L. R. Bass. *ppl Microbiol* 24(5):837-839, 1972.

ne replication of Maloney leukemia virus (MLV) as studied in several C-type virus-infected and oninfected rat cell lines. The MLV titer of LV-infected rat cells containing rat C-type iruses, as determined by the XC plaque assay echnique, was greater than that of MLV-infected at cells free from C-type virus at both three and ix days after infection. The enhanced MLV eplication in C-type virus-containing cells was elated to the presence of C-type virus and not the enhanced metabolic activity of these cells, ince an increased MLV titer was not observed in at C-type virus-free tumor cells infected with LV. The MLV progeny from the MLV-infected rat mor cells showed the same replication pattern mouse embryo fibroblast cells as did murine-erived MLV. In addition, rat tumor cell-derived LV was able to induce leukemia in mice which as histologically identical to that produced by urine MLV infection. Membrane immunofluorescence udies indicated that the rat c-type virus con-ent of the rat tumor lines was not changed by LV infection.

247 EARLY LESIONS IN CATS INOCULATED WITH FELINE LEUKEMIA VIRUS. *(E.)* Hoover, A. (Coll. Vet. Med., Ohio State U., Columbus), E. Perryman and G. J. Kociba. *Cancer Res* 33(1): 5-152, 1973.

rphological alterations in the thymus, lymph odes, and bone marrow were determined at two week tervals in 21 cats inoculated at birth with feline ukemia virus and in 19 age-matched control cats. vere thymic atrophy was detected at five weeks age. Thymic atrophy was due primarily to ex-nsive depletion of cortical thymocytes while the dullary components of many thymic lobules persisted. Apparent stages in the development of lymphosarcoma from atrophic thymuses were detected in cats examined between 11 and 19 weeks of age. Lymphosarcoma was detected as early as nine weeks in some cats, by examination of bone marrow biopsies.

0248 ACTIVATION OF A TYPE-C RNA VIRUS FROM TUMORS INDUCED BY RAT KIDNEY CELLS TRANSFORMED BY A CHEMICAL CARCINOGEN. *(E.)* Rhim, J. S. (Microbiol Assoc., Bethesda, Md.), H. Y. Cho, E. Elder and M. L. Vernon. *J Natl Cancer Inst* 50(1): 255-261, 1973.

A cell line derived from Fischer rat kidney was treated with 7,12-dimethylbenz[a]anthracene (DMBA) or dimethyl sulfoxide (control). Cells treated only with the carcinogen underwent morphologic transformation *in vitro* and produced progressively growing transplantable tumors when injected into homologous hosts. Although the transformed cell line was negative for infectious virus before inoculation into animals, a type-C RNA virus was activated from a cell line derived from the tumors. Since infectious type-C viruses are not usually demonstrable in rat tissues of normal or tumor origin, it is suggested that the chemical treatment and activation of the virus may be related.

0249 BIDIRECTIONAL REPLICATION OF SIMIAN VIRUS 40 DNA. *(E.)* Danna, K. J. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and D. Nathans. *Proc Nat Acad Sci USA* 69(11):3097-3100, 1972.

Monkey cells infected with SV40 were pulse-labeled with ^{3}H-thymidine and labeled, covalently-closed SV40 DNA I was purified from cells. Labeled DNA I was mixed with uniformly-labeled SV40 ^{32}P-DNA I and the mixture was digested with *Hemophilus influenzae* restriction endonuclease. The distribution of label in newly synthesized SV40 DNA molecules was studied by analyzing fragments produced by endonuclease. The temporal order of synthesis of these fragments was determined and compared with the order in which fragments were located on the SV40 DNA molecule. It was concluded that, in SV40, DNA replication begins at a specific site on the molecule and proceeds in both directions around the circular molecule, replication proceeding at about the same rate in each direction.

0250 REPLICATION OF RADIATION-INDUCED MURINE LEUKEMIA VIRUS IN NORMAL AND TRANSFORMED MOUSE CELLS. *(E.)* Nomura, S. (Natl. Cancer Inst., Bethesda, Md.), R. H. Bassin and P. J. Fischinger. *J Virol* 9(3):494-502, 1972.

Radiation-induced leukemia virus (RadLV) was characterized in 3T3 mouse cells and in murine sarcoma virus (MSV)-transformed, leukemia virus-negative (S+L-) mouse cells. RadLV could be detected in these cells by the helper virus assay with defective MSV. RadLV interfered with MSV; when RadLV-infected cells were challenged with MSV, they quickly became resistant to MSV infection. When S+L- cells were infected with RadLV, foci appeared five to six days later. Chronically RadLV-infected 3T3 and S+L- cell lines were established. Virus progeny were seen after the second passage of RadLV-infected S+L- cells. When RadLV was subjected to sucrose density gradient fractionation, the infectivity and the helper activity of the virus were found in the region of 1.16 g/cm^3 density. A soluble inhibitor of MSV focus formation could be isolated from infectious RadLV. Sublines derived from a single RadLV-infected 3T3 cell produced RadLV and inhibited MSV. It is suggested that RadLV represents a virus population of varying replicative potentials, the predominant component of this population being deficient growth in normal cells.

0251 POLYOMA VIRUS BASIC PROTEINS. *(E.)* Frearson, P. M. (Imperial Cancer Res. Fund Lab., London, England) and L. V. Crawford. *J Gen Virol* 14:141-155, 1972.

Polyoma virus-associated polypeptides were studied in infected secondary mouse embryo cultures by incorporation of isotopically labeled lysine or methionine. SDS phosphate gel electrophoresis of lysine-labeled virus protein gave rise to six peaks. Methionine-labeled virus showed the same peaks with the exception of polypeptide 5. Lysine-labeled histones from mouse embryo cells migrated with virus polypeptides 4, 5 and 6 which had a molecular weight between 15,000 and 20,000. Virus produced by cells prelabeled with a pulse of radioactive lysine showed labeling of the various polypeptides, but the pattern of labeling differed from that obtained by labeling after infection. An excess of label over that expected in the small polypeptides indicated that they were probably made before infection. Polyacrylamide gel electrophoresis and thin-layer chromatography indicated that the two major and several minor tryptic peptides from methionine-labeled acid-soluble virus polypeptides were identical. It is concluded that these polypeptides are therefore not virus-coded.

0252 ANTIGEN EXPRESSION AND COLONY FORMATION OF LYMPHOBLASTOID CELL LINES AFTER SUPERINFECTION WITH EPSTEIN-BARR VIRUS. *(E.)* Rocchi, G. (Children's Hosp. Philadelphia, Pa.), J. Hewetson, W. Henle and G. Henle. *J Natl Cancer Inst* 50(2):307-314, 1973.

The susceptibility of 21 lymphoblastoid cell lines to superinfection with Epstein-Barr virus (EBV) was examined through tests of EBV-specific immunofluorescence and colony formation in soft agar. Intracellular EBV-related antigen-producing cells after superinfection were generally most frequent in cell lines with little or no spontaneous antigen expression. Lines showing the best degree of viral adsorption seemed most susceptible. Cells that synthesized D and R antigen components of the EBV-induced early antigen complex always exceeded substantially those that also produced viral capsid antigens; therefore most cells sustained abortive infections. That the superinfecting virus could influence colony formation was taken as a measure of cytopathogenicity of EBV. Cell lines with the greatest susceptibility, as measured by immunofluorescence, also showed the greatest cytopathic effects. Reduction in colony formation was observed, however, in some lines without accompanying marked increases in antigen-producing cells; thus the two tests measure distinct viral properties. There was no correlation between the origins of the cell lines and susceptibility to superinfection.

0253 CELL LOCOMOTION AND CONTACT INHIBITION OF NORMAL AND NEOPLASTIC RAT CELLS. *(E.)* Veselý, P. (Inst. Expt. Biol. Genet., Prague, Czechoslovakia) and R. A. Weiss. *Int J Cancer* 11(1):64-76, 1973.

The morphology, locomotion and contact inhibition of locomotion of a normal embryonic rat fibroblast cell line (LWF), a spontaneously neoplastic variant (LW13), and a Rous sarcoma virus transformed line (RsK4) were studied in relation to oncogenicity. Both the LWF and LW13 lines were fibroblastic; the RsK4 line was epithelioid. All three lines grew as monolayers with the RsK4 cells attaining the highest, and the LWF cells the lowest densities. Scanning electron micrographs revealed a smooth exposed surface on LWF cells, a moderate number of microvilli on the exposed surface of the LW13 cells, and numerous microvilli, blebs and ruffles on the exposed surface of the RsK4 cells. Both LWF and LW13 cells exhibited polarized locomotion, moving with broad, fan-shaped lamellae and ruffling at the leading edge, and migrating rapidly into wounds made in confluent monolayers. RsK4 cells neither possessed large, fan-shaped lamellae, nor showed polarized movement, nor migrated efficiently into wounds. Time-lapse cinemicrography showed that LWF and LW13 cells were both sensitive to cell-cell contact, exhibiting paralysis of ruffling, contraction of the leading edge, cell to cell adhesion, and inhibition of pinocytosis. RsK4 cells did not display paralysis or contraction. In mixed cultures, collisions between LWF and either LW13 or RsK4 cells resulted in inhibition of cell locomotion and membrane ruffling only in the LWF cells. The responses to cell to cell contact in mixed cultures seemed to be more closely related to oncogenicity than were the responses to homotypic (non-mixed culture) contact. Glutaraldehyde-fixed LWF cells had no inhibitory effect on live LWF, LW13 or RsK4 cells placed upon them. Fixed LW13 cells inhibited the movement of LW13 and LWF cells, and fixed RsK4 cells inhibited all three cell types.

0254 REPLICATION OF SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID: ANALYSIS OF THE ONE-STEP GROWTH CYCLE. *(E.)* Manteuil, S. (Inst. Sci. Res. Cancer, Villejuif, France), J. Pages, D.

tehelin and M. Girard. *J Virol* 11(1):98-106,
973.

ne time course of replication of SV40 DNA was
tudied in growing monolayer cultures of subcloned
71 cells. Sucrose density centrifugation of
urified viral DNA extracted from continuously ^{3}H-
ymidine-labeled, infected cells indicated that
V40 DNA first appeared (component 1) about 11 hr
fter infection. Pulse label experiments showed
nat the rate of SV40 DNA synthesis constantly
ncreased from that time on to about the 20th hr
fter infection when it plateaued, remaining
onstant for an additional 16-20 hr. Replication
viral DNA ceased after 40 hr. Under these
onditions, the duration of the virus growth
rcle, as determined by the extent of cell lysis,
s 50 hr, and the time necessary for synthesis
one DNA molecule was estimated at 15 min or
ss. These growth kinetics were somewhat
pendent upon the multiplicity of infection.
though there were no major differences within
e range of 6 to 60 PFU per cell, the time
quired to reach the linear phase of DNA synthesis
s increased by four to six hr with a multipli-
ty of infection of 1 and lower. Even at 1 PFU
r cell, however, the final rate of DNA synthesis
s the same as that observed at 60 PFU per cell and
s, therefore, independent of the input multi-
icity. These results were interpreted to
dicate that the templates for the synthesis
viral DNA synthesis multiply in the cell during
e early phase of replication.

55 PRODUCTION AND CHARACTERIZATION OF
HUMAN CELL-ADAPTED MURINE RAUSCHER VIRUS
EUDOTYPE OF MURINE SARCOMA VIRUS. *(E.)*
lashi, D. V. (Natl. Cancer Inst., Bethesda,
.), G. R. Armstrong and W. Turner. *J Natl
ncer Inst* 50(2):381-385, 1973.

pseudotype of Moloney murine sarcoma virus (M-MSV)
s produced *in vitro* by cocultivation of human em-
yonic kidney cells persistently infected with Rau-
ner murine virus propagated in human cells (HRV),
nproducer hamster cells transformed by M-MSV,
a continuous line of uninfected human embryonic
iney cells. The pseudotype sarcoma virus [MSV
RV)] was capable of inducing focus formation
WI-38 and primary human embryonic kidney
lls (P-HEK). Rescue of the pseudotype virus
m the mixed cultures was successful as early as
days. The virus was effectively neutralized
rabbit anti-HRV serum. MSV (HRV) neither
fected monkey or mouse cells in culture nor
oduced tumors in newborn NIH Swiss mice and ham-
ers. An excess of leukemia virus in MSV (HRV)
s evidenced by colony-forming activity at a
lution of virus where focus formation was not
oduced in WI-38 and P-HEK cells. The addition
various concentrations of HRV in WI-38 cultures
fected with various dilutions of MSV did not
nance focus formation, indicating a one-hit
pe of kinetics.

0256 TRANSPLANTATION AND CYTOGENETIC STUDIES OF *HERPESVIRUS SAIMIRI*-INDUCED DISEASE IN MARMOSET MONKEYS. *(E.)* Marczynska, B. (U. Illinois Med. Ctr., Chicago), L. Falk, L. Wolfe and F. Deinhardt. *J Natl Cancer Inst* 50(2):331-336, 1973.

The response of marmosets to transplanted tumor tissues from *Herpesvirus saimiri* (HVS)-infected marmosets was studied. Recipient marmosets developed lymphoproliferative disease indistinguishable from the disease produced in marmosets by experimental infection with cell-free HVS. Clinical features of the disease in recipient marmosets were similar to those found earlier: HVS was isolated from peripheral lymphocytes by cocultivation methods, all marmosets developed HVS serum antibodies, and marmosets died from their disease 26-67 days postinoculation. Cytogenetic analysis of peripheral leukocytes of recipient marmosets indicated that the lymphoproliferative disease was a result of transformation *in vivo* of recipient lymphocytes and not of growth of donor tumor cells. Chromosomal analysis of hematopoietic chimeric marmosets, inoculated with tumor tissues or cell-free virus, suggested that the HVS-induced neoplastic disease was multiclonal in origin.

0257 COMPARATIVE STUDIES ON VISNA, PROGRESSIVE PNEUMONIA, AND ROUS SARCOMA VIRUSES BY ELECTRON MICROSCOPY. *(E.)* Takemoto, K. K. (Natl. Cancer Inst., Bethesda, Md.), T. Aoki, C. Garon and M. M. Sturm. *J Natl Cancer Inst* 50(2):543-547, 1973.

Visna virus and progressive pneumonia virus (PPV), agents which cause slow infections in sheep, were examined by electron microscopy and compared with Rous sarcoma virus (RSV). Purified visna and PPV virions contained projections or "spikes" on their outer membranes, and negatively stained particles resembled RSV. Internal strandlike structures similar to those described for the avian, murine, and feline C-type viruses were also noted. Examination of thin sections of visna-infected particles also revealed budding as well as extracellular virus with remarkable similarities to Rous sarcoma virus.

0258 INHIBITION OF MORTALITY AND INDUCTION OF IMMUNITY TO FRIEND DISEASE BY LECTIN-TREATED VIRUS. *(E.)* Dent, P. B. (McMaster U. Med. Ctr., Hamilton, Ontario, Canada). *J Natl Cancer Inst* 50(2):511-513, 1973.

The interaction of the plant lectins, phytohemagglutinin and concanavalin A, with Friend leukemia virus preparations inhibited splenomegaly and mortality from Friend disease in DBA/2 mice. The specific activity of concanavalin A was at least 40 times greater than that of phytohemagglutinin. Ten of 16 animals surviving one i.p. injection of 0.2 ml

Friend virus pretreated with 250 μg/ml concanavalin A were completely protected from subsequent challenge with a lethal dose of Friend virus, as judged by absence of splenomegaly. Four animals were partially protected and had mild splenomegaly. The results suggest that lectin-treated virus injections may be an efficient means of immunization and even therapy in Friend disease.

0259 IRRADIATION AND VIRAL LYMPHOMAGENESIS IN THYMIC GRAFTS.

(E.) Hays, E. F. (U. California, Ctr. Hlth. Sci., Los Angeles). *J Natl Cancer Inst* 50(2):523-528, 1973.

AKR (AK) neonatal thymus was subjected to 850 and 5000 rad ^{60}Co irradiation *in vivo* before its removal for grafting to thymectomized syngeneic hosts. These irradiated grafts developed lymphoma after being placed in hosts inoculated as sucklings with cell-free filtrates of Gross Passage A, virus-accelerated AK lymphoma. The incidence was slightly lower and the latent period longer than found in nonirradiated control grafts in similar hosts. However, when thymus was exposed to these filtrates *in vitro* just before grafting or *in vivo* 24 hr before grafting, but placed in hosts not given neonatal virus inoculation, lymphoma incidence was reduced in the irradiated grafts when compared to that of virus-exposed nonirradiated control grafts. Thus heavily irradiated thymic grafts could develop lymphoma in virus-infected hosts, but irradiation of grafts exposed to filtrate *in vitro* and *in vivo* inhibited the ability of the virus-containing filtrates to produce lymphoma, probably by reducing the numbers of cells available for virus replication. One can conclude that lymphoma can develop in AK thymic grafts given 850 and 5000 rads of ionizing radiation when the host animal has its natural level of virus infection increased by inoculation of sucklings with GMuLV. However, lymphoma development in thymus infected with virus, *in vivo* and *in vitro* before grafting, can be inhibited by these same doses of irradiation. Therefore, the amount of virus available to the thymus from established host infection, as well as the numbers of thymic cells which can support virus infection in the thymic graft, must be major factors regulating lymphomagenesis in this system.

0260 STUDIES ON THE ACID MUCOPOLYSACCHARIDE COAT OF VIRUSES AND TRANSFORMED CELLS.

(E.) Shigematsu, T. (U. Texas M. D. Anderson Hosp. Tumor Inst., Houston) and L. Dmochowski. *Cancer* 31(1):165-174, 1973.

The ruthenium red staining method was applied to study of the properties of acid mucopolysaccharides (AMPS) in cells of cultures of normal, RNA virus-transformed, and RNA-virus producing tumor cells. The AMPS layer of plasma membranes of virus-transformed cells was thicker than that of the plasma membrane of normal cells and more resistant to ovine hyaluronidase digestion than that of control normal cells. Hyaluronidase digestion of the AMPS layer of virus-producing tumor cells followed by several centrifugation runs resulted in a considerable concentration of RNA virus particles. This procedure may therefore prove helpful in virus concentration studies. Ruthenium red staining revealed three distinct layers within the unit membrane of the plasma membrane of virus-producing tumor cells as well as in the envelopes of type C and type B RNA virus particles. Study of a mouse mammary tumor (D-W) cell line producing both type B and type C RNA virus particles after labeling by conjugated ferritin with appropriate anti-sera and staining by ruthenium red revealed the presence of ferritin granules in the AMPS layer of these particles. As demonstrated by ruthenium red staining, the functional and immunologic properties of the AMPS layer on virus particles are as follows: it plays a least a part in the adherence of RNA virus particles to the plasma membrane of cells; acts as a protective layer of virus particles against enzymatic damage, and it is a site of viral antigen-antibody reactions. This study also demonstrated similarities in the ultrastructure of plasma membrane of RNA virus-transformed and virus-producing tumor cells and of the envelope of RNA type B and type C virus particles.

0261 PROPERTIES OF AN SV40-TRANSFORMED HAMSTER CELL LINE, RESISTANT TO ACTINOMYCIN D.

(Fr.) Wicker, R. (Cancer Res. Inst., Villejuif, France), M.-F. Bourali, H. G. Suarez and R. Cassingena. *Int J Cancer* 10(3):632-640, 1972.

The biological properties of a hamster cell line Cl 2 TSV-5/R derived from an SV40 tumor and made resistant to 2 μg/ml Actinomycin D were investigated. Studies were focused on tumorigenicity, *in vitro* growth on plastic or in soft agar, persistence of SV40 specific neoantigens (T and TSTA), and karyotype. When compared to the parental cell line (Cl 2 TSV-5), the tumorigenicity and the ability to grow in soft agar of the Cl 2 TSV-5/R cells were strongly reduced (approximately 1,000 fold). Their growth on plastic was also slowed down. Although the SV40 T antigen was still present in all the resistant cells they no longer presented immunosensitivity to the graft rejection test, specific for the SV40 tumor-associated transplantation antigen. The karyotype of Cl 2 TSV-5/R cells was comparable to that of the parental Cl 2 TSV-5 cells.

0262 *IN VITRO* INTERFERENCE BETWEEN LOW AND HIGH LEUKAEMOGENIC VARIANTS OF RAUSCHER VIRUS.

(E.) Boyd, R. E. (Inst. Gustave-Roussy, Villejuif, France), J. K. Youn and G. Barski. *Int J Cancer* 11(1):224-230, 1973.

Cultures of the virus-free P4bis line, originating from a C57Bl mouse, were infected *in vitro* with a

low leukemogenic" variant (RC) of the Rauscher
eukemia virus (RV), prepared from long-term
ultures of this virus in syngeneic cells.
ater, after different delays, these infected
ultures (P4bis+RC) were superinfected with a
ighly pathogenic variant (RR) of the RV, obtained
rom short-term cultures of RV extracted from
pleens of leukemic mice. Eighteen days after
hese superinfections with RR, supernatants of
he doubly-infected cell cultures (P4bis+RC+RR)
nd their corresponding controls were assayed
or leukemogenic potential by i.p. inoculation
nto young adult BALB/c mice. The proportion of
ice which had developed leukemia by the 120th day
fter inoculation served for the evaluation of *in*
itro interference between the RC and RR virus
ariants. Even when the *in vitro* infection of cells
ith RC virus was followed only 1 hr later by the RR
uperinfection, there was a significant difference
etween the proportion of leukemic animals in the
roup inoculated with P4bis+RC+RR supernatant (56%)
nd that in the P4bis+RR positive control group (88%).
ith intervals of 12 or 21 days between the two
vitro viral infections, the drop in leukemo-
enicity of the doubly-infected cultures became
ore accentuated; the longer the time interval,
e greater was the interference effect. Indirect
munofluorescence tests made at intervals after
fection of P4bis with RC showed a steady in-
ease in the percentage of RV antigen positive
lls up to the 3rd week after infection. The
chanism of the observed interference is dis-
ssed, and it is suggested that a straight
lationship exists between the spread, horizontal
d/or vertical, of the RC virus infection in
e cultures and the blocking of their superinfec-
on by the highly pathogenic RR virus.

63 TRANSCRIPTION OF RIBONUCLEIC ACID BY THE RIBONUCLEIC ACID DIRECTED DEOXYRIBO- CLEIC ACID POLYMERASE OF ROUS SARCOMA VIRUS AND OXYRIBONUCLEIC ACID POLYMERASE I OF *ESCHERICHIA* LI.

(E.) Taylor, J. M. (Dept. Microbiol.,
California, San Francisco), A. J. Faras,
E. Varmus, H. M. Goodman, W. E. Levinson and
M. Bishop. *Biochemistry* 12(3):460-467, 1973.

e DNA synthesized *in vitro* by the purified RNA-
rected DNA polymerase of Rous sarcoma virus
SV), using tumor virus RNA as template, contains
th single- and double-stranded molecules. Both
rms are small and the double-stranded form is
pied primarily from particular regions of the
A. These same properties are found to apply
the DNA transcribed from poliovirus RNA. The
imer $(dT)_{12-18}$ augments the response of the
zyme to RSV RNA, heat-denatured RSV RNA, and
liovirus RNA. The size of the DNA products is still
all and the portion of the RNA template represented
double-stranded DNA is decreased. In transcrip-
on from RSV RNA, the use of $(dT)_{12-18}$ produces
ditional initiation sites on the template although
A is not transcribed from regions other than
ose previously transcribed. DNA synthesis on 70S
V RNA template is initiated by the formation of
a phosphodiester bond between a 3'-terminal adenosine moiety and deoxyadenosine. Addition of $(dT)_{12-18}$ stimulates the overall synthesis of DNA and does not interfere with the formation of the above-mentioned bond. The DNA-dependent DNA polymerase I of *Escherichia coli* does not respond to naturally occurring RNA templates. However, addition of $(dT)_{12-18}$ to poliovirus RNA and RSV RNA, which possess poly(A) sequences, permits the synthesis of DNA limited to poly(dA) and poly(dT) rather than heteropoloymer.

0264 EBV INFECTIONS IN BRAZIL. I. OCCURRENCE IN NORMAL PERSONS, IN LYMPHOMAS AND IN LEUKEMIAS.

(E.) Carvalho, R. P. S. (U. Sao Paulo Sch. Med., Brazil), A. S. Evans, P. Frost, G. Daldorf, M. E. Camargo and M. Jamra. *Int J Cancer* 11(1):191-201, 1973.

Sera from normal and leukemic children and adults living in different areas of Brazil were tested by the indirect immunofluorescence and precipitin tests for the presence of anti-Epstein Barr virus (EBV) antibody. Two Burkitt tumor cell lines (EB3 and Jijoye) were used as EBV antigen sources. In the middle and upper socio-economic groups, anti-EBV antibody was detected between the ages of 6 and 12 in over 50% of the normal population tested. However, EBV infection occurred earlier in the lower socio-economic groups (100% of children aged 1 to 5). Of the 69 lymphoma cases studied, all but one (Hodgkin's disease) had EBV antibody present in a titer ≥1:10 (IF test). Thirty-seven percent had elevated titers (i.e. ≥1:320). Elevated anti-EBV titers were also found in 41.9% of sera from the 43 cases of Hodgkin's disease which had antibody, and in four of eight Burkitt lymphoma cases. Antibody titers were 3.5 and 5 times higher, resp., than the titer of the healthy control group. In 13 cases of lymphosarcoma, 23.1% had titers of 1:320 or higher. Anti-EBV was present, but not elevated (≤1:160) in all four cases of reticulosarcoma studied. Only one of 43 leukemia patients had an anti-EBV titer less than 1:10, and in 31% of the positive patients it was elevated. The highest anti-EBV titers were observed in patients with acute myeloid leukemia.

0265 PATTERNS OF SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID TRANSCRIPTION. II. IN TRANSFORMED CELLS.

(E.) Khoury, G. (Natl. Inst. Allergy Infectious Dis., Bethesda, Md.), J. C. Byrne, K. K. Takemoto and M. A. Martin. *J Virol* 11(1):54-60, 1973.

The pattern of SV40 DNA transcription was examined in 11 non-virus-producing SV40-transformed cell lines. Hybridization studies performed with purified unlabeled, transformed cell RNA and ^{32}P-labeled fragments of purified SV40 "plus" or "minus" DNA strands, indicated that no transcription took place from the plus strand of 9 of the 11 cell lines. In the other two lines, less than

8% of the plus strand hybridized with transformed cell RNA. In these same nine lines, virus-specific RNA formed stable hybrids with 37-50% of the minus strand, a value similar to that obtained with RNA prepared during the early phases of a lytic infection of African green monkey kidney cells. RNA from the remaining two lines, however, hybridized with 65-75% of the minus strand, a value considerably higher than that (35%) which is expressed early or late in a productive infection. Results of hybridization experiments for four lines indicated that the same regions of the minus strand transcribed early in a lytic cycle were also transcribed in the SV-40 transformed cells. Also, the nine cell lines which transcribed no plus strand DNA all appeared to transcribe the same region of the minus strand.

0266 SYNTHESIS OF MURINE LEUKEMIA VIRUS IN CELL CULTURE.

(E.) Okano, H. (Dept. Pathol., Kyushu U., Fukuoka, Japan), M. A. Rick, L. Johns and R. Rich. *Int J Cancer* 11(1):95-103, 1973.

Macromolecular synthesis and virus assembly in murine leukemia virus (MuLV)-infected secondary (ICR/Ha Swiss) embryo fibroblast cultures were studied by high resolution electron microscopic autoradiography. Both continuous labeling and pulse-chase studies, with ^{3}H-uridine, of cells infected three weeks previously, revealed early nucleolar and extra-nucleolar nuclear labeling within 30 min. Peak nuclear labeling occurred after one hour with the nucleolus having a grain density twice that seen over the remainder of the nucleus. Cytoplasmic labeling which was evident at this time, did not reach a peak until 24 hr after the start of the labeling period, when nuclear labeling had diminished. By 30 min after initial exposure of cells to ^{3}H-uridine, or ^{3}H-leucine, 0.4-2.5% of the morphologically characteristic virions had incorporated label. The percentage rose to about 25-30% after 24 hr, thus paralleling the pattern of total cytoplasmic labeling. The proportion of labeled to total type-A particles was always greater than the proportion of labeled to total type-C particles.

0267 SUSCEPTIBILITY OF MAMMALIAN (HAMSTER) CELL CULTURES TO INFECTION WITH CELLULAR AND CELL-FREE TYPE II LEUKOSIS (MAREK'S DISEASE) VIRUS.

(E.) Bedigian, H. G. (U. Massachusetts, Amherst) and M. Sevoian. *J Natl Cancer Inst* 50(1): 129-135, 1973.

Primary hamster kidney (HK) cell cultures are susceptible to infection by the JM strain of Marek's disease virus (MDV). HK cells inoculated with either 10,000 PFU of cellular or 5000 PFU of cell-free MDV showed a typical cytopathic effect (CPE) after four to six passages. Infected HK cells showed intranuclear inclusions upon staining with H and E, and nuclear herpesvirus type particles could be seen in specimens viewed under the electron microscope. Fluorescent microscopic examination of infected cells revealed nuclear and cytoplasmic fluorescence in the areas with CPE. Infected HK cells also contained complement-fixing antigens, and MDV from these cultures was neutralized by anti-MDV antisera prepared in rabbits. MDV could be recovered from the kidneys of chickens inoculated with virus-infected HK cell cultures. Plaque formation in MDV-infected HK cultures was almost completely prevented by 10 μg/ml BUdR.

0268 SOME PHYSICAL AND BIOLOGICAL CHARACTERISTICS OF THE MOUSE MAMMARY TUMOR VIRUS.

(E.) Moore, D. H. (Inst. Med. Res., Camden, N.J.), N. H. Sarkar, J. Charney and B. Kramarsky. *Cancer Res* 33(1):5-10, 1973.

Mammary tumor virus (MTV) was separated from a sample of RIII mouse milk on density gradients made of different materials. Titrations of infectivities, the isopycnic densities, and the appearance of MTV virions after fractionation are reported. Neither the infectivity nor the morphology of the MTV was appreciably affected by abrupt dilutions from strong solutions of cesium chloride, sodium chloride, or sodium iothalamate. It was not necessary to use solutions of low osmotic activity in the purification of this virus by density gradient centrifugation. In sucrose, sodium iothalamate, potassium tartrate, or citrate, the buoyant density of the main virion band was 1.18 g/ml; in cesium chloride, it was approximately 1.22 g/ml. A correlation was found between the apparent integrity of particles and their infectivity in bands recovered from various gradients. The infectivity titer of the main virion band varied with the gradient medium used; but sucrose, cesium chloride, Ficoll, potassium tartrate, or citrate and sodium iothalamate all were satisfactory, sucrose probably being preferred. The distribution and appearance of bands varied greatly from one gradient to another. Gradients of neutralized sodium phosphotungstate destroyed both the infectivity and the morphological integrity of the virions.

0269 COMMON ORIGINAL FEATURES OF PICORNA VIRUSES, INDUCED IN CELL CULTURES DERIVED FROM PRIMARY MALIGNANT ADENOPATHIES IN HUMANS.

(Fr.) Gevaudan, P. (Med. Sch. Hyg. Lab. Marseille, France), G. Pieroni, M. J. Gevaudan and J. Charrel. *C R Acad Sci [D] (Paris)* 275(23): 2799-2801, 1972.

A study of five picorna virus strains propagated as latent forms and induced under decreased cell cultivation temperatures (referred to in a previous paper) in five cell lines derived from five patients with malignant adenopathies is presented. Development of these viruses during a single

ycle (6 hr at 35 C) in the presence of labeled
mino acids generated virions and empty capsides
ith 130 and 75 S sedimentation coefficients,
ith respect to ribosomes, separable by sucrose
radient centrifugation. A new structure
as observed to occur when these virus strains
eveloped during 18 hr at 35 C. This structure
resented a 115 S sedimentation coefficient and
igrated slower than the virions in the sucrose
radient. The structure incorporated tritiated
ridine and ^{14}C-labeled amino acids at rates
dentical to those observed in the virion, but
iffered from the latter by its susceptibility
o pancreatic RNase. Its synthesis and accumu-
ation rates reached a maximum at 35 C and were
uch lower at 32 and 37 C. Due to its homogeneity
he structure was postulated to be an intermediate
roduct of viral metabolism. This structure was
dentified only in picorna viruses, in all five
trains, and attempts to verify its involvement
n the process leading to viral latency are in
rogress.

70 TUMOR INDUCTION IN MICE WITH ROUS SARCOMA VIRUS.

(Rus.) Chepulis, G.-K. S. (D. I.
anovskii Inst. Virol., Acad. Med. Sci. USSR, Moscow),
M. Zhdanov, V. Ya. Shevlyagin and L. G. Kiseleva.
pr Virusol 17(4):427-430, 1972.

e possibility of overcoming species and age barriers
induction of a neoplastic process by Rous sarcoma
rus (RSV) is shown. Chick Rous sarcoma virus
mor cells were administered s.c. in the spinal
ea to low-carcinogenic line C57Bl mice, in which
munologic tolerance had been induced one day after
rth by i.p. administration of various cell antigens
tracted from guinea pig kidneys, human embryo
broblasts, rabbit serum, or chick embryo muscular
ssue. All mice tolerant to chicken antigens (normal
orio-allantoic membrane, muscular tissue) and
fected with $10^5 OD_{50}$ (oncogenic dose) of RSV at
5 to 4 months of age developed tumors. No
mors were observed in mice, tolerant to guinea
kidney, human fibroblast or rabbit serum
tigens, upon infection with RSV. One out of 30
-tolerant mice infected with RSV developed tumors.
mors appeared locally in the spinal area in chick
tigen-tolerant infected mice 2.5-3.5 months
m the time of infection. Histologically the
ors developed were polymorphous cell sarcomas.
primary tumors were passaged in adult mice of the
e line and developed sarcomas in three weeks.
inistration of normal chorio-allantoic membrane
racts to non-infected tolerant and non-tolerant
e produced no tumors.

1 REPLICATION OF POLYOMA DNA IN ISOLATED NUCLEI. I. CHARACTERIZATION OF THE TEM FROM MOUSE FIBROBLAST 3T6 CELLS.

(E.)
nacker, E.-L. (Karolinska Inst., Stockholm,
den), G. Magnusson and P. Reichard. *J Mol*
l 72(2):523-537, 1972.

lei isolated from polyoma virus-infected mouse fibroblast 3T6 cells incorporated radioactive [^{3}H]dTTP into the replicative intermediate of polyoma DNA. The replicative intermediate was characterized by centrifugation through neutral and alkaline sucrose gradients, isopycnic banding in a propidiumiodide/cesium chloride gradient, by chromatography on benzoylated naphthoylated DEAE-cellulose and DNA-DNA hybridization. A small but significant amount of radioactivity was incorporated into mature polyoma DNA. The incorporation of radioactivity into polyoma DNA required the presence of dATP, dCTP and dGTP and was stimulated by ATP, Mg^{2+} and monovalent cations. A substantial part of the radioactivity was incorporated into short chains during the first five minutes of incubation while after 30 minutes most of the incorporation had occurred into longer chains which, in an alkaline sucrose gradient, sedimented close to 16 s, the sedimentation coefficient of unit-length linear polyoma DNA. The time-course of the reaction was studied at 25, 30 and 35 C. The incorporation was non-linear at all temperatures but reached a similar plateau value in each case. It is proposed that the nuclei *in vitro* retained the capacity to elongate replicative intermediate molecules present at the time of their isolation but only had a very limited ability to initiate the replication of new polyoma DNA molecules.

0272 REPLICATION OF POLYOMA DNA IN ISOLATED NUCLEI. II. EVIDENCE FOR SEMI-CONSERVATIVE REPLICATION.

(E.) Magnusson, G. (Med. Nobel Inst., Stockholm, Sweden), E.-L. Winnacker, R. Eliasson and P. Reichard. *J Mol Biol* 72(2):539-552, 1972.

Nuclei from polyoma-infected mouse fibroblast 3T6 cells catalysed the incorporation of [^{3}H]dTTP into the replicative intermediate of polyoma DNA. The nature of this process was studied by the introduction of a density label into the viral DNA. For this purpose dBrUTP replaced dTTP in the *in vitro* system and the product formed in the presence of [^{14}C]dATP was analysed by equilibrium centrifugation in neutral CsCl or alkaline Cs_2SO_4 gradients. The results suggest that the density label was incorporated into newly synthesized progeny strands and that the incorporation process represented semi-conservative replication. In a second type of experiment the replicative intermediate was first pulse-labelled *in vivo* with [^{3}H]thymidine. The isolated nuclei were then incubated *in vitro* with dBrUTP and [^{14}C]dATP. Analysis of the distribution of the two isotopes in buoyant density gradients suggested that *in vitro* synthesis represented elongation of essentially all replicative intermediate molecules present in the nuclei at the time of isolation. From the observed density shifts it was calculated that the initial ratio of the *in vitro* elongation process corresponded to a replication time of about 20 minutes and thus may amount to about 20% of the reported *in vivo* rate.

0273 A WORKING HYPOTHESIS EXPLAINING THE MAINTENANCE OF THE TRANSFORMED STATE BY SV40 AND POLYOMA.

(E.) Levine, A. J. (Dept. Biochem. Sci., Princeton U., N.J.) and M. M. Burger. *J Theor Biol* 37(3):435-446, 1972.

A model is presented that attempts to explain how SV40 or polyoma maintains the transformed state or phenotype. It is proposed that SV40 or polyoma codes for a protein involved in the initiation of cellular DNA synthesis (positive control element). This viral directed cellular DNA synthesis is different in some way from normal cellular DNA synthesis. This difference, in addition to other factors (possibly a second viral coded protein), results in a permanent cell surface alteration. This same cell surface alteration is observed transiently (only during mitosis) in the normal cell cycle. The permanent cell surface change permits the synthesis or activation of the viral positive control element for cellular DNA replication by-passing the normal cellular control for the initiation of DNA synthesis and allowing the cell to enter a new S period. This results in loss of density growth and expression of the transformed phenotype. The model is presented as a working hypothesis that makes specific predictions for new experiments.

0274 RETRANSFORMATION OF TS SV40 TRANSFORMANTS BY MURINE SARCOMA VIRUS AT NON-PERMISSIVE TEMPERATURE.

(E.) Renger, H. C. (New York U. Med. Ctr., N.Y.). *Nature New Biol* 240(96):19-21, 1972.

Temperature sensitive (ts) SV40 transformed mouse fibroblasts (tsSV3T3) were retransformed at 39 C with the same efficiency as normal 3T3 cells by murine sarcoma virus (MSV). The number of transformed foci in MSV-infected tsSV3T3 cultures growing at 39 C was similar to that in identically infected 3T3 cells. MSV production was also normal: virus titers at 39 C were similar for MSV-transformed tsSV373 and 3T3 cells. MSV-tsSV3T3 cells lost growth control at 39 C and grew to high saturation densities (1×10^6 cells/sq cm). In comparison, tsSV3T3 cells reach densities of only 1×10^5 cells/sq cm at this temperature. Agglutination tests with Concanavalin A indicated that the same surface sites were permanently uncovered in MSV-tsSV3T3 cells which were normally only exposed in tsSV3T3 cells during growth at 32 C.

0275 TRANSFORMATION OF RAT LIVER CELLS WITH CHICKEN SARCOMA VIRUS B77 AND MURINE SARCOMA VIRUS.

(E.) Altaner, C. (Cancer Res. Inst., Bratislava, Czechoslovakia) and E. Hlavayova. *J Virol* 11(2):177-182, 1973.

Rat liver cells *in vitro* were transformed with chicken sarcoma virus B77, giving RL(B77) cells, and with murine sarcoma virus (Harvey), giving RL(MSV) cells. Rat liver cells transformed spontaneously *in vitro* were designated RL cells. In addition, the RL(MSV) cell line was adapted for growth in culture fluid containing 25 μg of 5-bromodeoxyuridine per ml. All cell lines were tumorigenic in 1-wk-old rats. The number of cells needed for induction of tumor growth was 1,000-fold higher in the case of RL(B77) cells in comparison with RL(MSV) cells and RL cells. No production of viral particles from any of the cell lines investigated was detected by plating concentrated supernatant fluid of the cultures on different secondary embryo cells with and without fusion by Sendai virus, by labeling with uridine-5-^{3}H, or by assay for deoxyribonucleic acid polymerase activity. The viral genome was rescued by fusion of RL(B77) cells with chicken cells. Chicken sarcoma virus rescued from (RL(B77) cells differed in plating efficiency on duck cells from B77 virus rescued from transformed rat embryo cells. No virus was rescued after fusion of RL(MSV) and RL cells with mouse, rat, or chicken embryo cells. Infectious murine sarcoma virus can be induced by 5-bromodeoxyuridine from RL(MSV) cells.

0276 WILD-TYPE GROSS LEUKEMIA VIRUS: CLASSIFICATION OF SOLUBLE ANTIGENS (GSA).

(E.) Aoki, T. (Natl. Cancer Inst., Bethesda, Md.), R. B. Herberman, P. A. Johnson, M. Liu and M. M. Sturm. *J Virol* 10(6):1208-1219, 1972.

Blood plasma from leukemic and nonleukemia AKR or C58 mice was reacted as sources of Gross soluble antigen (GSA) with mouse anti-GSA Gross-typing (G-typing) serum in indirect immunofluorescence tests and tests of the inhibition of the G-typing sera by GSA sources. Ascites fluid of AKR mice bearing a transplanted leukemia was also used as a GSA source in some tests. GSA in plasma of leukemic and nonleukemic mice and in ascites fluid showed various specificities of reaction with G-typing serum. The G cell surface antigens GCSA$_a$, b and c, and type-specific and group-specific Gross virus envelope antigens tsVEA and gsVEA were among the antigens whose presence in the GSA sources was indicated. Plasma from nonleukemic C58 mice appeared to lack GSAC$_c$. The G$_{IX}$ antigen was not seen in the GSA source material.

0277 ULTRASTRUCTURAL FINDINGS OF ADENOVIRUS TYPE 12-INDUCED TUMOR CELLS TREATED BY DIFFERENTIAL HYPOTHERMIA.

(E.) Tabuchi, K. (Okayama U. Med. Sch., Japan), M. Arimori, H. Yoshizu, K. Suga, T. Ohashi, O. Yamada and A. Nishimoto. *Gann* 63(6):725-730, 1972.

Hamsters were injected s.c. in the cheek pouch with material from an adenovirus 12-induced tumor. Tumor-bearing hamsters were anesthetized and cooled to 10-15 C while their cheek pouches were maintained at 37 C; differential hypothermia thus established was continued for 10 hr, after which tumors were removed and examined under the electron microscope.

mmediately after hypothermic treatment, nuclear
hromatin appeared condensed and cystic dilatation
f perinuclear cisternae was observed. Swelling
f mitochondria and disappearance of mitochondrial
ristae were also noted. Condensation of nuclear
hromatin and cystic dilatation of perinuclear
isternae were more prominent 12 hr after
reatment. Vacuolization of the nucleus was
lso seen at 12 hr. By 24 hr nuclear destruction
as prominent. Fibrocytes and fibroblasts in
heek pouch submucosa were not changed by hypothermia.
ntracisternal C-type particles sere seen in tumor
ells 12 hr after, but not immediately after or
hr after, hypothermic treatment.

78 CYTOLOGICAL, CYTOGENETIC AND ULTRASTRUCTURAL STUDIES OF RAT CELL LINES CARRYING
IAN AND MURINE SARCOMA GENOMES.

(E.) Karpas,
(Dept. Med., U. Cambridge, England), J. Cawley
d E. Tuckerman. *Z Krebsforsch* 78(1):51-64,
72.

o rat cell lines (RSK 4 and XC) which carry
e RSV genome but do not produce infectious virus
re studied together with a control rat cell line
W 13). One rat cell line (MSB-1) which carries
e MSV genome and produces infectious particles
s also studied. The cells carrying viral sar-
ma genome showed pleomorphic cytology. All cell
nes showed acid phosphatase positivity, indica-
ng the presence of lysosomes, but only cells of
e MSB-1 line contained glycogen granules. Cyto-
netic studies revealed a wide range of chromo-
mal changes. Most cells carrying sarcoma viral
nome contained large metacentric and/or sub-
tacentric "marker" chromosomes. Such chromosomes
not present in normal rat cells even after
olonged *in vitro* cultivation. Many of the RSK
cells showed a chromosome number ranging between
to 80, while XC cells showed a pseudodiploid
tern with a chromosome number around the normal.
ut half the MSB-1 cells contained between 50-60
omosomes. Ultrastructural studies revealed
ther viral induced changes in the RSK 4, XC and
-1 cell lines. The cytoplasm of some cells
the three lines contained vesicular structures
ch contained numerous electron dense particles,
sibly immature C-type virus. Such structures
e not found in any cells of the control LW 13 rat
l line.

9 SURFACE GLYCOPROTEINS OF CELLS BEFORE AND AFTER TRANSFORMATION BY ONCOGENIC VIRUSES.

) Warren, L. (Sch. Med., U. Pennsylvania,
ladelphia), J. P. Fuhrer and C. A. Buck.
Proc 32(1):80-85, 1973.

double label method employing L-fucose-^{14}C
-^{3}H was used to compare the carbohydrate com-
ents of the surface glycoproteins of control
virus-transformed cells. A material eluting
ly from a column of Sephadex G-50 was found
relatively large quantity in transformed cells
compared with controls. The cells must be in a growing state for this peak to appear. This material elutes early because it contains extra residues of sialic acid. A sialyl transferase was found that may account for the formation of this material. Its activity is 2.5- to 11-fold greater in transformed cells than in controls and activity decreases when the cells are not dividing. Work with chick embryo fibroblasts transformed by T5, a temperature-sensitive mutant of Rous sarcoma virus, shows that the peak of early eluting material and the sialyl transferase activity are present in considerably greater amounts at the permissive temperature of 36 C where transformation is manifested than at 41 C where the cells appear and behave as if normal.

0280 DETECTION OF AVIAN TUMOR VIRUS RNA IN UNINFECTED CHICKEN EMBRYO CELLS.

(E.) Hayward, W. S. (Public Hlth. Res. Inst. City New York, N.Y.) and H. Hanafusa. *J Virol* 11(2):157-167, 1973.

Uninfected chicken embryo cells were analyzed for the presence of viral ribonucleic acid (RNA) by molecular hybridization with the single-stranded deoxyribonucleic acid (DNA) product of the RNA-dependent DNA polymerase contained in avian sarcoma-leukosis virions. Viral RNA was detected in all cells which contained the avian tumor virus group-specific antigen and the virus-related helper factor. The amounts of viral RNA in these cells ranged from approximately 3 to 40 copies of viral-specific sequences per cell. In general, the viral RNA content correlated with the level of helper activity in the cells. Cells infected with Rous-associated virus 2 contained 3,000 to 4,000 copies of viral RNA per cell. RNA from these infected cells hybridized with nearly 100% of the viral ^{3}H-DNA. By contrast, a maximum of less than 50% hybridization was obtained with RNA from the uninfected helper-positive cells, suggesting that not all of the viral RNA sequences were present in these cells. No viral RNA was detected in cells which lacked group-specific antigen and helper activity. Under the conditions used in these studies, less than 0.3 viral genome equivalents of RNA per cell would have been detected.

0281 ON THE PRESENCE OF RIBOSOME-LIKE PARTICLES IN AVIAN MYELOBLASTOSIS VIRIONS (AMV) AND UNUSUAL ULTRASTRUCTURE OF THEIR NUCLEOID.

(E.) Smetana, K. (Lab. Ultrastructure Res. Cells Tissues, Czechoslovak Acad. Sci., Prague), J. Korb and J. Riman. *Neoplasma* 19(5):437-441, 1972.

The ultrastructure of avian myeloblastosis virus (AMV) was studied *in situ* with special regard to the presence of ribosome-like structures in this virus. The viral C particles contained dense granules 90-230 A in diameter, most of which were localized between the inner and external membrane of virus particles. However, some particles were also present inside the nucleoid. In addition, a tetrade of dense granules joined together by a dense filament was observed in a few viral particles. The nucleoid in such particles appeared to be divided or split into two parts. The presence of ribosome-like granules in the virus particles supports recent biochemical observations according to which a specific ribosome is a constant component of AMV viruses. The interpretation of these findings is not possible at present.

0282 FURTHER STUDIES ON MOUSE SARCOMA VIRUS (MOLONEY) REPLICATION IN HUMAN CELLS. PARTIAL HOST RANGE SHIFT OF PROGENY VIRUS. *(E.)*

Bernard, C. (St. Louis Hosp., Paris, France), J. C. Chuat, I. Laprevotte and M. Boiron. *Int J Cancer* 10(3):518-526, 1972.

Several human cell lines were examined for their susceptibility to infection by mouse sarcoma virus, strain Moloney (M-MSV). Successful transformation was achieved only with concentrated virus from rat cell line 78 A1. The lag time and the extent of cellular alteration were found to be related to the employed multiplicity of infection (total transformation within 4 days at a multiplicity of 0.1). All the experiments performed showed the tropism of the progeny virus (HU-MSV) to shift from a double murine and human tropism to exclusive human tropism as far as the transforming function is concerned. On the contrary, nontransforming replication in mouse cells was continuously obtained. *In vivo* studies yielded parallel results: only the leukemogenic potential of HU-MSV for mice was found to persist. The mechanism of this dissociated host-range shift of transforming and non-transforming virions is discussed.

0283 *IN SITU* HYBRIDIZATION OF RD114-VIRUS RNA WITH HUMAN METAPHASE CHROMOSOMES. *(E.)*

Price, P. M. (Mt. Sinai Sch. Med., New York, N.Y.), K. Hirschhorn, N. Gabelman and S. Waxman. *Proc Natl Acad Sci USA* 70(1):11-14, 1973.

Virus was isolated from RD114 cells, a line of cells cultured from a human rhabdomyosarcoma. RNA was isolated from the RD114-virus and labeled with ^{3}H. Labeled RNA was introduced into preparations of human metaphase chromosomes from long-term lymphoid cultures. Hybridization of RD114-virus RNA with chromosomes was observed. In 8 of 100 metaphase spreads, association above the background level was observed. Statistical analysis of data obtained from radioactive labeling of chromosomes indicated that a D group chromosome contained stretches of DNA having sequence homology with RD114-virus RNA.

0284 SURFACE CHANGES IN TEMPERATURE-SENSITIVE SIMIAN VIRUS 40-TRANSFORMED CELLS. *(E.)*

Noonan, K. D. (Dept. Biochem. Sci., Princeton U., N.J.), H. C. Renger, C. Basilico and M. M. Burger. *Proc Nat Acad Sci USA* 70(2):347-349, 1973.

Temperature-sensitive (ts) SV40-transformed cells were incubated with concanavalin A (Con A) and the binding of Con A by cells was observed at different temperatures. Normal and SV40-transformed 3T3 cells bound unchanging amounts of Con A at 32 and 39 C, but ts SV40-transformed 3T3 cells bound less Con A at 39 than 32 C. Binding of Con A to transformed 3T3 cells increased after the temperature of cell media was reduced from 39 to 32 C. Inhibition (by hydroxyurea) of DNA synthesis in ts cells prevented the increase in Con A binding which occurred when cells were reduced from 39 to 32 C.

0285 RELEASE OF INFECTIOUS EPSTEIN-BARR VIRUS BY TRANSFORMED MARMOSET LEUKOCYTES. *(E.)*

Miller, G. (Yale U. Sch. Med., New Haven, Conn.) and M. Lipman. *Proc Natl Acad Sci USA* 70(1):190-194, 1973.

Epstein-Barr virus (EBV) from infectious mononucleosis patients was used to transform marmoset blood leukocytes and the EBV produced by the marmoset cells (EBV line B95-8) was compared with EBV produced by human leukocytes transformed by EBV. Transformation of umbilical cord leukocytes was the measure of EBV activity. B95-8 cells released abundant EBV extracellularly, while transformed human cells released only an occasional EBV nucleocapsid. EBV from B95-8 cells showed higher titers of transforming activity than EBV from human cells. EBV from marmoset cells also contained more EBV complement fixation antigen and more immunofluorescence antigen than EBV from human cells.

0286 CHARACTERIZATION OF MORPHOLOGIC REVERTANTS OF MURINE AND AVIAN SARCOMA VIRUS-TRANSFORMED CELLS. *(E.)*

Stephenson, J. R. (Natl. Cancer Inst., Bethesda, Md.), R. K. Reynolds and S. A. Aaronson. *J Virol* 11(2):218-222, 1973.

Growth characteristics, tumorigenicity, and ^{14}C-glucose uptake were examined in morphological revertant cells isolated from mouse cells transformed by Kirsten murine sarcoma virus (KiMSV) or Schmidt-Ruppin Rous sarcoma virus. Saturation densities of revertant lines were well below those of parent transformed cell lines, and were similar to those of nontransformed parent mouse cells. Colony-forming efficiency (CFE) of revertants, like that of normal, nontransformed parent cells, was markedly lower than the CFE of transformed parent lines. Glucose uptake had reverted to normal in revertant cells and the malignant potential of revertants was reduced relative to that of transformed parent cells. Revertant lines and transformed cells contained similar concentrations of avian group-specific antigen. Revertants from KiMSV-transformed parent lines showed reduced susceptibility to transformation by KiMSV.

0287 ROLE OF MITOCHONDRIA IN THE PRODUCTION OF RNA-CONTAINING TUMOR VIRUSES. *(E.)*
ader, A. V. (Natl. Cancer Inst., Bethesda, Md.).
Virol 11(2):314-324, 1973.

ouse spleen and thymus cells (JLS-V5) infected
ith Rauscher murine leukemia virus and chick
mbryo cells (CE) transformed by Rous sarcoma
irus (RSV) were treated with ethidium bromide
EB); total exposure time to EB was 37 days for
ome JLS-V5 cells. The effects of EB on cell
orphology and on virus production were observed
lectron microscopically. JLS-V5 cells exposed to
B grew more slowly than untreated cells. The
itochondria of EB-treated JLS-V5 cells increased
n size and assumed bizarre shapes, but this
ffect of EB was reversible. In JLS-V5 cells,
B affected neither the production of virions nor
e morphology of virions produced. The same was
een for CE cells; EB produced mitochondrial
anges in CE cells but did not affect the
productive cycle of RSV in CE cells.

88 INCREASED SPLENIC TRANSPLANTABLE COLONY-FORMING UNITS RELATED TO INCREASED
DIORESISTANCE AFTER RAUSCHER LEUKEMIA VIRUS
FECTION OF THE SJL/J MOUSE. *(E.)* Markoe,
M. (Allegheny Gen. Hosp., Pittsburgh, Pa.),
P. OKunewick, P. Erhard and E. Phillips. *J*
tl Cancer Inst 50(2):449-455, 1973.

e Till and McCulloch exogeneous colony-forming
it (CFU) assay was used to study changes in
lenic CFU content as a function of time after
scher leukemia virus (RLV) infection of
L/J mice. The relationship of splenic CFU
tent to previously reported changes in radio-
sitivity after infection was examined. Total
lenic CFU content [colony-forming potential (CFP)/
leen] first increased after the 6th day of infec-
on, correlating with the appearance of spleno-
galy and of radioresistance. CFP/spleen
tinued to increase until day 22 after infection,
ching 16 times normal levels. This increase
positively correlated with greater radioresis-
ce. Beyond the 22nd day of infection, CFP/spleen
l somewhat but was never <9 times normal values.
ever, calculations based on existing information
arding spleen cell radiation survival and turn-
r indicated that this level of splenic CFU
sufficient to provide radioprotection at the
el of X-ray exposure used (51 R/min for a total
950 R). It was concluded that RLV infection
reased the number of splenic hematopoietic stem
ls of the SJL/J mouse and that this increase
ulted in the greater radio-resistance.

9 ENHANCEMENT OF ADENOVIRUS TRANSFORMATION BY TREATMENT OF HAMSTER CELLS WITH
RAVIOLET IRRADIATION, DNA BASE ANALOGS, AND
ENZ(a,h)ANTHRACENE. *(E.)* Casto, B. C.
oLabs, Inc., Northbrook, Ill.). *Cancer Res*
2):402-407, 1973.

Treatment of hamster embryo cells with ultraviolet irradiation, 5-bromodeoxyuridine, 5-iododeoxyuridine, 5-bromodeoxycytidine, or dibenz(a,h)anthracene prior to the addition of an oncogenic adenovirus, SA7, enhanced the frequency of viral transformation among surviving cells. An increase of over 100-fold was demonstrated with 5-bromodeoxyuridine, 12.5 μg/ml, and over 80-fold with 5-bromodeoxycytidine, 100 μg/ml. Increases in the viral transformation frequency were directly related to dose; however, excessive treatment often resulted in a marked decrease in enhancement. The stimulation of SA7 transformation could not be related to the selection of transformation-sensitive cells, since the absolute number of SA7 foci per plate increased up to eight-fold under conditions where over 90% of the cells were unable to form colonies. These data suggest that the enhancement results from a direct effect upon the cells, thereby increasing their susceptibility to viral transformation.

0290 INFECTION OF THYMUS CELLS *IN VIVO* AND *IN VITRO* WITH A GUINEA PIG HERPES-LIKE VIRUS AND THE EFFECT OF ANTIBODY ON VIRUS REPLICATION IN ORGAN CULTURE. *(E.)* Tenser, R. B. (Yale U. Sch. Med., New Haven, Conn.) and G. D. Hsiung. *J Immunol* 110(2):552-560, 1973.

0291 *HERPESVIRUS SAIMIRI:* ENHANCEMENT OF ADENOVIRUS-ASSOCIATED VIRUS. *(E.)* Dolin, R. (Natl. Cancer Inst., Bethesda, Md.) and A. S. Rabson. *J Natl Cancer Inst* 50(1):205-208, 1973.

0292 THE EFFECT OF CYTOSINE ARABINOSIDE ON VIRUS PRODUCTION IN VARIOUS CELLS INFECTED WITH HERPES SIMPLEX VIRUS TYPES 1 AND 2. *(E.)* Nutter, R. L. (M. S. Hershey Med. Ctr., Pennsylvania St. U., Hershey) and F. Rapp. *Cancer Res* 33(1):166-170, 1973.

0293 AMMONIA EFFECTS IN CULTURES OF NORMAL AND TRANSFORMED 3T3 CELLS. *(E.)* Visek, W. J. (Dept. Biol., Massachusetts Inst. Tech., Cambridge), G. M. Kolodny and P. R. Gross. *J Cell Physiol* 80(3):373-382, 1972.

0294 THE EFFECT OF DIBUTYRYL ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE ON THE SYNTHESIS OF GLYCOLIPIDS BY NORMAL AND TRANSFORMED Nil CELLS. *(E.)* Sakiyama, H. (Dept. Biol., Massachusetts Inst. Tech., Cambridge) and P. W. Robbins. *Arch Biochem Biophys* 154(1):407-414, 1973.

0295 SOLUBILIZATION OF G (GROSS) ANTIGENS ON THE SURFACE OF G LEUKEMIA CELLS. *(E.)* Herberman, R. B. (Natl. Cancer Inst., Bethesda, Md.), T. Aoki and M. E. Nunn. *J Natl Cancer Inst* 50(2):481-490, 1973.

0296 RELEASE OF MOUSE LEUKEMIA-SARCOMA VIRUS FROM SYNCHRONIZED CELLS. *(E.)* Panem, S. (Dept. Path., U. Chicago, Ill.) and W. H. Kirsten. *J Natl Cancer Inst* 50(2):563-566, 1973.

0297 DETECTION OF VIRUS IN SQUAMOUS PAPILLOMAS OF THE WILD BIRD SPECIES *FRINGILLA COELEBS*. *(E.)*. Lina, P. H. C. (Radiobiol. Inst., Rijswijk, Netherlands), M. J. van Noord and F. G. de Groot. *J Natl Cancer Inst* 50(2):567-571, 1973.

0298 SIMULTANEOUS OCCURRENCE OF "AMERICAN BURKITT'S LYMPHOMA" IN NEIGHBORS. *(E.)* Levine, P. H. (Natl. Cancer Inst., Bethesda, Md.), S. G. Sandler, D. M. Komp, G. T. O'Conor and D. M. O'Connor. *N Engl J Med* 288(11):562-563, 1973.

0299 GLYCOSYLTRANSFERASE AND SIALIC ACID LEVELS OF NORMAL AND TRANSFORMED CELLS. *(E.)* Grimes, W. J. (Coll. Med., U. Arizona, Tucson). *Biochemistry* 12(5):990-996, 1973.

0300 INHIBITION OF RNA-DEPENDENT DNA POLYMERASE OF ROUS SARCOMA VIRUS BY THIOSEMICARBAZONES AND SEVERAL CATIONS. *(E.)* Levinson, W. (Dept. Microbiol., U. California, San Francisco), A. Faras, B. Woodson, J. Jackson and J. M. Bishop. *Proc Natl Acad Sci USA* 70(1):164-168, 1973.

0301 ULTRASTRUCTURAL STUDIES OF CELL-VIRUS INTERACTION IN REPTILIAN CELL LINES. I. INTRAMITOCHONDRIAL VIRUSES IN "C-TYPE" VIRUS-PRODUCING VSW CELLS. *(E.)* Lunger, P. D. (Dept. Biol. Sci., U. Delaware, Newark) and H. F. Clark. *J Natl Cancer Inst* 50(1):111-117, 1973.

0302 VIRUS REPLICATION IN IMMATURE GRANULOPOIETIC CELLS OF RAUSCHER LEUKEMIA-BEARING MICE. *(Ger.)* Forteza-Vila, J. (Basic Clin. Res. Ctr. U. Ulm, W. Germany), H.-J. Seidel and W. Calvo. *Acta Haematol* 47(3):168-174, 1972.

0303 COMPARING THE PROTECTIVE POWER OF VIRUS AND TRANSFORMED ALLOGENIC CELLS IN THE POLYOMA VIRUS-MOUSE SYSTEM. *(Fr.)* Favre, R. (Natl. Inst. Hlth. Med. Res., Marseille, France) and G. Meyer. *C R Acad Sci [D] (Paris)* 275(16):1835-1837, 1972.

0304 INVESTIGATION OF CONTINUOUS LINES OF MOUSE SPLEEN CELLS INFECTED WITH FRIEND AND RAUSCHER LEUKEMIA VIRUSES. *(Rus.)* Shubladze, A. K. (D. I. Ivanovskiy Inst. Virol., USSR Acad. Med. Sci.), V. N. Blyumkin, A. M. Amchenkova, M. B. Petukhova, F. P. Filatov and G. A. Delimbetova. *Vopr Virusol* 17(4):464-469, 1972.

0305 ISOLATION OF A LEUKEMIA VIRUS FROM A CELL CULTURE OF HUMAN ORIGIN. *(Rus.)* Zhdanov, V. M. (Central Inst. Postgrad. Med., Moscow, USSR), V. D. Soloviev, T. A. Bektemirov, A. F. Bykovsky and F. A. Filatov. *Vopr Virusol* 17(4):419-427, 1972.

0306 THE STIMULATING EFFECT OF HISTONE ON ONCOGENICITY OF MOLONEY MURINE SARCOMA VIRUS *IN VIVO*. *(Rus.)* Tatarinova, Yu. N. (N. F. Gamaleya Inst. Epid. Microbiol., USSR Acad. Med. Sci. Moscow), V. D. Koltsov, A. A. Lushnikov, L. L. Krymskaya, A. M. Amchenkova and I. G. Balandin. *Vopr Virusol* 17(4):486-490, 1972.

0307 MALIGNANCY IN A SMALLPOX VACCINATION SCAR. REPORT ON ANOTHER CASE. *(E.)* Friedman, M. M. (London, England) and J. A. W. Miller-Cranko. *Cent Afr J Med* 18(7):142, 1972.

0308 TRANSLATION OF ONCOGENIC VIRAL RNA AND EUKARYOTIC MESSENGER RNA IN THE *E. COLI* CELL-FREE SYSTEM. *(E.)* Gielkens, A. L. J. (Dept. Biochem., U. Nijmegen, Netherlands), M. H. L. Salden, H. Bloemendal and R. N. H. Konings. *Fed Eur Biochem Soc Lett* 28(3):348-352, 1972.

0309 EFFECT OF RIFAMPICIN ON THE DEVELOPMENT OF TUMOURS INDUCED BY ADENOVIRUS IN MALE HAMSTERS. *(E.)* Toolan, H. W. (Putnam Mem. Hosp., Bennington, Vt.) and N. Ledinko. *Nature [New Biol]* 237(76):200-202, 1972.

0310 VARIATION IN ONCOGENIC AND TRANSFORMING POTENTIAL OF PARA (DEFECTIVE SV40)-ADENOVIRUS 7. *(E.)* Duff, R. (Milton S. Hershey Med. Ctr. Pennsylvania St. U., Hershey), P. Knight and F. Rapp. *Virology* 47:849-853, 1972.

0311 TRANSIENT INHIBITION OF POLYOMA VIRUS SYNTHESIS BY SENDAI VIRUS (PARAINFLUENZA I). I. DEMONSTRATION AND NATURE OF THE INHIBITION BY INACTIVATED VIRUS. *(E.)* Smith, G. L. (Div. Biol., Kansas State U., Manhattan) and R. A. Consigli. *J Virol* 10(6):1091-1097, 1972.

0312 TRANSIENT INHIBITION OF POLYOMA VIRUS SYNTHESIS BY SENDAI VIRUS (PARAINFLUENZA I). II. MECHANISM OF THE INTERFERENCE BY INACTIVATED VIRUS. *(E.)* Smith, G. L. (Division Biol., Kansas State U., Manhattan) and R. A. Consigli. *J Virol* 10(6):1098-1108, 1972.

0313 INHIBITION OF HIGH MOLECULAR WEIGHT RNA SYNTHESIS IN A HAMSTER MELANOMA BY ETHIDIUM BROMIDE *IN VIVO*. *(E.)* Birkmayer, G. D. (Inst. Cell Biol. U. Munich, West Germany), F. Miller and B.-R. Balda. *Hoppe Seylers Z Physiol Chem* 353(11):1749-1754, 1972.

0314 EPSTEIN-BARR VIRUS AND GUILLAIN-BARRE SYNDROME. *(E.)* Grose, C. (Albert Einstein Coll. Med., New York, N.Y.) and P. M. Feorino. *Lancet* 2(7790):1285-1287, 1972.

0315 C-PARTICLE VIRUSES IN CELL-FREE EXTRACT-INDUCED HAMSTER LEUKEMIA AND RAT RETICULOSES. *(Ger.)* Graffi, I. (Central Inst. Cancer Res. German Acad. Sci., Berlin, East Germany), E. Bender, T. Schramm, D. Bierwolf and A. Graffi. *Arch Geschwulstforsch* 40(1):1-11, 1972.

316 IMMUNOLOGIC STUDIES ON A C-TYPE VIRUS OF BOVINE CULTURES. *(E.)* Ferrer, J. F. U. Pennsylvania, Kennett Square), N. D. Stock and . Avila. *Proc Am Assoc Cancer Res* 13(March):11, 972.

317 HORMONAL ENHANCEMENT AND INHIBITION OF ADENOVIRUS TRANSFORMATION. *(E.)* Schaller, . P. (Ohio St. U., Coll. Vet. Med., Columbus), G. . Milo and D. S. Yohn. *Proc Am Assoc Cancer Res* 3(March):8, 1972.

318 PROPERTIES OF FIVE HAMSTER CELL LINES TRANSFORMED BY HERPES SIMPLEX VIRUS TYPE 2. *E.)* Duff, R. (M. S. Hershey Med. Ctr., Pennsylvania . U., Hershey) and F. Rapp. *Proc Am Assoc Cancer s* 13(March):8, 1972.

319 DRUG-INDUCED ENHANCEMENT OF VIRUS PRODUCTION IN LYMPHOCYTIC CELLS FROM A PATIENT TH HODGKIN'S DISEASE. *(E.)* Krishan, A. (Harvard d. Sch., Boston, Mass.). *Proc Am Assoc Cancer s* 13(March):7, 1972.

20 INFECTION AND TRANSFORMATION OF MAMMALIAN CELL CULTURES WITH FELINE SARCOMA VIRUS SV). *(E.)* Chan, E. W. (Natl. Cancer Inst., thesda, Md.), P. E. Schiop-Stansly and T. E. Connor. *Proc Am Assoc Cancer Res* 13(March):7, 72.

21 ULTRASTRUCTURE OF HAMSTER CELLS TRANSFORMED BY ULTRAVIOLET IRRADIATED HERPES MPLEX VIRUS TYPE 2. *(E.)* Glaser, R. (M. S. rshey Med. Ctr., Pennsylvania St. U., Hershey), Duff and F. Rapp. *Proc Am Assoc Cancer Res* (March):5, 1972.

0322 HELPER RELATED CHANGES IN PATHOLOGIC PROPERTIES OF DEFECTIVE FRIEND VIRUS (FV). *(E.)* Dawson, P. J. (U. Oregon Med. Sch., Portland) and A. H. Fieldsteel. *Proc Am Assoc Cancer Res* 13(March):3, 1972.

0323 METHYLATED POLYNUCLEOTIDES AS INHIBITORS OF THE DNA POLYMERASE OF MURINE LEUKEMIA VIRUS. *(E.)* Farrelly, J. G. (Biol. Division, Oak Ridge Natl. Lab., Tenn.), D. Joseph, L. Roberson, F. W. Tuominen, R. W. Tennant and F. T. Kenney. *Proc Am Assoc Cancer Res* 13(March):89, 1972.

0324 RESPONSE TO ERYTHROPOIETIN IN C57Bl MICE MADE POLYCYTHEMIC BY INFECTION WITH HOST-ADAPTED FRIEND LEUKEMIA VIRUS. *(E.)* McGarry, M. P. (Roswell Park Mem. Inst., Buffalo, N.Y.) and E. A. Mirand. *Proc Am Assoc Cancer Res* 13(March): 86, 1972.

0325 ETIOLOGY OF HUMAN OSTEOSARCOMA. *(E.)* Reilly, C. A., Jr. (Argonne Natl. Lab., Ill.), D. J. Pritchard and M. P. Finkel. *Proc Am Assoc Cancer Res* 13(March):80, 1972.

0326 ON THE INTRA-ENVELOPE INCORPORATION OF "DENSE MATERIAL" BY IGUANA HERPES VIRUS. *(E.)* Zeigel, R. F. (Roswell Park Mem. Inst., Buffalo, N.Y.) and H. F. Clark. *Proc Am Assoc Cancer Res* 13(March):67, 1972.

0327 *IN VITRO* DETECTION OF CELLULAR AND HUMORAL RESPONSES TO MURINE SARCOMA VIRUS (MSV) IN MICE. *(E.)* Lavrin, D. (Bionetics Res. Lab., Bethesda, Md.), M. Nunn and R. B. Herberman. *Proc Am Assoc Cancer Res* 13(March):66, 1972.

0328 GRANULOCYTOPOIESIS AND VIRULENT MURINE LEUKEMOGENIC VIRUS PRODUCTION IN LONG-TERM CULTURE. *(E.)* Soule, H. (Michigan Cancer Fdn., Detroit) and T. Maloney. *Proc Am Assoc Cancer Res* 13(March):65, 1972.

0329 ENHANCEMENT OF VIRAL TRANSFORMATION BY CHEMICAL CARCINOGENS. *(E.)* Casto, B. C. (BioLabs, Inc., Libertyville, Ill.), W. J. Pieczynski and J. A. DiPaolo. *Proc Am Assoc Cancer Res* 13(March):61, 1972.

0330 INHIBITION BY BROMODEOXYURIDINE (BUdR) OF DIMETHYL SULFOXIDE (DMSO)-STIMULATED ERYTHROID DIFFERENTIATION AND HEMOGLOBIN SYNTHESIS IN MURINE VIRUS-INDUCED LEUKEMIC CELLS. *(E.)* Scher, W. (Mount Sinai Sch. Med., City U. New York, N.Y.), H. D. Preisler and C. Friend. *Proc Am Assoc Cancer Res* 13(March):105, 1972.

0331 ASSOCIATION OF RIBONUCLEASE ACTIVITY WITH A MURINE LEUKEMIA VIRUS. *(E.)* McClain, K. (Dept. Path., Pediatrics, U. Chicago, Ill.) and W. H. Kirsten. *Proc Am Assoc Cancer Res* 13(March): 103, 1972.

0332 INTERACTION OF SV40 WITH SKIN FIBROBLASTS FROM PATIENTS WITH XERODERMA PIGMENTOSUM. *(E.)* Bowen, J. M. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), D. Mukerjee, M. D. Scanlon, J. Bettinger, J. M. Trujillo and L. Dmochowski. *Proc Am Assoc Cancer Res* 13(March):99, 1972.

0333 ENHANCEMENT OF FRIEND LEUKEMIA VIRUS (FLV) LEUKEMOGENIC POTENTIAL INDUCED BY FLV-ANTISERUM. *(E.)* Rossi, G. B. (Superior Inst. Hlth., Rome, Italy), M. Marchegiani, R. Gentile and L. Marcucci. *Proc Am Assoc Cancer Res* 13(March):100, 1972.

0334 SUPPRESSION OF MOLONEY LEUKEMIA VIRUS SYNTHESIS BY POLYADENYLIC ACID. *(E.)* Tennant, R. W. (Biol. Division, Oak Ridge Natl. Lab., Tenn.), F. T. Kenney and F. W. Tuominen. *Proc Am Assoc Cancer Res* 13(March):98, 1972.

0335 BONE MARROW CELL POPULATION CHANGES DURING ABELSON VIRAL LEUKEMOGENESIS. *(E.)* Lawrence, M. (Boston U., Mass.), J. Albright and R. Siegler. *Proc Am Assoc Cancer Res* 13(March): 97, 1972.

0336 STUDIES OF FACTORS INFLUENCING RECOVERABILITY OF THE SHOPE RABBIT PAPILLOMA VIRUS FROM KANSAS COTTONTAILS. *(E.)* Rogers, S. (Biol. Division, Oak Ridge Natl. Lab., Tenn.). *Proc Am Assoc Cancer Res* 13(March):97, 1972.

0337 AN INCREASE IN DEOXYCYTIDINE KINASE (CK) ACTIVITY IN CELLS INFECTED WITH HERPES VIRUS. *(E.)* Goz, B. (Yale U. Sch. Med., New Haven, Conn.). *Proc Am Assoc Cancer Res* 13(March): 26, 1972.

0338 TRANSFORMATION OF RHESUS CELLS BY M-PMV. *(E.)* Pienta, R. J. (Bionetics Res. Lab., Inc., Bethesda, Md.), T. Hurt, C. Smith, D. L. Fine and J. C. Landon. *Proc Am Assoc Cancer Res* 13(March):25, 1972.

0339 LOCALIZATION OF CONSTITUENTS IN AVIAN MYELOBLASTOSIS VIRUS (AMV). *(E.)* Stromberg, K. (Natl. Cancer Inst., Bethesda, Md.), R. R. Gantt, S. H. Wilson, M. Litwack and B. Julian. *Proc Am Assoc Cancer Res* 13(March):23, 1972.

0340 CHEMISTRY OF PLASMA MEMBRANE ISOLATED FROM *IN VITRO* AND *IN VIVO* VIRUS-TRANSFORMED CELLS. *(E.)* Perdue, J. F. (McArdle Lab. Cancer Res., U. Wisconsin, Madison). *Proc Am Assoc Cancer Res* 13(March):19, 1972.

0341 ALLOGENEIC CHEMOIMMUNOTHERAPY OF A MOLONEY LYMPHOMA. *(E.)* Fefer, A. (U. Washington, Sch. Med., Seattle). *Proc Am Assoc Cancer Res* 13(March):17, 1972.

0342 ADOPTIVE CHEMOIMMUNOTHERAPY OF A FRIEND VIRUS-INDUCED LYMPHOMA. *(E.)* Fass, L. (U. Washington, Seattle) and A. Fefer. *Proc Am Assoc Cancer Res* 13(March):17, 1972.

0343 EARLY STIMULATION OF NUCLEAR ACIDIC PROTEINS BY SV40 INFECTION OF PERMISSIVE AND NON-PERMISSIVE CELLS. *(E.)* Rovera, G. (Dept. Path., Temple U. Sch. Med., Philadelphia, Pa.), R. Baserga and V. Defendi. *Proc Am Assoc Cancer Res* 13(March):15, 1972.

0344 ENVIRONMENTAL CONTROL OF VIRAL REPLICATION IN FROG TUMORS. *(E.)* McKinnell, R. G. (U. Minnesota, Minneapolis), V. L. Ellis, D. C. Dapkus and L. M. Steven, Jr. *Proc Am Assoc Cancer Res* 13(March):11, 1972.

0345 PROPERTIES OF A DEFECTIVE MAMMALIAN SARCOMA VIRUS. *(E.)* Gazdar, A. (Natl. Inst. Hlth., Bethesda, Md.), P. Sarma, P. Peebles and H. Chopra. *Proc Am Assoc Cancer Res* 13(March): 55, 1972.

0346 ACTINOMYCINS WHICH INHIBIT AVIAN SARCOMA VIRUS RNA-DIRECTED DNA SYNTHESIS AND PREVENT VIRUS-INDUCED SARCOMA FORMATION *IN VIVO*. *(E.)* Apple, M. A. (U. California Med. Ctr., San Francisco), E. Wildenradt, L. Osofsky, P. Widrin, M. Friedman, C. Haskell and J. Paganelli. *Proc Am Assoc Cancer Res* 13(March):50, 1972.

0347 RIBOSOMES AND VIRUS SYNTHESIS IN THE MOUSE LYMPHOMA. *(E.)* Petrea, I. (Yale U. Sch. Med., New Haven, Conn.) and W. U. Gardner. *Proc Am Assoc Cancer Res* 13(March):44, 1972.

0348 RAUSCHER VIRUS-INDUCED MYELOMONOCYTIC LEUKEMIA CULTURE LINES. *(E.)* Ikawa, Y. (Natl. Cancer Inst., Bethesda, Md.), T. N. Fredrickson, H. L. Sims, H. J. Ortego and H. C. Chopra. *Proc Am Assoc Cancer Res* 13(March):41, 1972.

0349 ACTIVE-SITE-DIRECTED INHIBITORS OF RAUSCHER LEUKEMIA VIRUS (RLV) RNA-DEPENDENT DNA POLYMERASE. *(E.)* Schrecker, A. W. (Natl. Cancer Inst., Bethesda, Md.), R. C. Gallo and M. B. Sporn. *Proc Am Assoc Cancer Res* 13(March):39, 1972.

0350 CELL CYCLE ANALYSIS AND EFFECTS OF ANTIMETABOLITES ON HERPES SIMPLEX VIRUS INDUCED CHROMOSOME CHANGES. *(E.)* O'Neill, F. J. Hershey Med. Ctr., Pa.). *Proc Am Assoc Cancer Res* 13(March):36, 1972.

0351 QUANTITATIVE ASPECTS OF EB-VIRUS INFECTION. *(E.)* Horoszewicz, J. S. (Roswell ark Mem. Inst., Buffalo, N.Y.), L. Kosuda and S. . Leong. *Proc Am Assoc Cancer Res* 13(March):35, 972.

See also:

* (Rev): 0002, 0003, 0008, 0014, 0016, 0022
* (Chem): 0034, 0080
* (Phys): 0217
* (Immun): 0359, 0361, 0362, 0366, 0367, 0368, 0377, 0379, 0387, 0388, 0393, 0407, 0409, 0410, 0412, 0415, 0421, 0423, 0426, 0431, 0434, 0445, 0447, 0454, 0463, 0464, 0468, 0471, 0474, 0478, 0479, 0502, 0504, 0507, 0514
* (Path): 0541
* (Epid-Biom): 0572, 0575

0352 THE ONTOGENY OF ALPHA-FOETOPROTEIN IN THE FOETAL AND NEONATAL RABBIT, AND ITS EXPERIMENTAL INDUCTION IN ADULT RABBITS. *(E.)* Branch, W. R. (Dept. Zool., U. Southampton, England). *Int J Cancer* 10(3):451-457, 1972.

Concentrations of α-fetoprotein (AFP) in sera, embryonic fluids and fetal fluids of pregnant rabbits was determined by immunoelectrophoresis at various stages of gestation. AFP was also assayed in rabbits with liver lesions induced by carbon tetrachloride i.p. or N.nitrosodiethylamine (DEN) feeding. AFP could be found in all fetal fluids. AFP concentrations peaked between 24-26 days of gestation, decreasing thereafter; newborn serum at 28 days postpartum lacked detectable AFP. Between 20-25 days of gestation, the concentration of AFP in fetal serum rose from 327-525 mg/100 ml. In rabbits aged seven to 24 wk which had been given carbon tetrachloride, sera contained AFP only in animals less than 13 wk old. DEN-induced hepatoma-bearing rabbits also showed AFP.

0353 LOCALIZATION OF TUMOUR SPECIFIC ANTIGEN ON EXTERNAL SURFACE OF PLASMA MEMBRANE. *(E.)* Dickinson, J. P. (Newcastle Gen. Hosp., Newcastle upon Tyne, England), E. A. Caspary and E. J. Field. *Nature New Biol* 239(93):181-183, 1972.

The macrophage electrophoresis method was used to determine the location of tumor-specific antigen on human cancer cells. Antigen-bearing human cancer cell lines, including HeLa, Hep-2 and KB, were reacted with sensitized lymphocytes to liberate a slowing factor which reduced the mobility of normal guinea-pig macrophages. Lymphocytes, taken from 12 cancer patients, slowed macrophage action to various degrees; slowing was mediated through cancer lymphocytes in all cases. Since intact tumor cells could effectively be substituted for cancer basic protein as antigen, it was concluded that antigenic determinants, which stimulated lymphocytes to produce macrophage slowing factor, were located on the cell surface. Antigenic activity could be transferred from surfaces of cancer cells to the ambient fluid by trypsin treatment. The association of the tumor antigen with the cell plasma membrane was further confirmed by cell fractionation.

0354 STUDIES ON THE TUMOR-HOST RELATIONSHIP. IV. TUMOR CELL AGGLUTINATION WITH SURVIVAL MOUSE SERUM. *(E.)* Tanino, T. (Inst. Med. Sci., U. Tokyo, Japan) and T. Tsumita. *Jap J Exp Med* 42(4):401-413, 1972.

C3H/He mice were inoculated i.p. with cells of a spontaneous syngeneic mammary carcinoma (MM2); serum (RS) was collected from survivors and mixed with MM2 cells. RS agglutinated 70-80% of MM2 cells; serum from normal mice did not agglutinate MM2 cells. RS failed to agglutinate normal mouse lymphatic cells, did agglutinate Ehrlich ascites tumor cells and sarcoma 180 cells, and failed to agglutinate MH 134 cells (a C3H/He mouse hepatoma). MM2 cells collected 7-11 days after inoculation of tumor cells were agglutinated by RS, but not MM2 cells collected 12-20 days postinoculation. Maximal agglutination of MM2 cells by RS was seen at 25 C; agglutination proceeded at a constant rate at pH 6.5-7.4. Agglutination was stable against heating at 60 C for 30 min, but was inactivated by 65 C heat. Agglutinating factors were found in the α- and β-globulin fractions of RS. These fractions, isolated, were six to eight times more active as agglutinators than RS. Agglutinating factors were fixed to the MM2 cell surfaces in the presence of Ca and Mg divalent ions. RS agglutination was inhibited by galactose, lactose and galactosamine.

0355 ANTIBODY-MEDIATED SUPPRESSION OF GRAFTED LYMPHOMA CELLS. II. PARTICIPATION OF MACROPHAGES. *(E.)* Shin, H. S. (Johns Hopkins U. Sch. Med., Baltimore, Md.), N. Kaliss, D. Borenstein and M. K. Gately. *J Exp Med* 136(2):375-380, 1972.

C3HeB mice were inoculated with 6C3HED lymphoma cells mixed with an anti-lymphoma alloantibody preparation. Growth of the lymphoma was found to be suppressed. However, graft growth was not suppressed in mice given 500 R wholebody irradiation one day before inoculation of the lymphoma-antibody mixture. Ability to suppress tumor growth was restored in irradiated mice by three days after irradiation. Whem lymphoma cells were mixed with normal mouse peritoneal cells and inoculated in irradiated hosts, tumor growth was suppressed. Spleen, thymus, lymph node or bone marrow cells, mixed with lymphoma cells, did not suppress lymphoma growth in irradiated hosts. Peritoneal macrophages mixed with tumor cells suppressed tumor growth in irradiated mice.

0356 IMMUNOLOGICAL ANALYSIS OF PARTICULATE ANTIGENS OF HUMAN LEUKEMIA. *(E.)* Lee, R. (Dept. Med. Microbiol., U. Manitoba, Winnipeg, Canada) and J. B. G. Kwapinski. *Can J Microbiol* 13(12):1789-1792, 1972.

Livers and spleens of eight leukemia cases were obtained at autopsy; seven of the cases were acute and the eight cases together represented six different types of leukemia. Organs were minced and centrifuged and hexagonal virus-like particles, with diameters of 80-90 nm, and having a central core and a surrounding capsid, were isolated. These particles were reacted in immunodiffusion and immunoferritin tests with antisera prepared in rabbits by injection with purified leukemic virus-like particles or with crude antigen preparations from normal human liver and spleen. The virus-like particles reacted with all antisera to leukemic particulate antigen, but did not react with any of the antisera to normal human tissue.

357 ULTRASTRUCTURAL OBSERVATIONS ON THE
HUMAN MYELOMA CELLS SECRETING γ-GLOBULIN:
PROPOSAL OF POSSIBLE MECHANISM OF SECRETION VIA
ESICLE. *(E.)* Shigematsu, T. (Hamanomachi Hosp.,
ukuoka, Japan) and K. Hattori. *Acta Haematol Jap*
5(1):76-82, 1972.

one marrow material from 16 patients with IgG-type
ultiple myeloma was examined under the electron
icroscope. Myeloma cells showed a well-developed
ough endoplasmic reticulum, prominent Golgi appara-
us and uniformity of maturation. Vesicles were seen
t the peripheries of cytoplasm in some cells;
imilar vesicles were also seen outside cells.
xtracellular vesicles appeared to contain material
f less electron density than that in vesicles
nside the cells. Vesicles were thought to have
role in IgG secretion by myeloma cells. Cyto-
lasmic dense bodies, often associated with Golgi
odies and vesicles, were also seen in myeloma cells.
hese bodies were also thought to be involved in IgG
ompletion and secretion.

358 THE USE OF QUANTITATIVE IMMUNOFLUORESCENCE
TO MEASURE IMMUNOGLOBULIN LEVELS IN
OUSE MYELOMA CELLS DURING THE CELL CYCLE. *(E.)*
andini-Attardi, D. (Washington U. Sch. Med., St.
ouis, Mo.), J. B. Fleischman, O. S. Pettengill
nd G. D. Sorenson. *Cell Immunol* 2(1):101-113,
971.

ells of a C3H mouse plasmacytoma which secreted
G-myeloma protein were stained with a fluorescent
oat antiserum to mouse-γG immunoglobulin, and
hotometry was used to determine levels of stained
mmunoglobulin in myeloma cells in various phases of
he cell cycle. The majority of fluorescence was
een in cells in the *S* cycle; this suggested that
mmunoglobulin content of cells was maximal in *S*.
hen cloned plasmacytoma cells in mitosis were
tudied, metaphase cells were clearly less fluores-
ent than intermitotic cells.

359 TRANSPLANTATION IMMUNITY TO SIMIAN VIRUS 40-
TRANSFORMED CELLS IN TUMOR-BEARING MICE.
I. EVIDENCE FOR MACROPHAGE PARTICIPATION AT THE
FFECTOR LEVEL OF TUMOR CELL REJECTION. *(E.)*
arling, J. M. (Baylor Coll. Med., Houston, Texas)
nd S. S. Tevethia. *J Natl Cancer Inst* 50(1):
49-157, 1973.

xperiments were performed to determine if normal
acrophages are recruited *in vivo*, as a result of
pecific interaction between immune lymphoid cells
nd tumor cells, to serve an effector role in the
nhibition of tumor development by simian virus 40
SV40)-transformed cells in syngeneic BALB/c mice.
he results obtained indicate that spleen cells
rom mice with tumors induced by SV40-transformed
ells (TU-5 line) could prevent tumor development
admixed TU-5 cells when inoculated together
nto normal adults at ratios as low as 1:1.

Tumor-neutralizing activity of the immune lymphoid cells was not diminished after treatment of lymphoid cells with 500 R γ-radiation. Neutralizing activity of immune lymphoid cells was lost after 2000 R. Tumor cell neutralization by immune lymphoid cells was less efficient in newborn mice and adult mice given 500 R whole-body γ-radiation, which suggests that immune lymphoid cells cooperate with cells lacking in number or function in newborn and irradiated mice. Normal bone marrow cells supplied the function necessary for tumor cell neutralization by immune lymphoid cells in irradiated mice (700 R). Tumor cell neutralization by immune lymphoid cells occurred less efficiently in mice treated with silica, a specific macrophage toxin. Thus normal macrophages are involved in tumor cell rejection *in vivo* as a consequence of specific immune lymphoid cell-tumor cell interaction.

0360 A TUMOR-ASSOCIATED ANTIGEN IN GASTRIC CANCER SECRETIONS. *(E.)* Deutsch, E. (Carney Hosp., Boston, Mass.), C. A. Apffel, H. Mori and J. E. Walker. *Cancer Res* 33(1):112-116, 1973.

An antigen, distinct from Gold's carcinoembryonic antigen, was identified in 91% of gastric cancer secretions (GCJ). Reacted with rabbit immune serum against GCJ, this antigen appears as one precipitin line among others on double diffusion in agar gel. When the immune serum has been absorbed with non cancer secretions, it gives a distinctly prevalent line with GCJ both on immunodiffusion and by immunoelectrophoresis. After absorption with normal human plasma, the gastric cancer antigen appears on immunoelectrophoresis as a sharp single line in the β region with a mobility greater than Gold's carcinoembryonic antigen. Gastric cancer antigen has an immunologically reactive carbohydrate moiety and is different from blood group substances. It was not detected in the gastric tumor itself. The corresponding precipitin line disappeared when the immune serum was absorbed with GCJ, thus establishing its pathognomonic significance. Detection of gastric cancer antigen may be crucial for early diagnosis, particularly in younger, still salvageable patients, when there is free HCl on gastric analysis.

0361 EXPRESSION OF POLYOMA-INDUCED TRANSPLANTATION ANTIGEN IN HYBRID CELL LINES. *(E.)* Klein, G. (Karolinska Inst., Stockholm, Sweden) and H. Harris. *Nature New Biol* 237(75):163-164, 1972.

Twelve different clonal lines obtained from hybrids of polyoma virus-induced SEWA sarcoma of ASW(H-2^s) origin and diploid CBA(H-2^k) fibroblasts homozygous for the T6 translocation were studied for their ability to express polyoma-induced transplantation antigen (PTA). The clones could be divided into three lines with a low take-incidence and nine lines with a high take-incidence in syngeneic newborn x-irradiated ASWxCBA F_1 hybrids. Twenty-four tumor-free syngeneic adults which arose from newborns that had survived the s.c. inoculation of the low take-incidence clones were challenged by reinoculation with SEWA cells. One-half of these animals developed tumors compared with a 100% incidence in SEWA inoculated untreated adult controls. The low and high take-incidence lines could be compared by challenging the semi-allogeneic survivors (i.e. ASW mice x hybrids having ASW as one parent and a non-H-2^k strain as the other) with SEWA tumor cells. The take incidences in these cases were 33% for the low take-incidence clones and 41% for the high take-incidence lines. All untreated adult control mice developed tumors upon SEWA cell inoculation. These results indicated that the SEWA/CBA hybrids of low tumorigenicity did not lose PTA since these cells were immunogenic in both syngeneic and semi-allogeneic mice. Since PTA is probably controlled by the viral genome, the persistence of PTA in weakly tumorigenic hybrids implies that the viral genome, and the surface alteration associated with PTA, are not sufficient by themselves to determine malignant behavior *in vivo*.

0362 EPSTEIN-BARR VIRUS-ASSOCIATED ANTIGENS IN NON-PRODUCING CLONES OF HUMAN LYMPHOBLASTOID CELL LINES. *(E.)* Sugawara, L. (Hokkaido U. Sch. Med., Sapporo, Japan), F. Mizuno and T. Osato. *Nature New Biol* 239(95):242-243, 1972.

Several clonal lines from NC-37 and Raji, two non-Epstein-Barr virus (EBV)-producing lines, were studied for their ability to express EBV-associated antigens. All 23 NC-37 and all 27 Raji clones exposed to 5-bromodeoxyuridine or 5-iododeoxyuridine showed the production of both EBV-associated early antigens and viral capsid antigen as detected by indirect immunofluorescence studies. These findings strongly suggest that an unexpressed form of the EBV genome is present in all of the cells of the non-EBV-producing NC-37 and Raji lines.

0363 ANTIGENIC STRUCTURE OF SPONTANEOUS LYMPHATIC LEUKEMIA CELLS IN MICE OF THE INBRED AKR STRAIN. *(E.)* Harlozinska, A. (Polish Acad. Sci., Wroclaw) and R. Richter. *Arch Immunol Ther Exp (Warsz)* 19:801-829, 1971.

Tissue sections and cells of a lymphatic leukemia developing spontaneously in thymus of AKR mice naturally infected with Gross virus were reacted in immunofluorescence tests with allogenic immune sera prepared from C57Bl mice immunized with thymus or thymic tumor cells from AKR mice and with heterologous immune sera from rabbits immunized with AKR thymus and thymus tumor homogenates. Also tested were thymus cells of young AKR mice before onset of leukemia, lymphoid tissue of neonatal AKR mice, and thymus from the low-leukemia C57Bl mice. The antigenic structures of the test cells were compared. Peripheral fluorescence surrounding leukemia cells was seen after treating cells with allogenic sera. This reaction was thought to indicate the presence of an antigen produced during malignant transformation of cells. This antigen was characterized as a specific leukemia antigen. A different fluorescence pattern was seen when leukemic cells were reacted with allogenic anti-thymus serum. These cells showed only monofocal punctuate fluorescence. This reaction was thought to indicate the presence of an antigen which was a structural fragment of mature infectious Gross virus. The antigen was characterized as a specific viral antigen. Newborn lymphoid tissue and thymus of young AKR mice possessed only the specific viral antigen. Both antigens were localized on cell surfaces. Fluorescence reactions were not obtained with C57Bl mouse thymus cells. Fluorescence reactions were also obtained when tissue sections of thymus tumors were reacted with allogenic sera.

0364 EXPRESSION OF A NEW COMPLEMENT-FIXING ANTIGEN REACTIVE WITH MURINE SARCOMA VIRUS RAT ANTISERUM IN RAT CELLS TRANSFORMED BY POLYOMA VIRUS. *(E.)* Rhim, J. S. (Microbiol. Assoc., Inc., Bethesda, Md.), C. R. Lengel, H. Y. Cho, K. K. Takemoto, H. C. Turner, R. J. Huebner and R. V. Gilden. *Nature New Biol* 235 (58):188-190, 1972.

Rat embryo cells (RE) and the 39th and 53rd subculture passage were transformed by polyoma virus and reacted in complement fixation tests against broadly reactive rat antisera obtained from rats carrying transplanted sarcomas induced by Moloney murine sarcoma virus (M-MSV). In addition to the polyoma virus T antigen, a new antigen was activated in polyoma-transformed cells; this antigen was reactive with antisera produced in rats carrying the M-MSV sarcoma. The appearance of this new antigen in RE cells was correlated with the onset of polyoma virus transformation. The antigen persisted in tumors induced in rats by inoculation of polyoma-transformed cells. The new antigen seen in polyoma-transformed RE cultures was different from that seen in Rauscher leukemia virus-infected RE cells transformed by polyoma virus. RE cells transformed by polyoma virus, and rat tumors induced by these cells, did not react with murine sarcoma virus guinea-pig antisera. Furthermore, these tumors contained polyoma T antigen but not polyoma viral antigen. Thus, the new antigen is not the intraspecies gs-1 antigen of mice.

0365 TUMOR IMMUNITY TO MURINE PLASMA CELL TUMORS. I. TUMOR-ASSOCIATED TRANSPLANTATION ANTIGENS OF NZB AND BALB/c PLASMA CELL TUMORS. *(E.)* Röllinghoff, M. (Hall Inst. Med. Res., Melbourne, Australia), B. T. Rouse and N. L. Warner. *J Natl Cancer Inst* 50(1):159-172, 1973.

The presence of tumor-associated transplantation antigens on plasma cell tumors (PCT) derived from

ALB/c and NZB inbred and hybrid mice was demonstra-ed by the induction of immunity *in vivo* against alignant tumor challenge. Immunizations were ade by viable tumor cell inoculation followed by emoval of the tumor. Whereas most NZB-derived CT were highly effective at inducing immunity gainst all NZB PCT, only 1 of 5 BALB/c-type PCT howed similar immunogenicity. Immunization with illed or modified cells was either ineffective or arkedly inferior to the use of viable cells. he state of immunity was adoptively transferred with pleen cell suspensions at ratios as low as one pleen cell to one tumor cell. No evidence that CT-specific antibodies existed was found. In oth *in vivo* challenge and adoptive transfer studies, munity, when detectable, was specific for PCT. t is suggested that there is a shared PCT-specific ntigen, which may be associated with a subtype of the ross virus. The antigen is present to a considerably reater degree on NZB PCT than on most BALB/c PCT.

66 IMMUNOLOGIC STIMULI IN REALTION TO LEUKEMOGENESIS. I. POTENTIATION OF USCHER VIRAL LEUKEMIA BY FREUND'S ADJUVANTS. *(E.)* egel, B. V. (U. Oregon, Med. Sch., Portland) and I. Morton. *J Nat Cancer Inst* 48(6):1681-1685, 72.

LB/c female mice were given one i.p. injection of eund's complete adjuvant (CFA) at different times efore and after i.p. or i.v. injection of Rauscher ukemia virus (RLV). Increased leukemic deaths were ted in mice given CFA ten days before to three days ter RLV. Of 40 mice receiving CFA two days before V inoculation, 13 were dead within 47 days. In ntrast, only two of 30 mice receiving RLV alone re dead by day 47. Endogenous spleen colony rmation increased tenfold in mice receiving CFA ree days prior to exposure to 500 R whole body irradiation. Similar adjuvant administration fore immunization with sheep erythrocytes produced ofold to threefold increases in peak numbers of leen antibody plaque-forming cells. Incomplete eund's adjuvant (IFA) also increased leukemia rtality in BALB/c mice; all IFA-treated mice died ring the first six weeks of infection as compared th less than 60% of RLV-only mice. An increased ral target-cell population rather than immunologic hancement appears responsible for the potentiation Rauscher leukemogenesis by adjuvant treatment.

67 INDUCTION OF ANTINUCLEAR ANTIBODIES IN (C X B6) F1 MICE INOCULATED WITH GRAFFI D RAUSCHER LEUKEMOGENIC VIRUSES. *(E.)* Cannat, (Saint-Louis Hosp., Paris, France) and B. ret. *Proc Soc Exp Biol Med* 141(3):1077-1080, 72.

ALB/c x C57/B16) F1 hybrid mice maintained in a thogenfree environment and inoculated i.p. with purified Graffi or Rauscher leukemia virus (RLV) eparations developed antinuclear antibodies which were detectable in their sera by indirect immunofluorescence assay. Antinuclear antibodies were present in significantly higher levels in mice inoculated with the more leukemogenic virus strains than in mice inoculated with less leukemogenic strains. Little or no antibody was detected in sera of control mice inoculated with nonvirus-infected spleen cell sonicates, nor in mice inoculated with specifically neutralized RLV. Antinuclear antibody production did not correlate with the appearance or growth or leukemias. Antibody activity of pooled positive sera could almost be completely eliminated by absorption with calf thymus nucleoproteins. Homogenous staining of rat liver nuclei by antibody-positive sera could be prevented by both DNase and trypsin pretreatment.

0368 TRANSPLANTATION IMMUNITY TO SIMIAN VIRUS 40-TRANSFORMED CELLS IN TUMOR-BEARING MICE. I. DEVELOPMENT OF CELLULAR IMMUNITY TO SIMIAN VIRUS 40 TUMOR-SPECIFIC TRANSPLANTATION ANTIGENS DURING TUMORIGENESIS BY TRANSPLANTED CELLS. *(E.)* Zarling, J. M. (Baylor Coll. Med., Houston, Texas) and S. S. Tevethia. *J Natl Cancer Inst* 50(1):137-147, 1973.

The immune response of inbred BALB/c mice to tumor-specific transplantation antigens (TSTA) of transplanted syngeneic cells transformed by simian virus 40 (SV40) was studied throughout the period of tumorigenesis. Immune lymphoid cells, capable of specificially preventing or delaying tumor growth of admixed SV40-transformed mouse or hamster cells when inoculated into adult hosts, were detected in lymph nodes, spleens, and peritoneal exudates of mice with established tumors. Furthermore, tumor-bearing mice resisted high secondary challenges of SV40-transformed cells when inoculated at a site distant from the primary inoculation site, which thus demonstrated the presence of a functional effector mechanism for tumor rejection. By four days after the inoculation of 10^5 SV40-transformed cells, mice could resist challenges of similar doses of the virus-transformed cells. Transformed cells inoculated only a day after a previous inoculation at a distant site were delayed in developing a tumor; however, the rate of development of the tumor at the first site was unaffected. These results indicate that cellular immunity to SV40 TSTA develops early during tumorigenesis by transplanted cells; nevertheless, the tumors develop and grow progressively. It is suggested that either the level of immunity is not sufficient during early tumorigenesis to prevent the tumor from establishing or, alternatively, there is a local efferent block at the site of tumor development.

0369 EFFECT OF TUMOR IMMUNITY ON PRODUCTION OF LUNG TUMORS AFTER INTRAVENOUS INOCULATION OF ANTIGENICALLY IDENTICAL TUMOR CELLS. *(E.)* Wexler, H. (Natl. Cancer Inst., Bethesda, Md.), P. B. Chretien and A. S. Ketcham. *J Nat Cancer Inst* 48(3):657-663, 1972.

C57BL/6N mice were inoculated i.v. or i.m. in the legs with cells of an immunogenic methylcholanthrene-induced tumor (MCA-1); tumor-bearing legs were amputated when tumors had attained diameters of 0.8-1.0 cm. Some mice were challenged i.m. or i.v. with MCA-1 cells and tumor production in legs or lungs by challenge by different routes was observed. Two of 19 mice pretreated with MCA-1 cells injected i.m. developed leg tumors after i.m. challenge while 18 of 20 mice given no pretreatment developed leg tumors after i.m. challenge. Seventeen of 18 mice pretreated with i.v. injections of tumor cells developed lung tumors after i.v. challenge while 19 of 20 mice not pretreated with tumor cells developed lung tumors after i.v. challenge. Differences in tumor incidence occurring after inoculation of pretreated mice were related to route of inoculation. The percent of mice with lung tumors after i.v. inoculation of tumor was higher than the percent of mice with leg tumors at the site of i.m. inoculation. Furthermore, the numbers of tumors produced with 1000 challenge tumor cells was only slightly greater than that produced with 100 tumor cells in i.v. or i.m. challenged mice. A non-immunogenic methylcholanthrene-induced tumor (MCA-3) produced similar leg tumor incidences when inoculated i.v. or i.m. into pretreated mice. Inoculation i.v. with MCA-3 and MCA-4 (weakly immunologenic) tumor cells produced similar incidences of lung tumors in pretreated and non-pretreated mice. Data indicated that immunization with the immunogenic MCA-1 tumor facilitated the survival and propagation of these tumor cells when they were inoculated i.v.

0370 AUTORADIOGRAPHIC AND FLUORESCENCE STUDIES OF THE HUMAN HOST IMMUNE RESPONSE TO GLIOMAS. *(E.)*

Eggers, A. E. (Columbia U., Coll. Phys. Surg., New York, N.Y.). *Neurology* 22(3): 246-250, 1972.

Stimulation of autologous lymphocytes in tissue culture by human glioma cells was observed. Monolayer cultures were prepared from tissues from six gliomas (including three astrocytomas) and from malignant tumors arising outside the central nervous system (control tumors). Serum and lymphocytes were taken from the glioma patients and from a normal donor (control serum and lymphocytes). Glioma cells were incubated with autologous lymphocytes or with control lymphocytes; other glioma cells were incubated with autologous lymphocytes plus autologous serum, or with autologous lymphocytes plus control serum. Lymphocyte stimulation was detected by autoradiography. Three of the gliomas were also studied for antigens by the indirect immunofluorescence antibody technique. All six gliomas showed stimulation of autologous lymphocytes, as indicated by aggregation of lymphocytes around tumor cells. In none of the six cases did control lymphocytes aggregate around tumor cells. In no case did autologous or control serum inhibit lymphocyte stimulation by glioma cells. In all four control tumors, autologous but not control lymphocytes were stimulated by tumor cells, and in all four cases autologous but not control serum inhibited lymphocyte stimulation. Fluorescence antibody staining was positive for three control tumors and for one of the astrocytomas. Cross-reactions were tested for in the cases of the gliomas, each time with the same pattern of control lymphocytes and control serum. Each glioma was cross-reacted with from one to three other gliomas. All patterns were positive for lymphocyte stimulation and negative for serum inhibition of lymphocyte stimulation.

0371 DIMENSIONS OF HUMORAL RECOGNITION FACTOR DEPLETION IN CARCINOMATOUS PATIENTS. *(E.)*

Pisano, J. C. (Tulane U. Sch. Med., New Orleans, La.), J. P. Jackson, N. R. DiLuzio and H. Ichinose. *Cancer Res* 32(1):11-15, 1972.

The influence of serum from patients with carcinoma on the phagocytic activity manifested by Kupffer cells incubated in normal plasma was studied. Blood from normal individuals, varying in age from 21 to 50 years, and from patients with various carcinomas, recently diagnosed but untreated, was obtained by venipuncture. Humoral recognition factor (HRF) activity of serum was evaluated by means of its ability to support the phagocytosis of a variety of colloidal materials by either Kupffer cells of rat liver slices, isolated rat peritoneal macrophages, or human alveolar macrophages. Alveolar and peritoneal macrophages, as well as Kupffer cells, phagocytized significantly less colloidal material when incubated in plasma derived from carcinomatous patients than when incubated in plasma obtained from normal control subjects. No relationship was demonstrated between the presence of a phagocytosis-inhibiting factor and the inability of serum obtained from cancer patients to support phagocytosis of diverse colloids. The loss in humoral recognition factor activity in neoplasia, therefore, relates to either depletion of HRF or loss of essential cofactors or activators. The data suggest that the HRF aides the discriminative ability of the macrophage; therefore, the loss of an effective recognition mechanism may contribute to the impairment of immunological rejection mechanisms and to tumor growth and development.

0372 CELL MIGRATION INHIBITION IN HUMAN LYMPHOMAS USING LYMPH NODE AND CELL LINE ANTIGENS. *(E.)*

Braun, M. (Natl. Acad. Med., Buenos Aires, Argentina), L. Sen, A. Bachmann and A. Pavlovsky. *Blood* 39(3):368-376, 1972.

Peripheral white blood cells were prepared from eight lymphosarcoma (LS) patients, four Hodgkin's (HD) patients, one acute lymphatic leukemia (ALL) patient and two cancer patients with lymph node metastases. The leukocytes were cultured and cell migration was observed in cultures of lymphoma patients' and normals' leukocytes treated with autologous or allogeneic normal or neoplastic cells or lymph node cell extracts as antigen. Migration s tested in direct tests of peripheral leukocytes d antigens, and in indirect tests using super- tants of cultured lymphocytes from patients on inea pig peritoneal macrophages. Leukocytes from ur LS patients showed inhibited cell migration when eated with autologous antigen. Two of four HD tients' leukocytes showed inhibition of cell igration by autologous antigen. Autologous antigen hanced cell migration in three cultures of normal ukocytes. Enhancement of cell migration by tologous antigen was also seen in cells from one tient with lymph node metastasis and in cells from e ALL patient. Allogeneic antigen inhibited cell gration in four LS cases. No clear-cut inhibition cell migration was seen when HD leukocytes were eated with allogeneic antigen. No allogeneic tigen produced migration inhibition in leukocytes om normal donors. Cross-reactions observed between me LS cases suggested that common antigen(s) were cated in neoplastic lymph nodes.

73 MACROPHAGE RECOGNITION-FACTOR DEPLETION AFTER ADMINISTRATION OF PARTICULATE ENTS AND LEUKEMIC CELLS.

(E.) Di Luzio, N. R. ulane U., New Orleans, La.), R. McNamee, E. F. ller and J. C. Pisano. *J Reticuloendothel Soc* (3):314-323, 1972.

udies were undertaken in rats to evaluate the chanism by which recognition factors (opsonins) e depleted following i.v. administration of a latinized RE test lipid emulsion. Administration the gelatinized RE test lipid emulsion resulted a 90% depletion in plasma recognition-factor tivity as determined by the *in vitro* ability of rmal rat liver slices to phagocytize ^{125}I-triolein- beled gelatinized test emulsion in the presence plasma from injected rats. A comparable depletion s not observed when nongelatinized RE test lipid ulsion was used, indicating that the gelatin com- nent was responsible for the depletion of the cognition-factor. Also, i.v. administration of ukemic leukocytes, but not normal leukocytes, duced a 68% decrease in recognition-factor ggesting that a surface characteristic of the ukemic cells may share a common feature with at of the gelatin, which results in recognition- ctor depletion.

74 TUMOR IMMUNITY *IN VIVO*. USE OF THE ^{125}IUdR PRELABELING TECHNIQUE TO MONITOR L DEATH IN IMMUNE ANIMALS.

(E.) Forman, J. ept. Physiol., Tufts U. Sch. Med., Boston, Mass.), M. Ketchel and K. G. Hofer. *Transplantation* 2):166-174, 1972.

death of ^{125}I-iododeoxyuridine (^{125}IUdR) pre- eled L5178Y$_f$ cells was studied following i.p. culation into normal and preimmunized allogeneic e. The death rate of tumor cells injected into e sensitized to H-2 or to a complex of non-H-2 igens was much greater, as determined by the ex- t of urinary ^{125}I excretion, than that found in non-immunized control mice. Over 91% of the ^{125}I was excreted from H-2 or non-H-2 primed animals within two days. Spleen cells were as effective as tumor cells in sensitizing the mice to H-2 or non-H-2 antigens. All preimmunized mice survived whereas nonimmunized control mice invariably died following tumor inoculation. The response was specific since C57BL/105n mice immunized with DBA/2 tumor excreted ^{125}I at an accelerated rate, while the same animals challenged with a C3H tumor did not. ^{125}I was not reutilized to any significant extent within the first three days of the experiment.

0375 ONCOGENIC EFFECT OF REPEATED INOCULATION OF MICE WITH SHEEP ERYTHROCYTES.

(E.) Tyndall, R. L. (Biol. Div., Oak Ridge Natl. Lab., Tenn.), J. A. Otten and P. C. Estes. *Transplantation* 14(2):269-271, 1972.

Fourteen of 19 BALB/c mice injected twice weekly for 12 wk with 1 ml of a 10% suspension of sheep blood cells (SRBC) developed reticulum cell sarcomas. However, no malignancies were observed in subsequent experiments in identically treated mice. In additional experiments, newborn BALB/c were injected with 1 X 10^6 spleen cells from SRBC-treated BALB/c mice. Twenty-five of 87 recipient mice developed overt neoplasia, which were classified as reticulum cell sarcomas. The latent period between spleen cell inoculation and appearance of overt malignancy was 4-16 months. Histological examination of donor spleen tissue revealed germinal center hypertrophy but no evidence of malignancy. These observations suggest a control mechanism, perhaps immunological, by which treated donor mice are capable of repressing the neoplastic response to SRBC treatment.

0376 MALIGNANT TUMORS ARISING *DE NOVO* IN IMMUNOSUPPRESSED ORGAN TRANSPLANT RECIPIENTS.

(E.) Penn, I. (U. Colorado Sch. Med., Denver) and T. E. Starzl. *Transplantation* 14(4):407-417, 1972.

Data from 75 cases of *de novo* malignancies arising in immunosuppressed renal transplant recipients throughout the world were collected between 1968 and 1971 at the Denver tumor registry. The patients ranged in age from 3 1/2 to 53 yr (mean age of 28 yr). The tumor incidence of the at risk population (4.5%) was 80 times greater than that of a comparably aged normal population. Forty-four of the 76 cases, all of which have been followed for a period of from six months to nine years, developed squamous or basal cell skin carcinomas. Thirty-two mesenchymal tumors, 28 of which were lymphomas, were found in 31 patients. Of the 27 lymphoma patients, 14 showed involvement of the brain or spinal cord. Mesenchymal tumor appeared, on the average, 21 months following transplantation, whereas the mean period for appearance of the carcinomas was 35 months. None of the donors showed any evidence of malignant disease during the follow-up period. Carcinomas of the thoracic or abdominal organs and mesenchymal tumors had a poor prognosis and drastic reduction or discontinuance of immunosuppression should be considered in such cases. Although chronic uremia could not be excluded as an etiologic factor in the pathogenesis of these tumors, they were most likely secondary to chronic post-transplantation immunosuppression.

0377 PROGRESSIVE LOSS OF H-2 ANTIGENS WITH CONCOMITANT INCREASE OF CELL-SURFACE ANTIGEN(S) DETERMINED BY MOLONEY LEUKEMIA VIRUS IN CULTURED MURINE LYMPHOMAS. *(E.)* Cikes, M. (Karolinska Inst. Sch. Med., Stockholm, Sweden), S. Friberg, Jr. and G. Klein. *J Natl Cancer Inst* 50(2):347-362, 1973.

Two Moloney leukemia virus (MLV)-induced murine lymphomas (YCAB and YAC) and a spontaneous murine lymphoma (WL4) were serially propagated in the ascites form in syngeneic (A/Sn x A.CA)F_1 mice. At intervals, culture lines were established and maintained as stationary suspension cultures for a few days to three years. *In vitro* lines derived from all three lymphomas had less H-2 antigens and more MLV-determined cell-surface antigen(s) than the corresponding *in vivo* lines in syngeneic hosts. This was shown by the sensitivity of the cells to complement-dependent, antibody-mediated cytolysis, indirect membrane immunofluorescence, and quantitative absorption tests. The antigenic changes took place progressively after explanation and were correlated with the number of cell generations in culture. The final cell densities of the culture lines were also correlated with the number of cell generations in culture; e.g., a sevenfold increase in the final cell density was observed in a long-established YCAB culture line, compared with a newly established line of the same tumor.

0378 CELLULAR AND HUMORAL IMMUNITY TO RAT HEPATOMA-SPECIFIC ANTIGENS CORRELATED WITH TUMOUR STATUS. *(E.)* Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), M. J. Embleton and R. A. Robins. *Int J Cancer* 11(1):1-10, 1973.

Cellular and humoral immune reactions were studied in Wistar rats bearing either primary or transplanted aminoazo-dye-induced hepatomas. Thirteen of 17 lymph node lymphocyte preparations from tumor-bearing rats displayed *in vitro* cytotoxicity when tested against cells of the immunizing hepatoma. There was no correlation between *in vivo* tumor size and the degree of colony inhibition produced; however, the inhibitory effect was tumor-specific. Results from colony inhibition assays indicated that all sera from rats bearing primary hepatomas and eight of nine sera from rats bearing tumor grafts lacked complement-dependent cytotoxic antibody. Although sera from rats bearing hepatomas lacked detectable cytotoxic antibody, tumor bearer sera blocked the inhibitory activity of sensitized lymph node cells. Lymph node cells retained their cytotoxic ability for at least 20 days following excision of the tumor from the host. Sera from animals in which tumor regrowth did not occur reacted positively in colony inhibition tests in the presence of guinea-pig serum as a source of complement. Serum blocking activity in these rats disappeared within three days after tumor excision.

0379 EFFECTS OF BCG ON VARIOUS FACETS OF THE IMMUNE RESPONSE AGAINST POLYOMA TUMORS IN RATS. *(E.)* Bansal, S. C. (Pacific Northwest Res. Fdn., Seattle, Wash.) and H. O. Sjögren. *Int J Cancer* 11(1):162-171, 1973.

The effects of BCG on tumor growth *in vivo* and tumor immunity as assayed *in vitro* were studied. Administration of BCG intracutaneously either two wk before, or at the time of isografting of polyoma-induced sarcomas onto W/Fu strain rats, moderately inhibited tumor growth and stimulated the production of lymphocytes which were cytotoxic *in vitro* toward tumor target cells. Inoculation of BCG into rats with palpable tumor nodules, however, enhanced rather than inhibited tumor growth. Cell-mediated immunity was not enhanced under these conditions, and serum blocking activity was increased. BCG-treated tumor-bearing rats also showed no production of cytotoxic antibodies detectable with rat complement. Partial tumor excision followed immediately by BCG inoculation resulted in increased serum blocking activity and in an increased rate of growth of the remaining tumor tissue. However, when such BCG-treated partial tumor-bearing rats also underwent splenectomy and were inoculated with unblocking serum, cell-mediated immunity increased in all animals tested and tumors regressed in some.

0380 REGIONAL LYMPHATICS AND CANCER IMMUNITY. *(E.)* Futrell, J. W. (Natl. Cancer Inst., Bethesda, Md.) and G. H. Myers, Jr. *Ann Surg* 177(1):1-7, 1973.

The relationship of the regional lymphatics to the immunologic response to cancer was studied in inbred Sewall-Wright strain 2 female guinea pigs. Skin pedicles devoid of their afferent lymphatic drainage prevented early recognition of tumor-specific transplantation antigens. An inoculum of 5 x 10^5 syngeneic MCA-A liposarcoma cells, although insufficient to produce a progressive tumor when injected into intact skin, resulted in a large lethal tumor when injected into alymphatic skin pedicles despite the eventual development of an active antitumor immune response. These results demonstrate that regional lymphatics are not necessary for the eventual development of tumor-specific transplantation antigens. Removal of local lymphatics, once a tumor has eached a clinically detectable size, should not dversely affect the host's ability to mount an mmune response to the tumor since systemic umor-specific sensitization has already occurred.

381 CARCINOEMBRYONIC ANTIGEN IN PANCREATITIS. *(E.)* Delwiche, R. (Boston City Hosp., ass.), N. Zamcheck and N. Marcon. *Cancer* 1(2):328-330, 1973.

erum specimens of 42 patients with pancreatitis 36 alcoholics, 6 nonalcoholics) were analyzed or carcinoembryonic antigen (CEA). Eighteen atients (43%) with alcoholic pancreatitis were EA positive compared with none of the six patients ith nonalcoholic pancreatitis. Of the CEA-ositive patients, 12 had mild-to-moderate liver isease and six were clinically free of liver isease. CEA positivity did not correlate with erum amylase levels or liver function tests. uantitative levels of CEA were usually lower i.e., below 5 ng/ml) than those seen in dissemi-ated pancreatic carcinoma. Accordingly, caution s warranted in interpreting CEA tests in patients ith pancreatic disease. The possible role of moking in producing CEA positivity is discussed.

382 IMMUNOGLOBULIN ON TUMOR CELLS AND TUMOR-INDUCED LYMPHOCYTE BLASTOGENESIS IN JMAN ACUTE LEUKEMIA. *(E.)* Gutterman, J. U. J. Texas M. D. Anderson Hosp. Tumor Inst., Houston), . D. Rosen, W. T. Butler, K. B. McCredie, G. P. odey, Sr., E. J. Freireich and E. M. Hersh. *N Engl Med* 288(4):173-175, 1973.

ne interrelation of a lymphocyte blastogenic esponse to leukemia cells, the effect of the atient's own serum on this response, and the resence or absence of immunoglobulin on the umor cell surface was studied in 34 adults with cute leukemia. Nineteen of 24 patients with acute yelogenous leukemia and five of ten with acute ymphoblastic leukemia had a positive blastogenic esponse to their own tumor cells stimulated n their own or someone else's serum. Membrane mmunofluorescence analysis revealed immunoglobulin n the tumor cell surface in eight acute myelogenous eukemia patients but in none of the acute lympho-lastic leukemia patients. Only seven patients iowed both blastogenic inhibition and positive mmunofluorescence. There was a direct correlation etween degree of blastogenic response to autologous eukemia cells and clinical remission: 17 of 24 itients with a stimulation index greater than iree achieved at least temporary remission, while ily 9 of 14 patients with a stimulation index elow three went into remission. Sixteen (67%) the acute myelogenous leukemia group had a imulation index greater than three but only four 0%) of the acute lymphocyte leukemia patients iowed such a strong response. These results idicate the importance of regular immunologic aluation of patients with acute leukemia.

0383 STUDIES ON SURFACE ANTIGEN VARIANTS. ISOLATION OF TWO COMPLEMENTARY VARIANTS FOR THY 1.2. *(E.)* Hyman, R. (Salk Inst., San Diego, Calif). *J Natl Cancer Inst* 50(2):415-422, 1973.

Two variants for the antigen Thy 1.2 were characterized. These variants had <0.001 the amount of Thy 1.2 antigen present on parental cells; T1M1 cells, derived from a radiation leukemia virus-induced tumor of a C57BL/6 mouse, and BALB/c lymphoma S49.1TB.2 cells. Cell fusion studies indicated that absence of Thy 1.2 antigen was recessive in these variants and that the Thy 1.2 antigen was expressed when the two variants were fused to each other. Possible genetic or epigenetic explanations for the variants are discussed in terms of these results.

0384 DELAYED CUTANEOUS HYPERSENSITIVITY TO CELL-FREE TUMOR ANTIGENS. *(E.)* Holmes, E. C. (Natl. Cancer Inst., Bethesda, Md.), R. A. Reisfeld and D. L. Morton. *Cancer Res* 33(2):199-202, 1973.

The exposure of two different methylcholanthrene-induced strain 2 guinea pig sarcomas (MCA-A liposarcoma and MCA-25 osteosarcoma) to low-intensity sonic energy provided cell-free extracts that could elicit tumor-specific delayed cutaneous hypersensitivity reactions. These extracts, when fractionated by diethylaminoethyl column chromatography, yielded a partially purified component capable of eliciting delayed cutaneous hypersensitivity reactions in tumor-immunized syngeneic guinea pigs, which reactions were highly specific for the tumor from which the extract was derived. These partially purified antigens, which were derived from tumors induced by the same chemical agent, could thus evoke, in tumor-immunized syngeneic guinea pigs, cellular immune responses that are specific for the immunizing tumor.

0385 IMMUNOGLOBULIN ON TUMOR CELLS AND TUMOR-INDUCED LYMPHOCYTE BLASTOGENESIS IN HUMAN ACUTE LEUKEMIA. *(E.)* Gutterman, J. U. (U. Texas M. D. Anderson Hosp. Tumor Inst., Houston), R. D. Rossen, W. T. Butler, K. B. McCredie, G. P. Bodey, Sr. E. J. Freireich and E. M. Hersh. *N Engl J Med* 288(4):169-173, 1973.

Combined studies of direct membrane immunofluorescence with anti-immunoglobulin serum and of lymphocyte blastogenic responses to autologous leukemia cells were carried out in 34 adult patients with acute leukemia. Twenty-four of 34 (71 per cent) had positive blastogenic responses to their own leukemia cells. Eight of 19 patients with acute myelogenous leukemia and one of five with acute lymphoblastic leukemia had complete or partial abrogation of this positive blastogenesis when the lymphocytes were cultured in autologous rather than allogeneic serum. Direct membrane immunofluorescence with anti-immunoglobulin serum showed bound IgG on the cells of seven of eight patients with acute myelogenous leukemia and serum inhibition, and one without the serum inhibitory effect. Membrane immunoflourescence was negative in two patients with positive blastogenesis and serum inhibition, 14 of 15 with positive blastogenesis and no serum inhibition and, finally, in all 10 patients with negative blastogenic responses to leukemia cells. A good prognosis was correlated with a positive blastogenic response, its inhibition by autologous serum, and IgG bound to the cell surface.

0386 PROOF OF ANTITUMOR CELL-MEDIATED IMMUNITY IN HUMANS BY MEANS OF ALLOGENEIC ANTIGENS.

(Fr.) Febvre, H. (Natl. Inst. Hlth. Med. Res., Paris, France), A. Moutte and J.-P. Constans. *C R Acad Sci* [*D*] *(Paris)* 275(19):2191-2194, 1972.

Allogeneic antigens prepared from human brain tumor-derived cell lines cultivated *in vitro* over long periods of time were utilized to prove the existence of a certain antitumor immunity in tumor patients. Two methods were applied simultaneously, a delayed hypersensitivity reaction (DHSR) *in vivo* and a cytotoxicity reaction *in vitro*. The delayed hypersensitivity reaction performed in 60 patients with antigens from cell lines derived from malignant brain tumors produced positive reactions in patients with gradually developing tumors. Antigens obtained from Rous sarcoma virus-transformed cells *in vitro* caused similar reactions; both types of antigens were refered to as strong antigens. Antigens derived from fast-growing extracerebral tumors produced weak delayed hypersensitivity reactions and were referred to as weak antigens. Antigens derived from normal adult brain or from diploid embryonal cells caused no reaction. The patients with relapsed primary brain tumor (24 cases) exhibited positive cutaneous DHSRs when treated with one of the strong antigens. Patients with extracerebral tumor metastases (12 cases) and those with gradually evolving extracerebral tumors (12 cases with Hodgkin's disease or melanoma) reacted strongly to the strong antigens and elicited sometimes a positive reaction to weak antigens. Patients with no tumors presented no DHSR (5 cases) and patients with benign tumors (meningioma or hypophysary adenoma, 7 cases) elicited irregular (positive or negative) responses. Cytotoxicity reactions *in vitro* performed with lymphocytes from tumor patients on allogeneic target cells produced results concordant with the *in vivo* data. Lymphocytes from patients having no tumor disease or diploid embryonal cells exhibited no cytotoxicity whereas lymphocytes from tumor patients produced considerable destruction of the target cells after 24 hr. Lymphocytes from patients with extracerebral tumors but with metastases to the brain were much more cytotoxic than those from patients with brain tumors. The above results indicate the existence of antitumor immunity in cancer patients; this immunity is not specific for the histological type of the tumor. Further clinical studies are required to evaluate the validity of the above tests in terms of early cancer diagnosis.

0387 ANTIBODIES TO EPSTEIN-BARR VIRUS AND CELLULAR IMMUNITY IN HODGKIN'S DISEASE AND CHRONIC LYMPHATIC LEUKEMIA.

(E.) Hesse, J. (Inst. Med. Microbiol., U. Copenhagen, Denmark), E. Andersen, P. H. Levine, P. Ebbesen, P. Halberg and J. I. Reisher. *Int J Cancer* 11(1):237-243, 1973.

Antibodies to Epstein-Barr virus capsid antigen (VCA), early antigen (EA) and cellular immunity as measured by skin reactivity to keyhole limpet hemocyanin (KLH) and Brucella antigen (BA) were measured in 15 cases of Hodgkin's disease (HD) and 14 cases of chronic lymphatic leukemia (CLL) before treatment and approximately one year later. Both groups of diseases were associated with elevated VCA and EA antibody levels when compared with 18 controls, and in both groups the mean titers were unchanged after therapy. A negative skin test to KLH was associated with a short survival in the HD group and was found more frequently in CLL patients with active disease. In young HD patients, a higher EBV titer was found in pretreatment sera of nonsurvivors compared with those who are still alive. There was no correlation, however, between tests for cell-mediated immunity and humoral antibodies against EBV. The finding of similar titers in patients with depressed and normal skin reactivity indicates that the elevated titers are probably not the result of a non-specifically depressed immune defense.

0388 SPECIFIC "ARMING" OF NORMAL LYMPH-NODE CELLS BY SERA FROM TUMOR-BEARING MICE.

(E.) Pollack, S. (U. Washington Med. Sch., Seattle). *Int J Cancer* 11(1):138-142, 1973.

Lymph-node cells of nonsensitized mice were "armed" (i.e., made specifically cytotoxic) by exposure *in vivo* to passively tranferred sera from tumor-bearing mice. Four hours after an i.p. injection of antitumor serum, the lymph nodes were removed from recipient BALB/c mice and the activity of the lymph-node cells was tested on tumor cells in an *in vitro* microcytotoxicity test. Specific cytotoxicity was demonstrated to the respective target tumors (Moloney virus or 3-methylcholanthrene-induced sarcoma) carried by the tumor-bearing serum donors. The most significant reduction in the number of target cells (six out of eight cases) was obtained with a dose of 0.1 ml of transferred serum. With a 0.5 ml dose, reduction occurred in only three of nine cases. The 0.5 ml serum may have contained enough blocking factor (e.g., antigen-antibody complexes) to inhibit the passively armed lymphoid cells.

389 TRANSITORY CELL ANTIGENS OF RAT LIVER. III. THE REAPPEARANCE OF α-FETOPROTEIN URING CHEMICAL CARCINOGENESIS.

(Fr.) Nechaud, B. e (Inst. Sci. Res. Cancer, Villejuif, France) and J. riel. *Int J Cancer* 11(1):104-115, 1973.

hen rats were fed a basal diet containing 0.01 to .04% 3'-methyl-4-dimethylaminoazobenzene (3'DAB), o serum α-fetoprotein (αFP) was detected by gel munodiffusion techniques during the first month f ingestion. With 0.06% 3'DAB in the same basal iet, α-FP was detected after only two weeks in the era of almost all animals, irrespective of age t the onset of feeding. An even earlier "re- ppearance" of α-FP was observed with the hepato- arcinogen dimethylnitrosamine, given in drinking ater. However, the frequency of α-FP reappearance as dependent on the age of the animals when reatment was begun. Once detected, the α-FP per- isted in the blood for some time and was ndependent of the continued administration of arcinogen. The reappearance was characterized by everal waves of α-FP secretion over a given eriod. Animals whose serum α-FP level became tabilized had a high probability of developing n hepatocellular carcinoma. Nevertheless, in 30% f hepatoma bearing animals, α-FP could not be etected. Administration of the basal diet either lone or supplemented with 0.06% of the noncarcino- enic 4'-methyl-4-dimethylaminoazobenzene also esulted in α-FP reappearance. Here the reappear- nce took place later and at a frequency equal o or lower than 30%. In the light of these esults, the reported specificity of the association etween serum α-FP and primary liver cancer should e reconsidered, at least during the early stages f the neoplastic transformation.

390 LEUKEMIA INDUCTION IN C3H MICE FOLLOWING THEIR INOCULATION WITH NORMAL AKR LYMPHOID ELLS.

(E.) Nemirovsky, T. (Weizmann Inst. Sci., ehovoth, Israel) and N. Trainin. *Int J Cancer* 1(1):172-177, 1973.

ewborn C3H mice were injected i.p. with viable r *in vitro* X-irradiated cell suspensions from ymph node and spleen mixtures obtained from ealthy AKR donors aged 12-18 months. Of 382 njected with normal intact cells, 30% developed lymphocytic leukemia, whereas none of 56 recipi- nts of irradiated cells did so. Genetic analysis howed that the leukemias appearing in C3H mice riginated *de novo* in the recipient animals appa- ently as a result of their inoculation with immuno- ompetent lymphoid cells. The results obtained by sing this model of leukemia induction are discussed n the basis of participation of a graft-versus-host GvH) reaction in this process. C3H recipients injec- ed at birth with small single inocula of viable AKR ymphoid cells from old, healthy donors exhibited mild GvH reaction and immunological impairment o allogeneic skin grafts. The slight impairment f immunologic capacity which accompanied the GvH eaction probably allowed Gross virus particles in he inocula to replicate and reach the necessary oncentration to induce leukemia.

0391 *IN VIVO* QUANTITATIVE ANALYSIS OF IMMUNE REACTIONS TO SOLID MURINE TUMORS IN SYNGENEIC HOSTS.

(Fr.) Donner, M. (Natl. Inst. Hlth. Med. Res., Exp. Cancer Radiobiol Res. Unit, Vandoevre-les-Nancy, France), D. Oth, D. Vailler and C. Burg. *Int J Cancer* 9(1):30-38, 1972.

The dose-response curves (in terms of amounts of inoculated tumor cells and tumor development) and the TD50 (the number of tumor cells required to produce neoplasia in 50% of the animal population) are considered to be biological characteristics of tumors. These parameters vary according to the immunological status of the host. The TD50 method was applied to perform a quantitative determination of immune reactions directed against tumor-associated transplantation antigens (TATA) of three solid murine tumors in C3H/He mice *in vivo*. The intensity of the primary immune reaction was evaluated by the difference between the TD50 values in normal and immune-depressed (450 R X-ray exposure) animals. The secondary immune response was estimated by the difference between the TD50 in normal and specifically immunized recipients. According to these criteria, the results show that the intensity of the primary and secondary immune reactions cannot be compared from one tumor to another. For one tumor, the secondary reaction is quantitatively much greater than the primary reaction. For another tumor, the exact opposite is the case. These results suggest that there is no correlation between the primary and secondary immune responses and that they are quantitatively dependent upon multiple factors. The results are discussed in relation to the determination of antigenic strength of tumors.

0392 IMMUNODEPRESSION AS A FACTOR DURING 3-METHYLCHOLANTHRENE CARCINOGENESIS AND SUBSEQUENT TUMOUR GROWTH IN MICE.

(E.) Rees, J. A. (U. Bristol Med. Sch., England) and M. O. Symes. *Int J Cancer* 11(1):202-211, 1973.

The spleen cell IGM antibody response of CBA/H-T6 mice to sheep red blood cells was significantly depressed 23 days (but no earlier) after injection of 3 or 6 mg 3-methylcholanthrene (3-MC). However, at this time, there was no reduction in the ability of spleen cells from these mice to induce a graft-versus-host (GvH) reaction in (A X T6)F_1 hybrid animals. On day 68 after 3-MC injection, six animals had developed tumors while six were still in the latent period. There was no difference in the capacity of spleen cells from these two groups of animals to induce a GvH reaction in F_1 hybrids. Three 3-MC induced sarcomata were serially passaged in isogenic hosts. The growth rates of these tumors increased and the regional lymph nodes and spleens became progressively more hypoplastic in successive hosts. However, there was again no decline in immunocompetence of spleen cells as judged by the induction of a GvH reaction in F_1 hybrids. It is, therefore, suggested that the increase in tumor growth rate reflected a change in the tumor rather than increasing immunodepression of successive hosts.

0393 *IN VITRO* ASSAY OF CELLULAR IMMUNITY TO TUMOR-SPECIFIC ANTIGEN(S) OF VIRUS-INDUCED TUMORS BY MACROPHAGE MIGRATION INHIBITION. *(E.)* Blasecki, J. W. (Baylor Coll. Med., Houston, Tex) and S. S. Tevethia. *J Immunol* 110(2):590-594, 1973.

A macrophage inhibition technique has been applied as an assay for cellular immunity to soluble SV40 tumor-specific antigen(s) (TSA). Soluble TSA prepared by two different methods from SV40-transformed BALB/c mouse embryo cells (VLM) inhibited the migration of peritoneal exudate (PE) cells from BALB/c mice immunized with SV40 virus or γ-irradiated VLM cells. The inhibition was specific and dependent upon the TSA concentration. PE cells from BALB/c mice immunized with adenovirus 12 or γ-irradiated spontaneously transformed BALB/ET3 cells were not significantly inhibited from migrating. The evidence for tumor specificity of the migration inhibition was further strengthened by similar results from a syngeneic hamster system. Soluble TSA prepared from a hamster line derived from SV40-transformed skin fibroblasts inhibited the migration of PE cells from tumor-free SV40-inoculated hamsters but not from tumor-bearing inoculated hamsters or normal, uninoculated animals.

0394 THE EFFECT OF TUMOR SIZE ON CONCOMITANT TUMOR IMMUNITY. *(E.)* Deckers, P. J. (Boston U. Med. Ctr., Mass.), R. C. Davis, G. A. Parker and J. A. Mannick. *Cancer Res* 33(1):33-39, 1973.

A methylcholanthrene-induced fibrosarcoma (S-10) in inbred C57BL/6 mice was used to study the effect of tumor size on the development and persistence of concomitant tumor immunity in tumor-bearing animals. S-10 tumor-bearing mice rejected a second S-10 tumor isograft inoculated 14 days after the initial S-10 tumor transplantation. An antigenically distinct S-11 tumor isograft grew progressively, however, indicating that concomitant tumor immunity was tumor specific. S-10 tumor-bearing mice were sacrificed 7, 21, 28, 35, or 49 days after each was given an inoculation of 10^5 viable S-10 cells in the right hind limb. The corresponding ratios of mean tumor weight to total body weight (TW:TBW) of the tumor-bearing mice were 0, 6, 14, 18, and 23%, resp. Viable spleen cells from these tumor-bearing animals were injected i.p. into untreated syngeneic mice that were challenged with 10^3 S-10 cells 14 days after spleen cell transfer. Controls received tumor cells alone or tumor cells plus spleen cells from normal syngeneic mice. Recipients of spleen cells from mice with TW:TBW of 6 to 18% demonstrated a significantly decreased incidence of tumor development ($p < 0.01$). Recipients of normal spleen cells or of spleen cells from S-10-bearing mice with TW:TBW greater than 20% demonstrated no decrease in tumor incidence when compared with recipients of tumor cells alone. Thus, concomitant tumor immunity could be adoptively transferred with spleen cells from mice with smaller growing tumors but could not be transferred with spleen cells from mice with very large tumors. In addition, spleen cells from mice with S-10 tumors at TW-TBW of 6 to 18% produced 50% (or greater) cytolysis of tumor cells in the lymphocyte microcytotoxicity assay. However, lymphoid cells from S-10 tumor-bearing mice at TW:TBW of 20% (or greater) produced no significant cytolysis. Furthermore, serum from S-10 tumor-bearing mice never blocked the cytolysis of S-10 tumor cells by lymphoid cells from immunized mice. These data suggest that tumor-specific immunity develops during tumor growth but is lost when the immunizing tumor is very large and that in this murine tumor-host system, humoral blocking factors are an inadequate explanation for the progressive growth of the S-10 tumor despite a concomitant significant cellular immune response.

0395 IMMUNE RESPONSES IN MICE TO TUMOUR CHALLENGE AFTER IMMUNIZATION WITH NEWCASTLE DISEASE VIRUS-INFECTED OR X-IRRADIATED TUMOUR CELLS OR CELL FRACTIONS. *(E.)* Beverly, P. C. L. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), R. M. Lowenthal and D. A. J. Tyrrell. *Int J Cancer* 11(1):212-23, 1973.

Studies were conducted to determine the conditions under which inbred CBA mice are protected from tumor challenge by preimmunization with Newcastle disease virus (NDV)-infected ascites tumor sarcoma 37 (S37) cells. I.p. inoculation of NDV-infected S37 cells, a partially purified virus-containing fraction from these cells, or x-irradiated S37 cells were able to protect mice against subsequent tumor challenge. In addition, mouse hyperimmune serum could also protect CBA mice from S37 tumor cell challenge. Treatment of x-irradiated S37 cells with either antilymphocyte or anti-theta serum abolished this protection, suggesting that cell-mediated immunity played an important role. Although immunization of CBA mice with NDV-infected S37 cells resulted in significant antibody production, the antibody titers did not correlate with survival after challenge. Preimmunization with homologous virus prior to inoculation with an NDV-containing tumor cell fraction did not enhance the immune response against tumor challenge.

0396 DIFFERENTIATED FUNCTIONS EXPRESSED BY CULTURED MOUSE LYMPHOMA CELLS. II. θ ANTIGEN, SURFACE IMMUNOGLOBULIN AND A RECEPTOR FOR ANTIBODY ON CELLS OF A THYMOMA CELL LINE. *(E.)* Harris, A. W. (Hall Inst. Med. Res., Victoria, Australia), A. D. Bankhurst, S. Mason and N. L. Warner. *J Immunol* 110(2):431-438, 1973.

Nineteen different radiation-induced thymomas originating in BALB/c, NZB and NZW mouse strains were tested for Ig synthesis by short-term culture with ^{14}C-amino acids followed by radioimmunoelectrophoresis. Twelve were negative, six produced light (L) chains and one synthesized a protein similar

IgM. The IgM producer, WEHI-22, and one of the
chain producers, WEHI-105, were established as
oned cultured cell lines and shown to possess
e lymphoid cell characteristics of susceptibility
inhibition by low concentrations of thymidine
d of cortisol. Both also possessed the θ antigen,
thymus-derived (T) lymphocyte marker. Cells of
e WEHI-22 line also possessed surface immunoglo-
lin readily detectable by binding of labeled
ti-Ig antibody and were able to bind mouse
tibody-coated sheep erythrocytes forming rosettes,
ile WEHI-105 did neither. The specificity of the
receptor mediating rosette formation appeared from
eloma protein competition experiments to be very
milar to that of the receptor described for non-
ymus-derived (B) cells. It is considered that
HI-22 cells either possess a mixture of T and B
ll characteristics or are neoplastic variants
T cells possessing surface IgM with antiglo-
lin activity.

97 REACTIONS OF MURINE LYMPHOMA CELLS WITH INFECTIOUS MONONUCLEOSIS SERA. *(E.)*

lave, I. (Sch. Med., State U. New York, Buffalo),
Kano and F. Milgram. *J Immunol* 110(2):439-
3, 1973.

actions of infectious mononucleosis sera with
rine lymphoma cells were studied by means of comple-
nt-mediated cytolysis and agglutination tests.
st experiments were performed with L5178Y lympho-
astic cell line of DBA/2 mouse origin, but
milar results were obtained with three other murine
mphomas (EL-4 leukemia of C57BL/6 mice, L#2 leuke-
a of A mice and DBA/2 thyoma). All infectious
nonucleosis sera with heterophile antibodies gave
sitive results with murine lymphoblasts. Cytoly-
c titers were considerably higher than agglutinating
ters. Absorption experiments showed that the
served reactions were produced by heterophile
tibodies combining with an antigen of murine
mphoblasts which is identical with or closely re-
ted to the heterophile antigen of bovine and
eep erythrocytes.

98 FIBROSARCOMA INDUCTION IN IMMUNOSUPPRESSED MICE. *(E.)*

Sanford, B. H. (Massachusetts
n. Hosp., Boston) and S. F. Soo. *J Immunol*
0(2):595, 1973.

nety C3H/St male mice were operated on at 60 ± 7
ys; half of these were thymectomized and the rest
am-thymectomized. Eight days later, whole body
radiation at 350 R was carried out. A significant
munosuppresion was induced by this method, with
0% of 30 thymectomized, irradiated C3H mice succumbing
thin 30 days to an inoculum of allogeneic tumor
A3) which was lethal for only 53 ± 9% of control
H mice. Three weeks later each mouse received a s.c.
plant of plastic film 0.14 mm thick. Mice were
ndomly divided into cages of eight and checked weekly
r 18 months thereafter. More thymectomized than
n-thymectomized mice succumbed during this period
0 ± 6% of thymectomized mice *vs* 11 ± 5% of sham-
thymectomized). The difference was not statistically significant (p = > 0.05). Similarly, fibrosarcoma incidence was slightly, but not significantly higher in the thymectomized group (20 ± 7% *vs* 12 ± 5%; p = > 0.05). It is concluded that the incidence of plastic-film induced fibrosarcomas is not substantially increased in C3H/St mice by adult thymectomy in combination with 350 R whole body irradiation.

0399 SURFACE IMMUNOGLOBULINS OF BOVINE LYMPHOSARCOMA CELLS. *(E.)*

Tsuyuguchi, I. (U. Pennsylvania Sch. Med., Philadelphia), M.-M. Ku and F. Karush. *J Immunol* 110(1):118-127, 1973.

Bovine lymphosarcoma cells were grown in tissue culture and examined for the presence of surface immunoglobulins. The experimental criterion employed was the specific binding of ^{125}I-labeled antibody directed to bovine immunoglobulin determinants. It was noted that lymph node cell suspensions and peripheral blood leucocytes from a lymphosarcomatous animal exhibit specific binding of rabbit antibody directed to such determinants. However, thymocytes from such an animal fail to bind antibody. Normal peripheral blood leucocytes also show specific binding or rabbit anti-$B_{\gamma}G$ and anti-μ chain antibody. It is also apparent that tissue culture cells adsorb exogenous immunoglobulin by exposure to equine serum and bovine serum. Tissue culture cells grown in medium containing 10% rabbit serum, however, exhibit $B_{\gamma}G$-like determinants. They clearly bind anti-$B_{\gamma}G$ antibody but this binding does not seem to involve Fc determinants of the γ chain. Anti-μ chain antibody is not specifically bound and the binding of anti-γ chain antibody is ambiguous. It appears, however, that there are at least L chain determinants on the cell surface.

0400 INTRACELLULAR LOCALIZATION OF RAT LIVER α-FETOPROTEIN BY MEANS OF TRITIATED ESTROGENS. *(Fr.)*

Uriel, J. (Cancer Res. Inst. Villejuif, France), N. Rubio and F. Loisillier. *C R Acad Sci [D] (Paris)* 275(17):1941-1942, 1972.

An autoradiographic procedure using tritiated estrogens for the localization of α-fetoprotein (α-FP)-producing hepatocytes in neonatal rat livers and in chemically-induced primary hepatoma, is presented. Primary hepatoma was induced in Wistar rats by administration of 3'-methyl-4,4'-dimethylaminoazobenzene according to a procedure referred to elsewhere. Liver and hepatoma nodule slice preparations were incubated at room temperature with 6,7-^{3}H-estradiol, 6,7-^{3}H-estrone, or 1,2-^{3}H-testosterone, buffered at a 7.5 pH in physiological solution, for 30 minutes. Each steroid carried a 2μCi/ml specific activity. No cellular label could be ascertained in preparations incubated with testosterone. Incubation with tritiated estrone or estradiol revealed typical intracellular α-FP site images in two-day-old rat livers. The number of labeled cells with respect to the total cell population was very small. The site of the label appeared to be only cytoplasmic and to involve only parenchymal cells. The labeled cells were scattered either individually or in groups of two, three or four cells. The label site in tumor nodule slice preparations resembled the findings in neonatal liver; small foci of labeled granules were found in addition in stromal reaction zones and were attributed to micronecrotic processes. Some labeled cells were detected in the peritumoral liver parenchyma. No intracellular label was detected in adult rat liver preparations upon treatment with the above estrogens. Experiments in progress confirmed the specificity of this technique for α-FP-producing cell labeling due to the high affinity of α-FP to estrogens.

0401 METABOLISM OF HUMAN CARCINOEMBRYONIC ANTIGEN IN XENOGENEIC ANIMALS. *(E.)* Shuster, J. (Montreal Gen. Hosp., Quebec, Canada), M. Silverman and P. Gold. *Cancer Res* 33(1):65-68, 1973.

The metabolism of radiolabeled carcinoembryonic antigen (CEA) was studied in rabbits and dogs. About 70% of injected CEA disappeared from the circulation of these animals in 1 hr. In rabbits and dogs, 50% of the injected radioactivity was sequestered in the liver by the end of 1 hr. Only a trace of radioactivity appeared in the bile, and less than 1% of injected material was detected in kidneys, lung, spleen or heart. There was a negligible accumulation of radioactivity in the thyroid and in the small or large bowel. Based on the metabolic parameters studied, the CEA molecule is biologically heterogeneous, even in preparations that appear physicochemically and immunochemically homogeneous.

0402 CHARACTERIZATION OF ANTISERUM AGAINST MYELOMA CELLS. *(E.)* Cawley, L. P. (Wesley Med. Ctr., Wichita, Kan.), W. L. Goodwin and B. J. Minard. *Cancer Res* 33(1):17-22, 1973.

An antiserum was raised in goats against human malignant plasma cells isolated from a patient with λ-chain myeloma in 1967 and maintained in tissue culture by the Roswell Park Memorial Institute (RPMI 8226). The RPMI cells were harvested, washed, and used without disruption in combination with complete adjuvant for immunization. By electroimmunodiffusion and gel diffusion, the goat anti-myeloma cell serum (AMC) disclosed one and sometimes two immunoprecipitin bands against sonically disrupted RPMI cells. The AMC did not react by electroimmunodiffusion or gel diffusion with human plasma proteins. It also failed to react in gel diffusion and electroimmunodiffusion studies against serum from seven plasma cell cancers (IgG, IgM, IgA, IgE, IgD, and κ and λ chains), serum from three patients with chronic lymphatic leukemia, and four serums showing polyclonal gammopathy. No reaction was found against sonically disrupted preparations from leukocytes removed from normal buffy coat. The AMC in immunofluorescent studies gave strong membrane reaction with RPMI cells but not with lymphocytes, segmented neutrophils, or intracellular components. Immunofluorescence studies with frozen sections of human liver, spleen, and kidney failed to give a reaction, and no reaction was discernible against three normal bone marrow preparations, seven from patients with myeloma (three IgG κ, two IgG λ, two IgA λ), and four from patients with chronic lymphatic leukemia. There was, however, a strong immunofluorescence at the surface of lymphoid plasma cells in bone marrow preparations from three patients with macroglobulinemia of Waldenström (two IgM κ and one IgM λ). It is unknown at this time whether the AMC depicts a tumor-specific antigen on lymphoid-plasma cells of macroglobulinemia or an antigen in the plasma cell system which is unique to lymphoid-plasma cells. The antiserum does not appear to react with the markers of B lymphocytes.

0403 METASTATIC SQUAMOUS CELL CARCINOMA OF THE CERVIX: THE ROLE OF IMMUNOLOGY IN ITS PATHOGENESIS. *(E.)* Davidsohn, I. (Mount Sinai Hosp. Med. Ctr., Chicago, Ill.), H. J. Norris, R. Stejskal and P. Lill. *Arch Pathol* 95(2): 132-134, 1973.

The effects of squamous cell carcinoma of the cervix on antigens A, B, and H, which are present in normal squamous epithelium and erythrocytes, were studied. Cause of death in the 50 patients studied was metastatic carcinoma of cervix. The three antigens were present in normal epithelium, but could not be detected in 92% of 38 primary and 91% of 88 metastatic carcinomas. In less than 10%, antigen loss was partial: cells containing antigens were present next to cells devoid of them. Without exception, loss of antigens preceded formation of distant metastases. Antigen loss was interpreted as evidence of immunologic dedifferentiation analogous to morphologic dedifferentiation of anaplasia. Specific red cell adherence test promises to be useful in early diagnosis and prognosis of squamous cell carcinoma of the cervix.

0404 IMMUNOLOGICAL ABNORMALITIES IN JUVENILE MYELOMONOCYTIC LEUKAEMIA. *(E.)* Cannat, A. (Saint-Louis Hosp., Paris, France) and M. Seligmann. *Br Med J* (5845):71-74, 1973.

Immunological studies were performed on 24 children suffering from juvenile myelomonocytic leukemia. Strikingly high serum immunoglobulin levels with a frequent tendency toward homogeneity and light-chain imbalance were present, together with a high incidence of antinuclear antibodies (52%) and antihuman IgG antibodies (43%). Members of the families of 19 of the children were also studied. A few similar abnormalities were found in eight families, mostly clustered in four of them. The significance of the association of these immunological aberrations with juvenile myelomonocytic leukemia is at present unknown. In view of the interrelation observed in experimental models between immunological abnormalities and oncogenic viruses, the possibility of a common etiological factor such as a viral infection during fetal life is suggested.

0405 LYMPHOCYTE SURFACE IMMUNOGLOBULINS. DISTRIBUTION AND FREQUENCY IN LYMPHOPROLIFERATIVE DISEASES. *(E.)* Piessens, W. F. (Brigham Hosp., Boston, Mass.), P. H. Schur, W. C. Maloney and W. H. Churchill. *N Engl J Med* 288(4):176-180, 1973.

The frequency and distribution of surface immunoglobulins, a B-cell marker, were studied on peripheral blood lymphocytes from normal controls and patients with infectious mononucleosis, chronic lymphatic leukemia, and lymphosarcoma. IgG, IgA, IgM, IgD, and IgE determinants were demonstrated on lymphocytes from normal donors. In infectious mononucleosis there was a transient increase of surface IgM early in the disease. When these patients were retested several months later, the percentage of lymphocytes with surface IgM had reverted to the normal range. Chronic lymphatic leukemia and lymphosarcoma could be divided into two groups: one with no detectable surface Ig on their lymphocytes; and another with increased numbers of lymphocytes with surface Ig. There was no correlation between surface and serum immunoglobulins. The absence of surface Ig in lymphosarcoma correlated with nonprogressive disease restricted to lymph nodes. These findings suggest that some forms of chronic lymphatic leukemia and lymphosarcoma may be B-cell diseases whereas other forms could be T-cell diseases.

0406 LYMPHOCYTE REACTIVITY IN CANCER PATIENTS: CORRELATION WITH TUMOR HISTOLOGY AND CLINICAL STAGE. *(E.)* Catalona, W. J. (Natl. Cancer Inst., Bethesda, Md.), W. F. Sample and P. B. Chretien. *Cancer* 31(1):65-71, 1973.

The *in vitro* reactivity of peripheral blood lymphocytes and the effect of serum on normal lymphocyte reactivity were determined in 89 patients with nonlymphoid tumors by quantitation of phytohemagglutinin (PHA)-induced tritiated thymidine incorporation. The reactivity of lymphocytes from patients with sarcomas and squamous carcinomas was impaired, and their sera suppressed normal lymphocyte reactivity. The cellular and serum abnormalities coexisted in patients with sarcomas but occurred independently in patients with squamous carcinomas. Patients with melanomas and adenocarcinomas did not differ from controls by these parameters. With the exception of patients with melanomas, a similar incidence of abnormalities occurred in patients with localized tumors and those with metastases. These data indicate that this test system assays certain early and tumor histology-related aspects of the host-tumor relationship and is potentially useful for the study of factors associated with development of cancer and monitoring effect of tumor treatment.

0407 LACK OF DISTINCTIVE SURFACE ANTIGEN ON CELLS TRANSFORMED BY MURINE SARCOMA VIRUS. *(E.)* Strouk, V. (Karolinska Inst., Stockholm, Sweden), G. Grundner, E. M. Fenyö, E. Lamon, H. Skurzak and G. Klein. *J Exp Med* 136(2):344-352, 1972.

Murine sarcoma virus (MSV)-specific surface antigens were looked for in tests with various cells including: S+L-, an MSV-transformed line of 3T3 mouse cells which possessed the MSV genome but did not produce infectious helper murine leukemia virus, D56, a Moloney leukemia virus (MLV)-transformed mouse line, and Ha2, derived from an MSV-induced CBA mouse tumor. In one group of tests, isoantibody-free sera, obtained by immunization with MLV or by allowing primary autologous MSV-induced sarcomas to regress, were reacted against MSV-induced sarcoma cells. In another group of tests alloantisera, obtained by immunizing C57BL mice with D56 cells, were absorbed with untransformed 3T3 cells to remove anti-3T3 activity and reacted against D56 cells. Neither S+L- nor D56 reacted with anti-MSV or anti-MLV sera. Both sera reacted well with MLV-superinfected D56 cells and with MSV(MLV)-producing Ha2 cells. Normal 3T3, S+L- and D56 cells all reacted with C57BL anti-D56 sera. Results suggested that S+L- cells, though they contained the MSV genome, were not different from the original 3T3 cells in their antigenic characteristics.

0408 SERUM IMMUNOGLOBULIN LEVELS IN CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKAEMIA AND THEIR MOTHERS AND SIBS. *(E.)* Chandra, R. K. (All India Inst. Med. Sci., New Delhi). *Arch Dis Child* 47(254):618-620, 1972.

Twenty-nine children aged two to ten yrs, who had been diagnosed as having acute lymphoblastic leukemia, were tested for serum IgG, IgA and IgM levels. Also tested were the patients' mothers, six neonate siblings, and 42 siblings aged two to ten yrs. All six neonate siblings had high IgM levels in cord blood. A significant increase in serum IgG and IgM was seen in patients' mothers, and a significant decrease in serum IgG was seen in siblings aged two to ten yrs. Patients themselves had lower serum IgG concentrations than healthy children. It was concluded that an antigenic stimulus, possibly viral, was present during the intrauterine life of leukemia-affected sibships.

0409 EVIDENCE FOR VIRUS-RELATED AND UNRELATED ANTIGENS ON MURINE LYMPHOMAS INDUCED BY CHEMICAL CARCINOGENS. *(E.)* Colnaghi, M. I. (Natl. Inst. Study Cure Tumors, Milan, Italy) and G. Della Porta. *J Natl Cancer Inst* 50(1):173-180, 1973.

Ether-treated extracts of seven C57BL lymphosarcomas induced by three different chemical carcinogens, urethan (Ur), N-nitrosomethylurea (NMU) or 7,12-dimethylbenz[*a*]anthracene (DMBA), were tested by immunodiffusion for the presence of group-specific antigens and all were found positive. Direct cytotoxic tests and absorption tests with a murine anti-G serum and a rat antiradiation leukemia virus (RadLV) serum, using nine other Ur, NMU, or DMBA-induced C57BL lymphomas, demonstrated the presence of common cell-surface, virus-related antigens on the virus-induced E♂G2 leukemia, the RadLV lymphoma, and the chemically induced lymphomas. An antiserum raised in C57BL against Ur-induced C3Hf or BALB/c lymphomas exhibited a complement-dependent cytotoxic activity against 21 of 22 C57BL lymphomas induced by the three different carcinogens, against the EL4 leukemia but not against the virus-induced E♂G2 leukemia. The E♂G2 leukemia also failed to absorb completely the specific cytotoxicity of the anti-chemically-induced lymphomas antiserum. C57BL multiparous or antiembryo sera exerted a complement-dependent cytotoxicity on the chemically induced lymphomas but not on the E♂G2 leukemia. Thus carcinogen-induced lymphosarcomas, in addition to Gross virus-related antigens, possess tumor-specific, cell-surface, cross-reacting antigens not related to the virus, which may represent modified expressions of cellular genetic information and/or a retrogenetic expression.

0410 POLYOMA T ANTIGEN SYNTHESIS BY TEMPERATURE-SENSITIVE MUTANTS OF POLYOMA VIRUS.

(E.) Oxman, M. N. (Children's Hosp. Med. Ctr. Boston, Mass.), K. K. Takemoto and W. Eckhart. *Virology* 49(3):675-682, 1972.

The production of T antigen detectable by an indirect fluorescent antibody technique was studied in secondary mouse embryo cell cultures infected by one "late" and four "early" temperature sensitive mutants of polyoma virus. Plaque assays using infectious viral DNA showed that all four early mutants are defective in viral DNA synthesis at 39 C, whereas the late mutant and wild type viruses are not. All four early mutants were also unable to synthesize T antigen at this higher temperature. However, the fact that inhibition of viral DNA synthesis by cytosine arabinoside or 5-fluorodeoxyuridine did not prevent T antigen synthesis by wild type virus or by the early mutants at the permissive temperature suggested that the failure of the early mutants to produce T antigen at 39 C was not due to defective DNA replication *per se*. Rather, the inability to produce T antigen at the non-permissive temperature may reflect an alteration in either the gene which codes for T antigen, or in a gene whose expression is necessary for both T antigen production and for viral DNA replication.

0411 REAGINIC ANTIBODY TO TUMOUR AND ALLOANTIGENS IN MICE.

(E.) Bartholomaeus, W. N. (Dept. Microbiol., U. Western Australia, Perth) and D. Keast. *Nature New Biol* 239(94): 206-207, 1972.

A reaginic antibody response to B16 tumor, a transplantable melanoma originating in C57 Black mice, was demonstrated in C57Bl mice. The response was greatest if $Al(OH)_3$ adjuvant was used but also occurred in response to tumor cells alone. Balb/c mice, which were used for passive cutaneous anaphylaxis reactions (PCA), produced reaginic antibody to B16 tumor alloantigens; this effect was increased by use of $Al(OH)_3$ adjuvant with the tumor cells. PCA reactions were also carried out with serum collected from C57Bl mice immunized with Prince Henry (PH) spleen cells and PH mice immunized with C57Bl spleen cells. Both strains produced reaginic antibody to alloantigens found on the spleen cells of the other strain. Again, an absolute requirement for $Al(OH)_3$ was not found. The observed production of reaginic antibody suggests that mast cells or basophils sensitized with reaginic antibody to tumor antigens could play a part in host immune responses to tumor cells. Similarly, production of reaginic antibody to antigens present on allografts may influence survival of such grafts.

0412 ANALYSIS OF IMMUNOGLOBULINS IN CHICKEN ANTIBODY TO AVIAN LEUCOSIS VIRUSES.

(E.) Meyers, P. (State U. New York, Upstate Med. Ctr., Syracuse) and R. M. Dougherty. *Immunology* 23(1):1-6, 1972.

Chicken sera containing antibody to the avian leucosis viruses RAV-1 or RAV-6 were fractionated on Sephadex G-200 columns. Sera from seven pathogen-free White Leghorns were taken 8 wk after i.v. infection with a single 1.0 ml dose of RAV-1; sera from eight other birds, including three congenitally infected with ALV-F42, were taken 16-20 wk after a single i.v. dose of 1.0 ml RAV-6 or two i.p. 1.0 ml doses. A small proportion of antibody activity was found in the serum IgM fractions from the majority of birds, but most of the antibody activity was recovered in the serum IgG fractions. The distribution of neutralizing antibody between IgG and IgM did not vary with age, nor between pathogen-free and congenitally infected birds.

0413 NATURAL HUMAN ANTIBODIES TO "HIDDEN" MEMBRANE COMPONENTS.

(E.) Rosenberg, S. A. (Natl. Cancer Inst., Bethesda, Md.) and G. N. Rogentine, Jr. *Nature New Biol* 239(94): 203-204, 1972.

The cytotoxicity of 148 normal human sera to human lymphocytes was studied. With the exception of anti-HL-A alloantisera, none of the sera was toxic to untreated lymphocytes. Toxicity of

era to neuraminidase (NASE)-treated lymphocytes,
owever, was positive for 36 of 42 normal
dult sera, all 16 sera from children less than 10
r old, 8 of 9 sera from patients with metastatic
ancer, 9 of 11 leukemic sera, and 54 of 60
lloantisera used for histocompatibility testing.
ll of six tested normal sera were toxic to auto-
ogous NASE-treated lymphocytes but not to un-
reated lymphocytes. Chromatography of normal
erum on a Sephadex G-200 gel filtration column
ndicated that the lysis of NASE-treated lympho-
ytes is mediated by a circulating antibody
hich travels with the IgM portion of serum
mmunoglobulins on the gel filtration column.

14 IMMUNOHISTOLOGICAL STUDIES ON MUCOSAL
GLYCOPROTEIN IN RECTAL CARCINOMA. *(E.)*
wasaki, H. (Kurume U. Sch. Med., Japan), K.
asato and E. Kimoto. *Gann* 63(6):743-746, 1972.

uorescein isothiocyanate-labeled antibodies
ainst the gastric mucosal protein (GMP) and the
testinal mucosal protein (IMP) were reacted with
ctal carcinoma material from 20 patients. Some
ctal carcinomas were anaplastic and lacked a
andular arrangement of cells; these did not
crete mucus. Others secreted mucus and/or showed
llular differentiation and glandular arrangement.
direct immunofluorescence tests, anaplastic
lls fluoresced intensely with anti-GMP and with
ti-IMP. Immunofluoresced and mucus-stainable
tes agreed well with one another in the
ctal carcinomas. Cells of more well-differentia-
d carcinomas were devoid of fluorescence. As
e glandular structure of the rectal carcinomas
came less orderly in arrangement, the cells
came more intensely fluorescent over a more
tensive area.

15 SUBCELLULAR LOCALIZATION OF AVIAN LEUCO-
SIS GROUP-SPECIFIC COMPLEMENT-FIXING
TIGENS IN MOUSE ASCITES SARCOMA CELLS (SR-C3H/
CELLS) INDUCED BY SCHMIDT-RUPPIN STRAIN OF ROUS
RCOMA VIRUS. *(E.)* Seki, S. (Okayama U. Med.
., Japan) and T. Oda. *Gann* 63(6):657-663, 1972.

uorescent antibody staining and complement fixa-
on were used to determine the location in the cell
the group-specific (gs) antigens for avian leuco-
-sarcoma complex viruses in a line of cells
an ascites sarcoma maintained *in vitro*. The
rcoma (SR-C3H/He) was originally induced in a
/He mouse by Schmidt-Ruppin Rous sarcoma virus.
rcoma cells were subjected to subcellular fractiona-
on before testing in complement fixation studies.
tumor cells continued to produce gs antigens
long as six years after the establishment of
sarcoma cell line. Antigen was confined to
oplasm and was found in soluble supernatant
microsomal fractions. Nuclei, mitochondria
plasma membranes all lacked gs antigen.

0416 SERUM MEDIATED INHIBITION OF THE IMMUNOLOGICAL REACTIONS OF THE PATIENT TO HIS OWN TUMOUR: A POSSIBLE ROLE FOR CIRCULATING ANTIGEN.

(E.) Currie, G. A. (Chester Beatty Res. Inst., Sutton, England) and C. Basham. *Br J Cancer* 26(6):427-438, 1972.

In a microcytotoxicity assay the lymphocytes from cancer patients were tested on autologous and allogeneic tumor cells *in vitro*. In patients with a variety of tumors, extensive washing of the lymphocytes from those cases with advanced disease was found to greatly enhance their specific cytotoxic effects. This specificity was restricted to autologous tumor cells and allogeneic cells of similar histological origin. This cross-reacting cytotoxicity was not, however, universal, especially in cases of malignant melanoma. The cytotoxicity evoked by washing was abolished by the addition of the patient's serum. This serum effect showed a similar specificity to that found for lymphocyte cytolysis. The effect of washing, and the specific inhibitory effect of serum, was not detectable in early cases of primary malignant melanoma. The serum component responsible for inhibiting lymphocyte cytotoxicity had no detectable affinity for the target cells and appears to act on the lymphocyte surface, implying that tumor antigen may well be implicated.

0417 GENETIC CONTROL OF THE IMMUNE RESPONSE IN MICE. IV. RELATIONSHIP BETWEEN GRAFT *VS* HOST REACTIVITY AND POSSESSION OF THE HIGH TUMOR GENOTYPES $A^{y}a$ AND $A^{vy}a$.

(E.) Gasser, D. L. (U. Pennsylvania, Sch. Med., Philadelphia) and T. Fischgrund. *J Immunol* 110(1):305-308, 1973.

The graft *vs* host (GVH) reactivity of spleen cells from mice possessing the high tumor genotypes $A^{y}a$ and $A^{vy}a$ was compared with the reactivity of spleen cells from histocompatible nonagouti animals. Spleen cells from YBR aa mice caused significantly greater GVH reactions than YBR $A^{y}a$ cells in 14 of 18 litters of (YBR x C57BL/10) F_1 mice. Spleen cells from C57Bl/6J aa donors were more reactive than cells from C57BL/6J $A^{vy}a$ mice in GVH assays in eight of nine litters of (C57BL/6J X B10.BR) F_1 mice. These results suggest that reduced immune responsiveness may be involved in the tumor susceptibility of $A^{y}a$ and $A^{vy}a$ mice.

0418 TOTAL SERUM GAMMAGLOBULIN AND IMMUNOGLOBULIN G, A, AND M ALTERATIONS OCCURRING IN CHILDREN WITH LEUKEMIA. I. CHANGES IN TOTAL GAMMAGLOBULIN CONTENT AND QUALITATIVE IMMUNOGLOBULIN ALTERATIONS OCCURRING IN THE SERUM DURING LEUKEMIA IN CHILDREN.

(Ger.) Blau, H.-J. (Children's Clin. U. Rostock, E. Germany). *Folia Haemat (Leipzig)* 97(3):139-145, 1972.

Quantitative gammaglobulin determinations were performed in 40 children with parablastic leukemia above 2 years-of age at the onset of the disease, in 8 children at the beginning of remission, in 9 children during stable remission, and in 16 children upon relapse. The absolute amount of gammaglobulin was computed from the paper electrophoretic fraction reading and the related total serum protein determined by refractometry. Data were compared to findings of 22 healthy age-matched children. Serum gammaglobulin averaged 895 mg/100 ml in normal children and 1267 mg/100 ml during the initial stage of the disease. Children in first remission (immune suppression therapy) had 826 mg/100 ml gammaglobulin. Average serum gammaglobulin amounted to 915 mg/100 ml in stable remission and dropped to 647 mg/ml under relapse conditions. Qualitative immunoglobulin studies through immuno electrophoresis of leukemic sera treated with polyspecific rabbit antihuman serum revealed no pathological immunoglobulins.

0419 COMBINED STUDIES OF COMPLEMENT RECEPTOR AND SURFACE IMMUNOGLOBULIN-BEARING CELLS AND SHEEP ERYTHROCYTE ROSETTE-FORMING CELLS IN NORMAL AND LEUKEMIC HUMAN LYMPHOCYTES. *(E.)* Ross, G. D. (Natl. Jewish Hosp. Res. Ctr., Denver, Colo.), E. M. Rabellino, M. J. Polley and H. M. Grey. *J Clin Invest* 52(2):377-385, 1973.

Human lymphocytes from normal peripheral blood, thymus, spleen, thoracic duct, and peripheral lymphocytes from patients with chronic lymphatic leukemia were studied for complement receptor sites (CRL), surface immunoglobin (SIg), and for the ability to form rosettes with sheep erythrocytes (TRFC) *in vitro*. In normal lymphocytes, CRL and SIG, as determined by immunofluorescence assay, were found to be overlapping, but not identical populations which were characteristic of B cells. Cells that were able to form rosettes, however constituted a totally unrelated population, and TRFC was thus considered to be a reliable marker for T cells. In normal peripheral blood, 24% of the lymphocytes had SIg and half of these also had CRL. Almost all of the non-SIg cells were TRFC. Practically all spleen and thoracic duct lymphocytes were $SIg^{+}CRL^{+}$. In leukemic individuals, three observations were made: (1) The presence or absence of CRL was independent of the presence or absence of SIg so that in individuals whose cells did not have SIg, CRL were usually abundant. (2) Leukemic cells were essentially negative for TRFC. (3) Leukemic cells reacted poorly with human C3 as compared to mouse C3. These data suggest that human chronic lymphatic leukemic cells contain altered SIg receptors and CRF. The lack of TRFC also suggests that the leukemic lymphocytes are of B cell origin.

0420 RADIOIMMUNOASSAY FOR HUMAN ALPHA$_1$-FETOPROTEIN. *(E.)* Silver, H. K. B. (Montreal Gen. Hosp., Quebec, Canada), P. Gold, S. Feder, S. O. Freedman and J. Shuster. *Proc Natl Acad Sci USA* 70(2):526-530, 1973.

Monospecific anti-alpha$_1$-fetoprotein (AFP) antiserum was obtained from human fetuses and used for radioimmunoassay for AFP with AFP from fetal serum. AFP was labeled with ^{125}I and labeled AFP was assayed by the technique of coprecipitation-inhibition. With this assay, it is possible to detect serum AFP concentrations of as little as 20 ng/ml serum; the assay is therefore more sensitive than the Ouchterlony assay. In tests of the new assay on sera from various subjects, normals never had more than 20 ng AFP/ml serum while hepatoma patients and testicular embryonal cell carcinoma patients had elevated serum AFP levels.

0421 CROSSREACTIVE ANTIGENS ON HUMAN CELLS INFECTED WITH RAUSCHER LEUKEMIA VIRUS AND ON HUMAN ACUTE LEUKEMIA CELLS. *(E.)* Mann, D. L. (Natl. Cancer Inst., Bethesda, Md.), R. Halterman and B. G. Leventhal. *Proc Natl Acad Sci USA* 70(2): 495-497, 1973.

Rabbits were inoculated with preparations of water-soluble components of membranes of cultured lymphoid cells from a Burkitt lymphoma patient. Antisera from these rabbits were tested in direct cytotoxicity or cytotoxicity inhibition tests against various monolayer adherent cells, lymphoid cells in long-term culture or peripheral leukocytes from acute leukemia patients. The antisera were able to detect certain antigens on acute leukemia cells. No reactions were seen between antisera and nine of ten nonlymphoid lines; however, sera were cytotoxic for a line of human embryonic kidney cells infected with Rauscher leukemia virus (RLV). Studies employing absorption of antisera indicated that there were cross-reactive antigens on the surfaces of human leukemia cells and on RLV-infected human embryonic kidney cells.

0422 MACROPHAGES IN SYNGENEIC ANIMAL TUMOURS. *(E.)* Evans, R. (Chester Beatty Res. Inst., Sutton, England). *Transplantation* 14(1): 468-473, 1972.

0423 DIFFERENCE IN HEAT STABILITY OF ANTIGENS ASSOCIATED WITH EPSTEIN-BARR VIRUS, DEMONSTRATED BY IMMUNOFLUORESCENCE. *(E.)* Demissie, A. (Central Microbiol. Lab. Stockholm County, Sweden) and A. Svedmyr. *J Natl Cancer Inst* 50(1):63-67, 1973.

0424 MITOTIC INHIBITION OF EPIDERMAL CELLS FROM PSORIASIS LESIONS *IN VITRO* BY EXTRACTS FROM NORMAL HUMAN SKIN. *(E.)* Chopra, D. P. (Temple U. Hlth. Sciences Ctr., Philadelphia, Pa.) and B. A. Flaxman. *J Natl Cancer Inst* 50(1):281-283, 1973.

425 THE ROLE OF THE PHAGOCYTE IN HOST-PARASITE
INTERACTIONS. XXXIV. THE EFFECT OF
IAGOCYTIZABLE PARTICLES AND PHYTOHEMMAGGLUTININ-P
I DNA SYNTHESIS BY SPLEEN CELLS FROM LEUKEMIC
ID NON-LEUKEMIC AKR MICE. *(E.)* Strauss, R. R.
t. Margaret's Hosp., Boston, Mass.) A. A.
cobs, B. B. Paul and A. J. Sbarra. *J*
ticuloendothel Soc 11(3):277-292, 1972.

26 CHANGES IN SERUM PROTEINS ASSOCIATED
WITH THE IMMUNOSUPPRESSIVE EFFECTS OF
UTE LEUCOSIS (MAREK'S DISEASE). *(E.)* Evans,
L. (Dept. Animal Sci., U. Arkansas, Fayetteville)
d L. T. Patterson. *J Reticuloendothel Soc*
(4):325-342, 1972.

27 IMMUNIZATION OF GUINEA PIGS AGAINST
LEUKEMIA BY SCARIFICATION OF SKIN WITH
UKEMIC CELL EXTRACTS. *(E.)* Gross, L. (VA
sp., Bronx, N.Y.). *Acta Haematol (Basel)*
(6):321-325, 1972.

28 CARCINOEMBRYONIC ANTIGEN: CURRENT
CLINICAL APPLICATIONS. *(E.)* Freedman,
O. (Montreal Gen. Hosp., Quebec, Canada).
llergy Clin Immunol 50(6):348-357, 1972.

29 INCIDENCE OF MONOCLONAL PROTEINS IN A
MINNESOTA COMMUNITY WITH A CLUSTER OF
TIPLE MYELOMA. *(E.)* Kyle, R. A. (Mayo
n. Fdn., Rochester, Minn.) S. Finkelstein, L. R.
eback and L. T. Kurland. *Blood* 40(5):719-724,
2.

0 FAILURE TO DEMONSTRATE IMMUNITY TO LEUKEMIA ASSOCIATED ANTIGENS BY LYMPHOCYTE
NSFORMATION *IN VITRO*. *(E.)* Schweitzer, M.
ntral Lab. Netherlands Red Cross Blood
nsfusion Serv., Amsterdam), C. J. M. Melief and
P. Eijsvoogel. *Int J Cancer* 11(1):11-18, 1973.

1 GROWTH AND ANTIGENIC PROPERTIES OF A
BIOPSY-DERIVED BURKITT'S LYMPHOMA IN
MUS-LESS *(NUDE)* MICE. *(E.)* Povlsen, C. O.
th. Anat. Inst., Copenhagen, Denmark), P. J.
lkow, E. Klein, G. Klein, J. Rygaard and F.
ner. *Int J Cancer* 11(1):30-39, 1973.

2 ESTABLISHMENT OF MULTIPLE LYMPHOBLASTOID
LINES FROM SINGLE LYMPH NODES AND
DY OF THEIR IMMUNOGLOBULIN SYNTHESIS. *(E.)*
het, J. M. (Wallenberg Lab., U. Uppsala,
den), K. Nilsson and J. Pontén. *Int J Cancer*
1):58-63, 1973.

3 DESTRUCTION OF CULTIVATED MELANOMA CELLS
BY LYMPHOCYTES FROM HEALTHY BLACK
TH AMERICAN NEGRO) DONORS. *(E.)* Hellström, I. (U. Washington Med. Sch., Seattle), K. E. Hellström, H. O. Sjögren and G. A. Warner. *Int J Cancer* 11(1):116-122, 1973.

0434 EFFECT OF LOCAL RADIOTHERAPY ON THE ANTIBODY LEVELS AGAINST EBV-INDUCED EARLY AND CAPSID ANTIGENS (EA AND VCA) IN PATIENTS WITH CERTAIN MALIGNANT TUMOURS. *(E.)* Einhorn, N. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), G. Henle, W. Henle, G. Klein and P. Clifford. *Int J Cancer* 9:182-192, 1972.

0435 SERUM β_2-MICROGLOBULIN IN VARIOUS DISORDERS. *(E.)* Evrin, P.-E. (Dept. Renal Med., U. Hosp., Uppsala, Sweden) and L. Wibell. *Clin Chim Acta* 43(2):183-186, 1973.

0436 ASSEMBLY OF IMMUNOGLOBULIN G: THE ROLE OF THE LIGHT-CHAIN POOL. *(E.)* Stott, D. I. (Dept. Biochem., U. Cambridge, England). *Biochem J* 130(4):1151-1152, 1972.

0437 LYMPHOCYTE ACTINOMYCIN BINDING CAPACITY IN CHRONIC LYMPHOCYTIC LEUKAEMIA. *(E.)* Masera, P. (Med. Clin. U. Torino, Italy), A. Pileri, J. Brachet and N. Hulin. *Experientia* 28(12):1484, 1972.

0438 CONTROL OF PYRIMIDINE BIOSYNTHESIS IN HUMAN LYMPHOCYTES. SIMULTANEOUS INCREASE IN ACTIVITIES OF GLUTAMINE-UTILIZING CARBAMYL PHOSPHATE SYNTHETASE AND ASPARTATE TRANSCARBAMYLASE IN PHYTOHEMAGGLUTININ-STIMULATED HUMAN PERIPHERAL LYMPHOCYTES AND THEIR ENZYME COPURIFICATION. *(E.)* Ito, K. (Res. Inst. Nuclear Med. Biol., Hiroshima U., Japan) and H. Uchino. *J Biol Chem* 248(2):389-392, 1973.

0439 ISOLATION AND CHARACTERIZATION OF β_2-MICROGLOBULIN FROM URINE OF KIDNEY TRANSPLANT AND MULTIPLE MYELOMA PATIENTS. *(E.)* Cejka, J. (Children's Hosp. Michigan, Detroit) and K. Kithier. *Arch Biochem Biophys* 153(2):854-857, 1972.

0440 TUMOR-ASSOCIATED ANTIGENS. A NEW PERSPECTIVE OF HODGKIN'S DISEASE. *(E.)* Order, S. E. (No affiliation) and S. Hellman. *JAMA* 223(2):174-175, 1973.

0441 ISOLATION AND CHARACTERIZATION OF AGGLUTININ RECEPTOR SITES. III. STUDIES ON THE INTERACTION WITH OTHER LECTINS. *(E.)* Janson, V. K. (Dept. Biochem. Sci., Princeton U., N.J.), C. K. Sakamoto and M. M. Burger. *Biochim Biophys Acta* 291(1):136-143, 1973.

0442 ASSOCIATION BETWEEN PREVIOUS TUBERCULOUS INFECTION AND CEREBRAL GLIOMA. *(E.)*

Ward, D. W. (Royal Southern Hosp., Liverpool, England), M. L. Mattison and R. Finn. *Br Med J* (5845):83-84, 1973.

0443 BLOOD CARCINOEMBRYONIC ANTIGEN, REGAN ISOENZYME, AND HUMAN CHORIONIC GONADOTROPHIN IN A MAN WITH PRIMARY MEDIASTINAL CHORIOCARCINOMA. *(E.)* Belliveau, R. E. (U.S. Public Hlth. Serv. Hosp., Baltimore, Md.), P. H. Wiernik and E. A. Sickles. *Lancet* (7793):22-24, 1973.

0444 STUDIES CONCERNING THE REGIONAL LYMPH NODE IN CANCER. V. HISTOLOGIC AND ULTRASTRUCTURAL FINDINGS IN REGIONAL AND NON-REGIONAL NODES. *(E.)* Fisher, E. R. (Dept. Path., U. Pittsburgh, Pa.), H. E. Reidbord and B. Fisher. *Lab Invest* 28(1):126-133, 1973.

0445 IMMUNOLOGIC STIMULI IN RELATION TO LEUKEMOGENESIS. II. EFFECT OF PHYTOHEMAGGLUTININ ON RAUSCHER VIRAL LEUKEMOGENESIS. *(E.)* Siegel, B. V. (U. Oregon Med. Sch., Portland) and J. I. Morton. *J Natl Cancer Inst* 50(2):539-542, 1973.

0446 STUDIES ON THE IMMUNOGENICITY OF VIRUS-INFECTED LYMPHOCYTES FOR THE PRODUCTION OF ANTILYMPHOCYTE SERUM. *(E.)* Rossier, E. (Dept. Microbiol., U. Sherbrooke, Quebec, Canada). *Can J Microbiol* 19(1):67-72, 1972.

0447 PREVENTION OF SIMIAN VIRUS 40 TUMORS BY HAMSTER FETAL TISSUE: INFLUENCE OF PARITY STATUS OF DONOR FEMALES ON IMMUNOGENICITY OF FETAL TISSUE AND ON IMMUNE CELL CYTOTOXICITY. *(E.)* Girardi, A. J. (Wistar Inst. Anatomy Biol., Philadelphia, Pa.), P. Reppucci, P. Dierlam, W. Rutala and J. H. Coggin, Jr. *Proc Natl Acad Sci USA* 70(1):183-186, 1973.

0448 MONOCLONAL IgA AND IgM IN THE SERUM OF A SINGLE PATIENT (SC). III. IMMUNOFLUORESCENT IDENTIFICATION OF CELLS PRODUCING IgA AND IgM. *(E.)* Silverman, A. Y. (Roswell Park Mem. Inst., Buffalo, N.Y.), Y. Yagi, D. Pressman, R. R. Ellison and D. C. Tormey. *J Immunol* 110(2): 350-353, 1973.

0449 MONOCLONAL IgA AND IgM IN THE SERUM OF A SINGLE PATIENT (SC). II. IDENTITY OF LIGHT CHAINS FROM IgA(κ) AND IgM(κ). *(E.)* Seon, B.-K. (Roswell Park Mem. Inst., Buffalo, N.Y.), Y. Yagi and D. Pressman. *J Immunol* 110(2): 345-349, 1973.

0450 A HUMAN IgG MYELOMA PROTEIN WITH ANTI-α2 MACROGLOBULIN ANTIBODY ACTIVITY. *(E.)* Seligmann, M. (St. Louis Hosp., Paris, France), C. Sassy and A. Chevalier. *J Immunol* 110(1):85-90, 1973.

0451 TUMOR-FETAL ANTIGENS IN HODGKIN'S DISEASE: AN IMMUNOELECTROPHORETIC ANALYSIS. *(E.)* Chism, S. E. (Boston, Mass.), S. E. Order and S. Hellman. *Am J Roentgenol Radium Ther Nucl Med* 117(1):5-11, 1973.

0452 ROLE OF CONCANAVALIN-A IN TUMOR CELL AGGLUTINATION AND ADHESION TO MACROPHAGES. *(E.)* Inoue, M. (Okayama U. Med. Sch., Japan), M. Mori, K. Utsumi and S. Seno. *Gann* 63(6):795-799, 1972.

0453 LOWERED TRANSPLANTABILITY OF CULTURED TUMOR CELLS BY PERSISTENT INFECTION WITH PARAMYXOVIRUS (HVJ). *(E.)* Yamada, T. (Cancer Res. Inst., Kanazawa U., Japan) and M. Hatano. *Gann* 63(6):647-655, 1973.

0454 INDUCTION OF FELINE ONCORNAVIRUS-ASSOCIATED CELL MEMBRANE ANTIGEN (FOCMA) IN HUMAN CELLS. *(E.)* Essex, M. (Karolinska Inst., Stockholm, Sweden), G. Klein, F. Deinhardt, L. Wolfe, G. Theilen and L. Pearson. *Proc Am Assoc Cancer Res* 13(March):57, 1972.

0455 THE ROSETTE FORMING CELL, A MEASURE OF CELLULAR IMMUNE DEFENSE AGAINST CANCER. *(E.)* Wybran, J. (U. California, San Francisco) and H. H. Fudenberg. *Proc Am Assoc Cancer Res* 13(March):57, 1972.

0456 ANTIGENIC DIFFERENCES BETWEEN NORMAL CELLS AND TUMORS OF HUMAN LYMPHOID ORIGIN SERIALLY TRANSPLANTABLE IN THE NEONATAL SYRIAN HAMSTER. *(E.)* Adams, R. A. (Children's Cancer Res. Fdn., Boston, Mass.), D. J. Lebwohl and S. J. Galli. *Proc Am Assoc Cancer Res* 13(March):57, 1972.

0457 GRAFT *VERSUS* LEUKEMIA. *(E.)* Bortin, M. M. (Med. Coll. Wisconsin, Milwaukee), A. A. Rimm, E. C. Saltzstein and G. E. Rodey. *Proc Am Assoc Cancer Res* 13(March):56, 1972.

0458 *IN VITRO* DEMONSTRATION OF COMMON TUMOR ANTIGENS IN MOUSE AND RAT BLADDER CARCINOMAS. *(E.)* Taranger, L. A. (U. Washington Sch. Med., Seattle), I. Hellström, W. H. Chapman and K. E. Hellström. *Proc Am Assoc Cancer Res* 13(March):56, 1972.

0459 ANTIGENIC ALTERATION OF LYMPHOBLASTIC LEUKEMIC CELLS INDUCED BY CELL CULTURE AND HYDROCORTISONE. *(E.)* Kollmorgen, G. M. (Coll. Med., U. Okalahoma, Okalahoma City), W. A. Cain

d R. Carubelli. *Proc Am Assoc Cancer Res*
(March):52, 1972.

50 TUMOR-SPECIFIC IMMUNITY AND POLY I · POLY
C (PIC). *(E.)* Kreider, J. W. (Coll.
d., Pennsylvania St. U., Hershey, Pa.) and S. A.
njamin. *Proc Am Assoc Cancer Res* 13(March):51,
72.

51 ENHANCEMENT OF TUMOR SYNGRAFT GROWTH BY
IMMUNOGENIC TUMORS AND ANTISERUM. *(E.)*
ssup, J. M. (New York Med. Coll., N.Y.) and R.
Madden. *Proc Am Assoc Cancer Res* 13(March):64,
72.

2 SPECIFIC INHIBITION OF RECEPTORS FOR
CYTOPHILIC ANTIBODIES ON MACROPHAGES BY
)-ANTIBODY. *(E.)* Mitchell, M. S. (Yale U.
. Med., New Haven, Conn.), M. B. Mokyr and D. J.
dwater. *Proc Am Assoc Cancer Res* 13(March):62,
2.

3 EVIDENCE FOR AN IMMUNE RESPONSE TO
ENDOGENOUS RNA TUMOR VIRUS IN RFM MICE.
) Hanna, M. G., Jr. (Biol. Division, Oak
ge Natl. Lab., Tenn.), R. W. Tennant, J. M. Yuhas
N. K. Clapp. *Proc Am Assoc Cancer Res*
March):59, 1972.

4 COAT AND CORE ANTIGENS OF M-PM VIRUS
ISOLATED FROM SPONTANEOUS MAMMARY CAR-
OMA OF A MONKEY. *(E.)* Ahmed, M. (Pfizer
., Maywood, N.J.), H. Molnar, S. Slattery and
Schidlovsky. *Proc Am Assoc Cancer Res* 13(March):
1972.

5 RELATIONSHIP BETWEEN GLYCOCALYX DISSOCIABILI-
TY, SURFACE ANTIGEN SOLUBILITY, IMMUNO-
ICITY, AND METASTASIZING CAPACITY OF MAMMARY
CINOMAS. *(E.)* Kim, U. (Roswell Park Mem.
., Buffalo, N.Y.) and C. Carruthers. *Proc*
Assoc Cancer Res 13(March):69, 1972.

5 CHROMOSOMAL AND BIOLOGICAL CHANGES OF
H-4-II-E RAT HEPATOMA CELLS AFTER
NSPLANTATION INTO HYDROCORTISONE TREATED AND X-
ADIATED HAMSTERS. *(E.)* Muragishi, H.
lahoma Med. Res. Fdn., Oklahoma City), B. J.
ening, W. Joel, C. A. Lovig and R. H. Bottomley.
Am Assoc Cancer Res 13(March):68, 1972.

A BETA MIGRATING FETAL PROTEIN FOUND IN
MALIGNANT AND DISEASED TISSUES. *(E.)*
ak, E. M. (U. Pennsylvania, Philadelphia),
. Lardis, L. M. Pizzaia, K. D. Rudberg and M.
a. *Proc Am Assoc Cancer Res* 13(March):68, 1972.

ANALYSIS OF IMMUNE COMPLEX DISEASE IN
LEUKEMIA AND LYMPHOMA PATIENTS: THE
ENCE OF AN ONCORNAVIRUS RELATED ANTIGEN. *(E.)*
Sutherland, J. C. (Natl. Cancer Inst., Baltimore, Md.) and M. R. Mardiney, Jr. *Proc Am Assoc Cancer Res* 13(March):68, 1972.

0469 INFLUENCE OF TUMOR CELL IRRADIATION AND SPECIFIC SERUM FACTOR(S) ON LYMPHOCYTE BLASTOGENIC RESPONSES TO SOLID TUMORS. *(E.)* Mavligit, G. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), E. Hersh and C. McBride. *Proc Am Assoc Cancer Res* 13(March):67, 1972.

0470 CARCINOEMBRYONIC ANTIGEN IN TUMOR TISSUES AND URINE FROM CANCER PATIENTS. *(E.)* Kithier, K. (Dept. Oncology, Wayne St. U., Detroit, Mich.), M. Al-Sarraf, J. Cejka and V. K. Vaitkevicius. *Proc Am Assoc Cancer Res* 13(March): 66, 1972.

0471 IMMUNITY TO ROUS SARCOMA VIRUS ELICITED BY HOMOLOGOUS AND HETEROLOGOUS AVIAN LEUKOSIS VIRUSES. *(E.)* Sigel, M. M. (U. Miami Sch. Med., Fla.), P. Meyers and H. T. Holden. *Proc Am Assoc Cancer Res* 13(March):66, 1972.

0472 SEROLOGIC DETECTION OF AN ACUTE LEUKEMIA ASSOCIATED ANTIGEN. *(E.)* Mann, D. L. (Natl. Inst. Hlth., Bethesda, Md.). *Proc Am Assoc Cancer Res* 13(March):76, 1972.

0473 SUPPRESSION OF CELLULAR IMMUNITY BY ANTIGENS RELEASED FROM DEAD TUMOR CELLS. *(E.)* Macdonald, J. L. (Natl. Inst. Hlth., Bethesda, Md.), W. J. Martin and J. R. Wunderlich. *Proc Am Assoc Cancer Res* 13(March):75, 1972.

0474 ANTIGENIC CONVERSION OF ESTABLISHED TUMORS WITH THE MOLONEY SARCOMA VIRUS (MSV). *(E.)* Basombrio, M. A. (Inst. Cancer Res., Philadelphia, Pa.). *Proc Am Assoc Cancer Res* 13(March):74, 1972.

0475 HUMAN CYTOTOXIC ANTIBODY TO ACUTE LEUKEMIA. *(E.)* Bias, W. B. (Johns Hopkins U., Baltimore, Md.), G. W. Santos, P. J. Burke, P. Anderson, D. Klein, G. Mullins and R. Humphrey. *Proc Am Assoc Cancer Res* 13(March):73, 1972.

0476 HYBRID RESISTANCE TO PARENTAL BONE MARROW GRAFTS: GENETIC AND IMMUNOLOGIC POLYMORPHISM. *(E.)* Lotzova, E. (Dept. Path., St. U. New York, Buffalo) and G. Cudkowicz. *Proc Am Assoc Cancer Res* 13(March):72, 1972.

0477 TUMOR IMMUNITY INDUCED WITH A SEA STAR EXTRACT. *(E.)* Graziano, K. D. (Johns Hopkins U. Med. Sch., Baltimore, Md.), R. A. Prendergast and G. W. Santos. *Proc Am Assoc Cancer Res* 13(March):72, 1972.

0478 ANTIGENIC ALTERATION OF FRIEND SPLEEN FOCUS-FORMING VIRUS (SFFV) GROWN WITH HUMAN LEUKEMIC TISSUE EXTRACTS. *(E.)* Eckner, R. J. (Roswell Park Mem. Inst., Buffalo, N.Y.) and R. A. Steeves. *Proc Am Assoc Cancer Res* 13(March):70, 1972.

0479 IMMUNOLOGIC PARAMETERS OF RESISTANCE TO ISOLOGOUS ADENOVIRUS 12 INDUCED MOUSE TUMORS. *(E.)* Berman, L. D. (Mallory Inst. Path., Boston, Mass.). *Proc Am Assoc Cancer Res* 13(March):85, 1972.

0480 HL-A ANTIGEN FREQUENCIES IN CANCER. *(E.)* Takasugi, M. (U. California, Los Angeles), B. Henderson, M. R. Mickey and P. I. Terasaki. *Proc Am Assoc Cancer Res* 13(March):85, 1972.

0481 LYMPHOCYTE BLASTOGENIC RESPONSE TO AUTOLOGOUS LEUKEMIA CELLS AND THE DEMONSTRATION OF A SERUM INHIBITORY EFFECT. *(E.)* Gutterman, J. U. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), K. B. McCredie and E. M. Hersh. *Proc Am Assoc Cancer Res* 13(March):80, 1972.

0482 GW-39: A HUMAN TUMOR - ANIMAL MODEL FOR CARCINOEMBRYONIC ANTIGEN (CEA). *(E.)* Goldenberg, D. M. (Temple U. Sch. Med., Philadelphia, Pa.) and H. J. Hansen. *Proc Am Assoc Cancer Res* 13(March):80, 1972.

0483 CELLULAR IMMUNITY (CMI) IN MALIGNANT MELANOMA. *(E.)* Clark, D. A. (New England Med. Ctr. Hosp., Boston, Mass.) and L. Nathanson. *Proc Am Assoc Cancer Res* 13(March):79, 1972.

0484 ANTIGENS ASSOCIATED WITH HODGKIN'S DISEASE TUMORS. *(E.)* Chism, S. E. (Harvard Joint Ctr. Radiation Therap., Boston, Mass.), S. E. Order and S. Hellman. *Proc Am Assoc Cancer Res* 13(March):78, 1972.

0485 CYTOTOXIC EFFECT OF AUTOLOGOUS LYMPHOCYTES AND PLASMA ON CULTURED HUMAN TUMORS. *(E.)* Giovanella, B. C. (Baylor Coll. Med., Houston, Tex.), D. M. Mumford, C. S. Santamaria and J. S. Stehlin. *Proc Am Assoc Cancer Res* 13(March):77, 1972.

0486 IMMUNOLOGIC CLASSIFICATION OF CARCINOMA OF THE URINARY BLADDER. *(E.)* Davidsohn, I. (Chicago Med. Sch., Ill.), R. Stejskal and P. Lill. *Proc Am Assoc Cancer Res* 13(March):77, 1972.

0487 EFFECTS OF MYCOBACTERIUM BOVIS (BCG) ON TUMOR CELL GROWTH IN MUSCLE. *(E.)* Littman, B. (Natl. Cancer Inst., Bethesda, Md.), B. Zbar and H. J. Rapp. *Proc Am Assoc Cancer Res* 13(March):96, 1972.

0488 DETECTION OF CELL SURFACE CHANGES IN HUMAN LEUKEMIC LEUKOCYTES USING PLANT LECTINS. *(E.)* Krauss, S. (U. Tennessee Mem. Res. Ctr., Knoxville). *Proc Am Assoc Cancer Res* 13(March):95, 1972.

0489 RAPID DETECTION OF CARCINOEMBRYONIC ANTIGEN BY RADIOIMMUNO-ELECTROPHORETIC PRECIPITATION. *(E.)* Coller, J. A. (Dept. Surg. Res., U. Pennsylvania, Philadelphia), L. K. Yin, R. W. Crichlow. *Proc Am Assoc Cancer Res* 13(March): 95, 1972.

0490 ABH ISOANTIGENS AND THEIR SYNTHETIC AND DEGRADATIVE ENZYMES IN HUMAN COLONIC CARCINOMA. *(E.)* Kiang, D. T. (U. Minnesota Med. Sch., Minneapolis), R. D. Edstrom and B. J. Kennedy. *Proc Am Assoc Cancer Res* 13(March):93, 1972.

0491 HL-A ANTIGENS IN HODGKIN'S DISEASE. *(E.)* Graff, K. S. (Natl. Inst. Hlth., Bethesda, Md.), R. M. Simon, G. N. Rogentine and R. A. Yankee. *Proc Am Assoc Cancer Res* 13(March):94, 1972.

0492 PROPERTIES OF THE UNIQUE GLYCOPROTEIN CHARACTERIZING THE PRESENCE OF THE MAMMARY TUMOR AGENT. *(E.)* Miroff, G. (Biomed. Res. Lab., Monroe, N.Y.) and H. M. Meade. *Proc Am Assoc Cancer Res* 13(March):87, 1972.

0493 USE OF IMMUNOHISTOCHEMICAL STAINING (IHCS) FOR RECOGNITION OF NEOPLASTIC AND PRENEOPLASTIC PITUITARY LESIONS. *(E.)* Furth, J. (Inst. Cancer Res., Columbia U., New York, N.Y.) and P. Moy. *Proc Am Assoc Cancer Res* 13(March):87, 1972.

0494 ENHANCED TUMOR GROWTH IN ANIMALS PRETREATED WITH COMPLETE FREUND'S ADJUVANT (CFA). *(E.)* Meltzer, M. S. (Natl. Cancer Inst., Bethesda, Md.), E. J. Leonard and T. Borsos. *Proc Am Assoc Cancer Res* 13(March):106, 1972.

0495 IMMUNOGLOBULINS OF BRONCHIAL SECRETIONS IN LUNG CANCER. *(E.)* DeCosse, J. J. (Med. Coll. Wisconsin, Milwaukee), M. A. Mandel and K. Dvorak. *Proc Am Assoc Cancer Res* 13(March):104, 1972.

0496 STUDIES OF THE SPECIFICITY OF THE SYSTEMIC ASPECT OF GOMPERTZIAN TUMOR GROWTH SLOWING. *(E.)* DeWys, W. (U. Rochester, N.Y.) and J. Mansky. *Proc Am Assoc Cancer Res* 13(March):104, 1972.

0497 TUMOR-HOST RELATIONSHIP IN IMMUNE RESPONSE TO MODIFIED LYMPHOMA CELLS. *(E.)* Prager, M. D. (U. Texas Southwestern Med. Sch., Dallas), S. Ticaric and C. L. Merrill. *Proc Am Assoc Cancer Res* 13(March):103, 1972.

98 STIMULATION OF LYMPHOCYTE PROTEIN SYNTHESIS: USE FOR THE RAPID EVALUATION OF
:'S AND CELL-MEDIATED IMMUNITY TO SOLUBLE ANTIGENS.
.) Rosenberg, S. A. (Natl. Inst. Hlth., Bethesda,
.), R. Levy, B. Schechter and S. Ficker. *Proc*
Assoc Cancer Res 13(March):100, 1972.

99 PREVENTION OF GRAFT *VERSUS* HOST DISEASE (GVHD) WITH ANTISERUM (AS) OR CYTOXAN (CY)
RING IMMUNOTHERAPY OF TRANSPLANTABLE MOLONEY
JKEMIA (LSTRA). *(E.)* Kende, M. (Natl. Cancer
t., Bethesda, Md.), L. Keys and J. P. Glynn.
oc Am Assoc Cancer Res 13(March):101, 1972.

00 IMMUNE RESPONSES TO FETAL TISSUES AND COMPARISON TO ANTI-TUMOR REACTIVITY.
) Ambrose, K. R. (U. Tennessee, Knoxville),
G. Anderson and J. H. Coggin, Jr. *Proc Am*
oc Cancer Res 13(March):97, 1972.

1 ADJUVANT EFFECTS IN TUMOR IMMUNITY: *MYCOBACTERIUM BOVIS* (BCG), FREUND'S
JVANT (CFA) AND *BORDATELLA PERTUSSIS*. *(E.)*
tlett, G. L. (Natl. Cancer Inst., Bethesda,
), B. Zbar and H. J. Rapp. *Proc Am Assoc*
cer Res 13(March):39, 1972.

2 REPRESSION AND DEREPRESSION OF GROSS LEUKEMIA VIRUS ANTIGENS. *(E.)* Ioachim,
L. (Lenox Hill Hosp., New York, N.Y.), B.
sett, M. Sabbath and S. Keller. *Proc Am Assoc*
cer Res 13(March):38, 1972.

3 PHYSIOLOGIC AND SEROLOGIC SIMILARITIES OF SERA FROM PREGNANT AND TUMOR-BEARING
MALS. *(E.)* Otten, J. A. (Biol. Division,
Ridge Natl. Lab., Tenn.) and R. L. Tyndall.
c Am Assoc Cancer Res 13(March):37, 1972.

4 THE EFFECT OF VARIOUS ENZYMES AND CHELATING AGENTS ON THE TUMOR SPECIFIC
NSPLANTATION ANTIGEN(S) (TSTA) OF SV-40 INDUCED
OR CELLS. *(E.)* Blakeslee, J. R., Jr. (Roswell
k Mem. Inst., Buffalo, N.Y.). *Proc Am Assoc*
cer Res 13(March):31, 1972.

5 INTERACTION OF CELLS AND ANTIBODIES IN IMMUNITY TO MAMMARY TUMORS IN MICE.
) Stutman, O. (Dept. Path., U. Minnesota,
. Sch., Minneapolis). *Proc Am Assoc Cancer*
13(March):2, 1972.

5 CELLULAR HYPERSENSITIVITY TO *IN SITU* BREAST CANCER. *(E.)* Black, M. M.
York Med. Coll., New York), S. J. Cutler and
H. C. Barclay. *Proc Am Assoc Cancer Res*
March):2, 1972.

0507 FETAL ANTIGENS AND TUMOR SPECIFIC CELL SURFACE ANTIGENS OF PAPOVA VIRUS-INDUCED TUMORS. *(E.)* Ting, C. C. (Natl. Cancer Inst., Bethesda, Md.), D. H. Lavrin and R. B. Herberman. *Proc Am Assoc Cancer Res* 13(March); 50, 1972.

0508 IMPAIRED INFLAMMATORY RESPONSE IN TUMOR BEARING ANIMALS. *(E.)* Bernstein, I. D. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 13(March):47, 1972.

0509 CORRELATION OF TUMOR SPECIFIC IMMUNITY WITH EXTENT OF DISEASE IN MALIGNANT MELANOMA. *(E.)* Byrne, M. (Tufts-Lemuel Shattuck Pondville Hosp., Boston, Mass.), G. Heppner, L. Stolback, F. J. Cummings, E. McDonough and P. Calabresi. *Proc Am Assoc Cancer Res* 13(March):46, 1972.

0510 EMBRYO-SPECIFIC ANTIGENS IN MURINE TUMORS. *(E.)* Stonehill, E. H. (Sloan-Kettering Inst., New York, N.Y.), A. Bendich, P. J. Higgins and E. Borenfreund. *Proc Am Assoc Cancer Res* 13(March):43, 1972.

0511 ENHANCEMENT OF TRANSPLANTED RadLV-INDUCED LYMPHOMAS IN C57BL MICE BY FREUND'S ADJUVANT. *(E.)* Wong, J. K. Y. (Dept. Path., U. California, Los Angeles), M. L. Hart and W. H. Carnes. *Proc Am Assoc Cancer Res* 13(March):42, 1972.

0512 PLASMA MEMBRANE PROTEINS OF CHICKEN LYMPHOID CELLS AND MAREK'S DISEASE TUMOR CELLS. *(E.)* Ragland, W. L. (Coll. Vet. Med., U. Georgia, Athens), J. L. Pace and R. L. Doak. *Proc Am Assoc Cancer Res* 13(March):42, 1972.

0513 IMMUNOGENIC ACTIVITY OF TUMOR ANTIGENS RETAINED BY LIVER RETICULOENDOTHELIAL CELLS. *(E.)* Vaage, J. (U. Texas M. D. Anderson Hosp., Houston). *Proc Am Assoc Cancer Res* 13(March):30, 1972.

0514 CELL-ASSOCIATED MAMMARY TUMOR VIRUS (MTV) ANTIGENS IN MOUSE MAMMARY TUMOR CELL CULTURES. *(E.)* Yagi, M. J. (Dept. Bacteriology, Immunology, U. California, Berkeley). *Proc Am Assoc Cancer Res* 13(March):29, 1972.

0515 ANTIGEN (S_2) IN HUMAN SARCOMAS AND FETAL TISSUES. *(E.)* Mukherji, B. (Sloan-Kettering Inst., New York, N.Y.) and Y. Hirshaut. *Proc Am Assoc Cancer Res* 13(March):21, 1972.

0516 HERPES ZOSTER AND MALIGNANT LYMPHOMAS. *(E.)* Miller, D. G. (Mem. Hosp. Cancer

Allied Dis., New York, N.Y.). *Proc Am Assoc Cancer Res* 13(March):21, 1972.

0517 HOST IMMUNITY TO A GROWING TRANSPLANTED MCA-INDUCED GUINEA PIG (GP) SARCOMA. *(E.)* Cohen, A. M. (Natl. Cancer Inst., Bethesda, Md.), R. C. Miller and A. S. Ketcham. *Proc Am Assoc Cancer Res* 13(March):18, 1972.

0518 EFFECT OF CYCLOPHOSPHAMIDE ON HOST RESPONSE TO SYNGENIC TUMOR TRANSPLANTS. *(E.)* Lausch, R. (M. S. Hershey Med. Ctr., Pennsylvania St. U., Hershey), S. Ross and F. Rapp. *Proc Am Assoc Cancer Res* 13(March):13, 1972.

0519 MIXED LEUKOCYTE CULTURES (MLC) REACTIVITY TO LEUKEMIA CELLS IN PATIENTS, THEIR TWINS, AND HL-A IDENTICAL SIBLINGS. *(E.)* Halterman, R. H. (Natl. Cancer Inst., Bethesda, Md.) and B. G. Leventhal. *Proc Am Assoc Cancer Res* 13(March):6, 1972.

0520 L-ASPARAGINASE AND LYMPHOCYTE BLASTOGENESIS: EFFECTS ON CELL SURFACE. *(E.)* Fidler, I. J. (Sch. Dental Med., U. Pennsylvania, Philadelphia) and P. C. Montgomery. *Proc Am Assoc Cancer Res* 13(March):5, 1972.

0521 AFFINITY LABELING OF A MOUSE IgG2a MYELOMA PROTEIN WITH BINDING AFFINITY FOR NITROPHENYL LIGANDS. *(E.)* Martin, N. (Dept. Biol., U. California at San Diego, La Jolla), N. L. Warner, P. E. Roeder and S. J. Singer. *Biochemistry* 11(26): 4999-5005, 1972.

0522 SOME FUNCTIONAL PECULIARITIES IN LYMPHOCYTES OF CHILDREN SUFFERING FROM ACUTE LEUKEMIA. *(Rus.)* Iskanderova, V. R. (Pediat. Inst. USSR Acad. Med. Sci. Moscow) E. B. Vladimirskaya and I. V. Koshel. *Probl Gematol Pereliv Krovi* (6):3-6, 1972.

0523 PRESENCE OF CV VIRUS ANTIGEN IN NORMAL AND CANCER PATIENT SERA. *(Sp.)* Valladares, Y. (Natl. Inst. Oncology, City U., Madrid, Spain), Y. Alvarez and J. Alvarez. *Rev Esp Oncol* 17(2):23-40, 1970.

0524 PURIFICATION OF HUMAN MYELOMA IgD'S BY MEANS OF IMMUNE ADSORPTION. *(Fr.)* Rivat, C. (Natl. Inst. Hlth Med. Res., Seine Maritime, France) and C. Ropartz. *C R Acad Sci [D] (Paris)* 275(23):2787-2790, 1972.

0525 NATURAL AND INDUCED RESISTANCE TO ATPC+ TUMOR IN MICE. *(It.)* Marconi, P. (Microbiol. Inst. U. Perugia, Italy), M. Pitzurra, F. Bistoni and A. Iorio. *Boll Ist Sieroter Milan* 51(1):27-43, 1972.

0526 BLOOD GROUPS OF ERYTHROCYTES IN THE ABO SYSTEM IN PATIENTS WITH MAMMARY CARCINOMA. *(Pol.)* Jaegerman, K. (Inst. Forensic Med., Acad. Med. Krakow, Poland), H. Glinska, K. Smolak and A. Urban. *Patol Pol* 23(3):327-334, 1972.

0527 MODIFICATION OF ABO SYSTEM TRANSPLANTATION ANTIGENS CARRIED BY HUMAN TUMOR CELLS: AN ATTEMPT IN AROUSING THE IMMUNE DEFENSE CAPABILITIES OF THE ORGANISM. *(Fr.)* Mousseron-Canet, M. (Superi Natl. Sch. Chem., Montpellier, France) R. Magous and C. Lecou. *C R Acad Sci* [*D*] *(Paris)* 275(25): 3065-3068, 1972.

0528 HETEROTRANSPLANTATION OF TUMOURS. IX. CONDITIONING OF THE RECIPIENT-CHECKING OF THE EFFECTIVENESS OF SUPPRESSION *IN VITRO (E.)* Oravec, C. (Cancer Res. Inst., Slovak Acad. Sci., Bratislava, Czechoslovakia), J. Cambelova, A. Sakalova and B. Ondrus. *Neoplasma* 19(5):531-545, 1972.

0529 HOST-TUMOUR RELATIONSHIP. XXX. A FAILURE TO DEMONSTRATE A SPECIAL HYALURONIDASE INHIBITOR IN THE BLOOD SERUM OF CANCER PATIENTS. *(E.)* Kolarova, M. (Cancer Inst., Brno, Czechoslovakia). *Neoplasma* 19(5):547-551, 1972.

0530 HYPERINFECTION SYNDROME WITH STRONGYLOIDES STERCORALIS IN MALIGNANT LYMPHOMA. *(E.)* Adam, M. (Dept. Med., U. West Indies, Kingston, Jamaica), O. Morgan, C. Persaud and W. N. Gibbs. *Br Med J* 1(5848):264-266, 1973.

0531 HL-A MATINGS IN TROPHOBLASTIC NEOPLASIA. *(E.)* Klouda, P. T. (Royal Marsden Hosp., London, England), S. D. Lawler and K. D. Bagshawe. *Tissue Antigens* 2(3):280-284, 1972.

0532 STUDIES ON SKIN ALLOGRAFT REACTION IN CLOSED COLONEY BLACK HAIRY MICE PREVIOUSLY IMPLANTED WITH SELECTED MALIGNANT TUMORS. *(E.)* Uchiyama, M. (Yokohama City U. Sch. Med., Japan). *Yokohama Med Bull* 22(3-4):49-71, 1971.

0533 ROLE OF REGIONAL LYMPHATICS IN TUMOR ALLOGRAFT REJECTION. *(E.)* Futrell, J. W. (Natl. Cancer Inst., Bethesda, Md.) and G. H. Myers. *Transplantation* 13(6):551-557, 1972.

0534 AN EXPERIMENTAL STUDY ON EFFECT OF THE SPLEEN AGAINST TUMOR TRANSPLANTATION. *(Jap.* Saito, Y. (Tokyo Med. Coll., Japan). *J Tokyo Med Coll* 29(6):759-772, 1971.

0535 PROTECTION BY SUBCELLULAR FRACTIONS PREPARED FROM LYMPH-NODES AND SPLEENS OF

ECIFICALLY ALLERGIZED C57 BL MICE AGAINST FATAL
ALLENGE WITH BP8 ASCITES TUMOUR CELLS IN C3H
CE. *(E.)* Fraser, L. B. (Dept. Path., U. Cambridge,
gland) and D. B. Cater. *Br J Exp Pathol* 53:98-110,
72.

6 A METHOD OF SEPARATING THE LYMPH-NODE
CELLS, OF C57 BL MICE ALLERGIZED WITH BP8
CITES TUMOUR, INTO TWO FRACTIONS WITH A FAST AND
W ELECTROPHORETIC MOBILITY: DEMONSTRATION THAT
Y THE "FAST LNC" WILL PROTECT C3H MICE AGAINST
ALLENGE WITH THE TUMOUR. *(E.)* Thomas, C. B.
ept. Path., U. Cambridge, England) and D. B.
ter. *Br J Exp Pathol* 53:111-124, 1972.

37 CARCINOEMBRYONIC ANTIGEN (CEA) IN COLONIC
CANCER. USE IN PREOPERATIVE AND POST-
ERATIVE DIAGNOSIS AND PROGNOSIS. *(E.)* Dhar, P.
ston City Hosp., Mass.), T. Moore, N. Zamcheck
H. Z. Kupchik. *JAMA* 221(1):31-35, 1972.

See also:

* (Rev): 0004, 0005, 0007, 0012, 0014, 0015, 0021
* (Chem): 0025, 0034, 0042, 0048, 0065, 0072, 0075, 0105, 0149
* (Phys): 0215
* (Viral): 0228, 0229, 0236, 0238, 0252, 0258, 0261, 0264, 0270, 0276, 0279, 0290, 0291, 0295, 0303, 0306, 0325
* (Path): 0541

0538 DNA POLYMERASE ACTIVITY AND DNA SYNTHESIS IN PRENEOPLASTIC NODULE OUTGROWTHS OF BALB/c AND C3H MOUSE MAMMARY GLAND. *(E.)*

Banerjee, M. R. (Dept. Zool. Inst. Cellular Res., U. Nebraska, Lincoln), R. G. Mehta and J. E. Wagner. *J Natl Cancer Inst* 50(2):339-345, 1973.

DNA polymerase activity and DNA synthesis in four outgrowth lines of preneoplastic nodules (HAN) of BALB/c and C3H mouse mammary gland were determined. The rate of ^{3}H-thymidine triphosphate (^{3}H-dTTP) incorporation into trichloroacetic acid-insoluble material *in vitro* was used as a measure of DNA polymerase (DNA nucleotidyl transferase) activity in the postmicrosomal supernatant fraction of the HAN tissues. Incorporation of ^{3}H-thymidine into DNA after a single intraperitoneal injection of the DNA precursor was used as a measure of DNA synthesis in the HAN cells *in vivo*. Both DNA polymerase and DNA synthesis in mammary tumor virus (MTV)-free and MTV-positive D1 nodule outgrowths of BALB/c mice were low. A relatively higher DNA polymerase activity and DNA synthesis were observed in the high tumor-producing, MTV-free D8 nodule of BALB/c mice. The highest level of DNA polymerase activity and DNA synthesis was in the high tumor-producing, MTV-positive CH33 nodule outgrowth line of C3H mice. Unlike the normal mouse mammary cells, both DNA polymerase activity and DNA synthesis in the preneoplastic lesions were independent of the ovarian hormones. The HAN outgrowth lines of both BALB/c and C3H mice, regardless of their tumorigenic potential, exhibit an altered responsiveness to normal regulation on DNA polymerase and DNA synthetic activities. The significance of this characteristic loss of normal control to suppress DNA polymerase and DNA synthesis in the preneoplastic lesions with regard to neoplastic transformation is discussed.

0539 CONTROL OF ADENYLATE CHARGE IN THE MORRIS "MINIMAL-DEVIATION" HEPATOMAS. *(E.)*

Criss, W. E. (U. Florida Coll. Med., Gainesville). *Cancer Res* 33(1):51-56, 1973.

The activity of several enzymes that contribute to the cellular adenylate energy charge and the change in the total pool of adenine nucleotides were measured in groups of highly and poorly differentiated Morris "minimal-deviation" hepatomas. Tabulation of enzymatic measurements were made for several substrate level phosphorylating enzymes by setting up a ratio of cytoplasmic to mitochondrial nonoxidative phosphorylation potential. The ratio was 0.44 in normal liver tissue, 0.50 in the group of highly differentiated hepatomas, and 22.2 in the group of poorly differentiated hepatomas. The activities of several enzymes that are involved in the transportation of reducing equivalents across the mitochondrial membrane were made. Cytoplasmic NAD$^+$-malate dehydrogenase was over 500 units in normal liver and less than 100 units in the group of poorly differentiated hepatomas. Cytoplasmic NAD$^+$-glycerophosphate dehydrogenase was 120 units in liver tissue and about 20 units in the group of poorly differentiated hepatomas. NAD$^+$-malate dehydrogenase is a key enzyme in the malate-aspartate shuttle system; NAD$^+$-glycerophosphate dehydrogenase is a key enzyme in the α-glycerophosphate shuttle system. Comparison of the ratio of the activities of NAD$^+$-glycerophosphate dehydrogenase *versus* NAD$^+$-lactate dehydrogenase allows for the examination of the competing hydrogen-accepting systems of phospholipid synthesis *versus* anaerobic metabolism. The ratio was 0.5 in liver tissue and 0.1 in the group of poorly differentiated hepatomas. Measurement of adenosine mono-, di, and triphosphates and calculations of *Q* (a reaction parameter based on the adenylate kinase reaction) and the energy charge of the adenylate system were performed. The *Q*'s for liver tissue and groups of highly and poorly differentiated hepatomas were 1.42, 1.88, and 2.06, resp. The calculated adenylate charges were 0.87 in normal liver tissue, 0.89 in a group of highly differentiated hepatomas, and 0.90 in a group of poorly differentiated hepatomas. It would appear that the poorly differentiated hepatomas have acquired the potential to maintain a high energy charge without the need for large mitochondria involvement.

0540 CONTROL OF ADENYLATE CHARGE IN NOVIKOFF ASCITES CELLS. *(E.)*

Criss, W. E. (U. Florida Coll. Med., Gainesville). *Cancer Res* 33(1):57-64, 1973.

The activity of several enzymes that contribute to the cellular adenylate energy charge, the changes in the total pool of adenine nucleotides, and the relationship of cytoplasmic *versus* mitochondrial maintenance of the adenylate charge were studied in Novikoff ascites cells. The activities of several substrate-level phosphorylating enzymes were analyzed. Tabulation of these enzymatic measurement were made by setting up a theoretical ratio of cytoplasmic to mitochondrial nonoxidative phosphorylatio potential. The ratio was 0.44 in liver tissue and 72.2 in the Novikoff ascites cells. The activities of several enzymes that are involved in the transportation of reducing equivalents across the mitochondrial membranes were made. NAD$^+$-malate dehydrogenas was 500 units in liver tissue and 110 units in Novikoff ascites cells. Cytoplasmic NAD$^+$-glycerophosphate dehydrogenase was 120 units in liver tissue an 11 units in Novikoff ascites cells. NAD$^+$-malate dehydrogenase is a key enzyme in the malate-aspartat shuttle system; NAD$^+$-glycerophosphate dehydrogenase is a key enzyme in the α-glycerophosphate shuttle system. Comparison of the ratio of the activities of NAD$^+$-glycerophosphate dehydrogenase *versus* NAD$^+$-lactate dehydrogenase allows for the examination of the competing hydrogen-accepting systems of phospholipid synthesis *versus* anaerobic metabolism. The ratio was 0.5 in liver tissue and 0.05 in Novikoff ascites cells. Measurement of adenosine mono-, di-, and triphosphates were made in Novikoff ascites cells incubated under various metabolic conditions. The reaction parameter *(Q)* as related to adenylate kinase reaction and the adenylate energy charge expression were calculated from these measurements. Anaerobically, and without a carbon source, the enylate charge fell from 0.91 to 0.12 within 30 n of incubation; the *Q* decreased from 4 to near (*Q* for adenylate kinase at equilibrium is 1.2). and adenylate charge were maintained during anaero- c metabolism with glucose but not with pyruvate or ccinate. Addition of 2-deoxyglucose to the aerobic system caused the adenylate charge to fall en more rapidly, but this effect was overcome with glucose:2-deoxyglucose ratio of 3:1. Aerobic tabolism in the presence of glyoxylate showed a crease in the adenylate charge from 0.91 to 0.64. utamate but not pyruvate, citrate, or isocitrate uld overcome the effect of glyoxylate. With a mbination of carbon sources and pathway inhibitors, would appear that either the cytoplasmic enzymatic stems or the mitochondrial enzymatic systems, by emselves, are capable of maintaining a high (>0.9) enylate charge in Novikoff ascites cells, if they ve the necessary and proper carbon source. It is kely that tumor mitochondria are functionally und but are unable to function at maximum capacity cause they are unable to obtain enough adequate ecursor metabolites.

41 PROPORTION OF T AND B LYMPHOCYTES IN LESIONS OF MAREK'S DISEASE: THEORETICAL IM- ICATIONS FOR PATHOGENESIS.

(E.) Rouse, B. T. all Inst. Med. Res., Melbourne, Australia), R. J. Wells and N. L. Warner. *J Immunol* 110(2):534- 9, 1973.

ll preparations from visceral tumors and nerve sions of chickens with Marek's disease (MD) were amined with antisera specific for avian T and B nphocytes. In all instances examined, the large jority of lymphoid cells in the infiltrate were T nphocytes, with only 3 to 10% of B lymphocytes esent. It is suggested that the pathogenesis of is disease is strictly dependent on immunologically diated events. It is believed that after infec- on with the MD agent the virus is established in rtain nonlymphoid cells of the body. After some gree of viral proliferation, an antigenic challenge pressed as viral-associated, cell surface antigen presented to the host. This induces a marked oliferative response in T cells which infiltrate e lesion. Under continued intense antigenic imulation, a pathologic process is established ich is primarily initiated by the T cell. Many ctors, such as are involved in T cell-mediated mune responses like delayed hypersensitivity, y then be liberated and contribute in a secondary nner to the full expression of the clinical ndrome.

42 CYCLIC ADENOSINE 3',5'-MONOPHOSPHATE PHOSPHODIESTERASE ACTIVITY IN NORMAL, FERENTIATING, REGENERATING, AND NEOPLASTIC LIVER.

) Clark, J. F. (Indiana U. Sch. Med., lianapolis), H. P. Morris and G. Webster. *Cancer* 33(2):356-361, 1973.

behavior of cyclic 3',5'-nucleotide phospho- sterases [cyclic adenosine 3',5'-monophosphate (cAMP) phosphodiesterases] was compared in normal resting liver, proliferating normal liver (from both developing and partially hepatectomized rats), and neoplastic liver (from both slowly and rapidly growing hepatomas). The enzyme activity was determined in the 100,000 X g supernant fluid at 1 mM cAMP (saturating substrate concentration, "high-K_m enzyme") and at 1 μM cAMP (near physiological substrate level, "low-K_m enzyme"). The pH curves for both liver and the rapidly growing hepatoma 3924A were between pH 6.0 and 9.0. The results of kinetic studies and enzyme assays at 2 cAMP concentrations suggest, but do not yet prove rigorously, the presence in normal, newborn, and regenerating liver, and in the hepatomas, of two cAMP phosphodiesterases, one with a low apparent K_m of 2 to 3 μM for cAMP and another with an apparent K_m of 100 to 600 μM. The high-K_m phosphodiesterase activity decreased in the slowly growing hepatoma 47C and the rapidly growing hepatoma 3924A to 58% and 13%, resp., of the values of control normal livers. The low-K_m phosphodiesterase activity in slowly and rapidly growing hepatomas increased to 185% and 265%, resp. This alteration in the isozyme pattern of cAMP phosphodiesterase appears to be specific to the neoplastic process, as no similar pattern was observed in the rapidly growing differentiating or regenerating liver. The behavior of cAMP phosphodiesterase is in line with the change in pattern observed for the hexokinase and pyruvate kinase systems, and the observations support the view that neoplastic transformation entails a progressive emergence of a reprogramming of gene expression that appears to be linked with tumor growth rate.

0543 ULTRASTRUCTURAL AUTORADIOGRAPHIC STUDIES OF THE EARLY VASOPROLIFERATIVE RESPONSE IN TUMOR ANGIOGENESIS.

(E.) Cavallo, T. (Mallory Inst. Path., Boston, Mass.), R. Sade, J. Folkman and R. S. Cotran. *Am J Pathol* 70(3):345-362, 1973.

The early local vasoproliferative response induced by live tumor cells and an extract derived from such cells was studied in rat subcutaneous tissue by means of electron microscopy and ultrastructural autoradiography after local injections of tritium-labeled thymidine. DNA synthesis was localized in endothelial cells, pericytes, and perivascular cells 6 to 8 hours after exposure to 10^6 live Walker ascites tumor cells. At this time, DNA-synthesizing endothelial cells in parent vessels exhibited a continuous basement membrane and could not be readily differentiated, ultrastructurally, from control endothelium. At 48 to 50 hours, the number of labeled cells increased and there was ultrastructural evidence of regenerating endothelium: marked increase in ribosomes and endoplasmic reticulum, scarce or absent pinocytotic vesicles, attenuated or discontinuous basement membrane and marked irregularities in cytoplasmic surfaces. Labeled endothelial cells were present in parent vessels, as well as along newly formed sprouts. Autoradiographic and ultrastructural findings after tumor extract or live tumor cells at 48 hours were similar. Evidence was also presented that cells which were recognizable as pericytes, by ultrastructural criteria and by their localization within the basement membrane, were capable of DNA synthesis and mitosis.

0544 DEVIATIONS IN ISOZYME PATTERNS OF ACID PHOSPHATASE AND ESTERASE, AND IN ULTRASTRUCTURE OF YOSHIDA ASCITES HEPATOMAS FROM RAT HEPATOCYTES. *(E.)* Kaneko, A. (Sapporo Med. Coll., Japan), K. Dempo, T. Onoe and H. Isaka. *Gann* 63(6):747-756, 1972.

Enzyme activities were compared in cells from four strains of Yoshida ascites hepatoma. The ratio of acid phosphatase activity toward substrate phenyl phosphate to that toward β-glycerophosphate was higher in hepatoma cells than in normal liver cells. Relative activities of acid phosphatase with 1-napthyl phosphate, β-glycerophosphate, CMP and AMP were lower in hepatomas than in normal liver. The electrophoretic pattern of esterase in three of the hepatoma lines showed Bands 2, 4 and 5 but lacked Bands 1, 3, and 6. The isozyme pattern of acid phosphatase in hepatomas was of the fetal liver type, characterized by the presence of Band A and the lack of Band B. Ultrastructurally, the ascites hepatoma cells appeared to have originated from hepatocytes; the cells contained structures partially transitional to the bile duct type of liver cell.

0545 DEFECTIVE *IN VITRO* COLONY FORMATION BY HUMAN BONE MARROW PRECEDING OVERT LEUKAEMIA. *(E.)* Senn, J. S. (Sunnybrook Hosp., Toronto, Canada) and P. H. Pinkerton. *Br J Haematol* 23(3):277-281, 1972.

0546 GRANULAR CELLS IN TUMORS AND IN NONTUMOROUS TISSUE. *(E.)* Pour, P. (U. Nebraska Med. Ctr., Omaha), J. Althoff and A. Cardesa. *Arch Pathol* 95(2):135-138, 1973.

0547 FINE STRUCTURE OF TWO LIPOSARCOMAS. *(E.)* Kalderon, A. E. (Roger Williams Gen. Hosp., Providence, Rhode Island) and W. Fethiere. *Lab Invest* 28(1):60-69, 1973.

0548 MESONEPHROMA OF THE OVARY. A CLINICAL, MORPHOLOGICAL, AND HISTOGENETIC APPRAISAL. *(E.)* Fine, G. (Henry Ford Hosp., Detroit, Mich.), H. D. Clarke and R. C. Horn, Jr. *Cancer* 31(2):398-410, 1973.

0549 ENZYME ACTIVIVTY AND DISTRIBUTION IN THE HYPERPLASTIC AND CANCEROUS HUMAN PROSTATE. *(E.)* Müntzing, J. (Res. Lab., AB Leo, Helsingborg, Sweden) and T. Nilsson. *Scand J Urol Nephrol* 6(2):107-111, 1972.

0550 HORMONAL FACTORS IN MAMMARY NODULIGENESIS AND TUMORIGENESIS IN A/Ki MICE. *(E.)* Liebelt, A. G. (Med. Coll. Georgia, Augusta) and R. A. Liebelt. *Proc Am Assoc Cancer Res* 13(March): 104, 1972.

0551 BINDING OF POLYAMINES TO TRANSFER RNA FROM *E. COLI* AND MOUSE LEUKEMIC CELLS. *(E.)* Hacker, B. (U. Rochester Med. Ctr., N.Y.) and B. J. McDermott. *Proc Am Assoc Cancer Res* 13(March):88, 1972.

0552 REQUIREMENT FOR THE ASSOCIATION OF NORMAL PARENCHYMAL CELLS WITH ABNORMAL EPITHELIAL OR MYOEPITHELIAL CELLS DURING NEOPLASTIC PROGRESSION IN MAMMARY GLAND. *(E.)* Slemmer, G. (Inst. Cancer Res., Philadelphia, Pa.). *Proc Am Assoc Cancer Res* 13(March):117, 1972.

0553 TUMORIGENESIS IN MICE AND CHANGES IN THYMIDYLATE BIOSYNTHETIC ENZYMES. *(E.)* Nahas, A. (U. Rochester, N.Y.), S. Abraham and T. C. Hall. *Proc Am Assoc Cancer Res* 13(March):113, 197

0554 RAPIDLY LABELED NUCLEAR RNA SYNTHESIS IN THE MAMMARY NODULE OUTGROWTHS. *(E.)* Banerjee, D. N. (Zoology Dept., U. Nebraska, Lincoln *Proc Am Assoc Cancer Res* 13(March):75, 1972.

0555 CYTOPHOTOMETRIC ESTIMATIONS OF RNA STAININ IN PRECANCEROUS LIVERS AND HEPATOMAS. *(E.)* Moulin, M.-C. (Cancer Inst., Montreal, Canada) and R. Daoust. *Proc Am Assoc Cancer Res* 13(March):59, 1972.

0556 A SPECIFIC CIRCULATING GRANULOCYTOPOIETIC STIMULANT (LEUKOPOIETIN G_S) IN PATIENTS WITH MYELOFIBROSIS. *(E.)* Bierman, H. R. (Inst. Cancer, Blood Res., Beverly Hills, Calif.) and J. E. Hood. *Proc Am Assoc Cancer Res* 13(March):43, 1972.

0557 EXFOLIATIVE CYTOLOGY DURING EXPERIMENTAL RESPIRATORY CARCINOGENESIS. *(E.)* Schreiber, H. (Biol. Div., Oak Ridge Natl. Lab., Tenn.) and G. Saccomanno. *Proc Am Assoc Cancer Res* 13(March):32, 1972.

0558 LIVING SOLID-STATE SURFACE IMPLANTS IN EVANS RATS. *(E.)* Bryson, G. (Cottage Hosp. Res. Inst., Santa Barbara, Calif.) and F. Bischoff. *Proc Am Assoc Cancer Res* 13(March):1, 1972

0559 HISTOLOGIC QUIESCENCE WITH SOLID-STATE IMPLANTS IN THE RAT. *(E.)* Bischoff, F. (Cottage Hosp. Res. Inst., Santa Barbara, Calif.) and G. Bryson. *Proc Am Assoc Cancer Res* 13(March):1, 1972.

See also:

- * (Rev): 0014
- * (Chem): 0097, 0103, 0108, 0132, 0134, 0144
- * (Viral): 0247
- * (Immun): 0403

60 CANCER PATTERNS IN ALAMEDA COUNTY, CALIFORNIA. *(E.)* Arellano, M. G.
alifornia Dept. Pub. Hlth.), G. Linden and J. E.
nn, Jr. *Brit J Cancer* 26:473-482, 1972.

ends in cancer incidence in Alameda County,
lifornia (near San Francisco) were analyzed from
ta covering the period 1960-1969. Cancer inci-
nce showed a consistently rising movement for
l sites, in both sexes. Outstanding features
male cancer incidence were the increases in
ng and prostate cancer during the study period.
creases in female cancer incidence were primarily
tributable to increases in lung, mammary and
erine corpus cancer. The recent increase in
ng cancer incidence among females was attributed
the fact that women as a group did not begin to
oke cigarettes until relatively recently. Negro
n had the highest incidence of prostate cancer.
ite women had the highest incidence of mammary
ncer. Negro women the highest incidence of
rvical cancer. Stomach cancer incidence appeared
be decreasing.

561 THE EPIDEMIOLOGY OF SKIN CANCER IN THE KARAGANDA REGION, USSR. *(Rus.)*
amchaninov, N. F. (No affiliation). *Vopr Onkol*
(9):56-59, 1972.

total of 2045 skin cancer patients reported in
e Karaganda region, USSR in 1950-1968 were studied
an attempt to clarify the role of climatic
ctors in the etiology of malignant tumors. Most
these patients (91.9% or 1880) were in the age
acket of 30-70. The average age of these
tients was 55.6. City residents predominated,
ing 85.5% or 1749 of the entire patients. The
cidence of cancer of the nose and cheeks was
ghest (figures not given). Cancer occurred in
covered areas of the skin in 1849 patients or
.4% and in covered areas of the skin in 172
tients or 9.6%. These data indicate a major
le of external factors in skin carcinogenesis.
in cancer occurred in people of all professions.
majority of the skin cancer patients were not
gaged in any professions. Precancerous skin
nditions including warts, moles, acnes, trophic
cers, frost bites, and burns were recorded in
0 patients or 14.5%. Trauma was one of the most
equent (96 cases) precancerous conditions. The
tent period fluctuated from 1.5-2 months to 60
ars, and did not depend on the severity of the
ecancerous injury or diseases. Squamous cell
rcinomas and basal cell carcinomas were found in
out equal numbers. The average incidence of
in cancer for the period 1958-1959 was 12.5/
0,000 residents.

562 THE ETIOLOGY OF LUNG CANCER IN NORTH-EASTERN OKLAHOMA. *(E.)* Asal, N. R.
J. Oklahoma Hlth. Sci. Ctr., Oklahoma City) and P. S.
nderson, Jr. *Health Serv Rep* 87(8):743-748, 1972.

e extremely high lung cancer mortality in northeastern Okalahoma was investigated in a study of smoking habits and occupation of 179 patients from the region. Death and incidence rates for lung cancer were found to be 37.8 and 25.1/100,000 population, resp. Adenocarcinoma was the most common cell-type of lung cancer among women, while squamous cell carcinoma predominated among men. Of 70 patients studied, 69 were smokers. The frequency of lung cancer among persons classified occupationally as "artisians" was high. In this category of occupation, two groups had especially high relative risks for lung cancer: carpenters (relative risk = 3.3) and glass factory workers (relative risk = 4.2). A correlation between smoking and occupations at high risk was noted, and a synergistic interaction between tobacco smoke and certain occupational exposures to carcinogens was suggested.

0563 LONG-TERM MORTALITY STUDY OF STEELWORKERS: VI. MORTALITY FROM MALIGNANT NEOPLASMS AMONG COKE OVEN WORKERS. *(E.)* Redmond, C. K. (Dept. Biostat., U. Pittsburgh, Pa.), A. Ciocco, W. Lloyd and H. W. Rush. *J Occup Med* 14(8):621-629, 1972.

Cancer mortality among coke oven workers in the steel industry in the United States was studied in a survey of 4,661 coke oven workers from seven plants. Men who worked full time at the top of the ovens ("top-side" workers) were more exposed tooven effluents than were men who worked elsewhere some or all of the time. Mortality from respiratory cancer among coke oven workers was greater than 2.5 times its predicted level. Relative risks for cancer of the lung, bronchus and trachea were higher in full-time topside workers (risk = 7.24) than in part-time topside workers (risk = 2.14) or side oven workers (risk = 1.73). Relative risks for respiratory cancer among coke oven workers seemed to be similar for white and nonwhite workers. Genito-urinary system cancers showed high frequencies among coke oven workers with five or more years experience. Location of work area relative to the coke oven tops did not affect risk for genito-urinary cancer. A previously unreported high risk for death from kidney cancer was found among coke oven workers.

0564 ENDEMIC NEPHROPATHY AND CANCER OF THE UPPER TRACT UROTHELIUM IN YUGOSLAVIA. *(E.)* Markovic, B. (Med. Ctr., Gnjilane, Yugoslavia) *Isr J Med Sci* 8(4):540-548, 1972.

The epidemiology of endemic nephropathy (EN) and upper urinary tract cancer in Yugoslavia were investigated. EN and its associated cancer are prevalent in rural communities located along the larger Yugoslavian rivers. In some villages, morbidity from EN reaches 9-16%. Polyps, papillocarcinomas and carcinomas have been found in the renal pelvis, ureters and urinary bladder of 33% of autopsy cases of EN in one study. The etiology of EN and related cancers in Yugoslavia appears to be related to land use. Extensive lumbering operations in the Balkans mountains have stripped the mountains of tree cover. The result has been erosion of the mountain soils and rocks into the rivers of the region. When the rivers flood, silicate particles eroded from the mountains enter public drinking water systems and are ingested. In the blood, these silicates are changed to silicic acid which causes necrotic changes in the renal parenchyma.

0565 CARCINOGENS IN THE NOSE AND THROAT: NASOPHARYNGEAL CARCINOMA IN KENYA.

(E.) Clifford, P. (Royal Marsden Hosp., London, England). *Proc R Soc Med* 65(8):682-686, 1972.

The incidence of nasopharyngeal cancer (NPC) in Kenya is described and environmental factors affecting the occurrence of NPC in the Kenyan population are described. Between 1961-1970 there were 434 NPC cases registered at a center in Nairobi; males dominated NPC cases by 2.65:1 over females. The highest age-specific incidence of NPC occurred in the 50-54-yr-old age group. It was observed that Kenyans in high NPC-incidence areas, especially in the highlands, live in huts and cook over open fires in the huts. They are consequently exposed to soot and wood smoke. Soot samples from Kenyan huts showed the presence of carcinogenic hydrocarbons. Other studies showed a direct correlation between the altitude of huts and the benzo(a)pyrene and benz(a)anthracene contents of the air of huts. Other factors thought to contribute to NPC in Kenya included vitamin A deficiency, urinary estrogen excretion, and the Epstein-Barr virus.

0566 LEUKAEMIA AND ALLIED DISORDERS. 2. EPIDEMIOLOGICAL DATA BASED ON 200 PERSONAL CASES.

(E.) Woodliff, H. J. (Royal Perth Hosp., W. Australia). *Med J Aust* 21(1):17-23, 1972.

The incidence of leukemia and allied diseases in Western Australia was investigated on the basis of a series of 200 cases. There were 34 cases of polycythemia vera, 32 cases of chronic lymphocytic leukemia and 25 cases of acute lymphoblastic or myelomonocytic leukemia. No space clustering of leukemia was seen in the case material. Age distribution showed two peaks, one in childhood due mainly to acute lymphoblastic leukemia, and one in old age due mainly to myeloblastic leukemia.

0567 KAPOSI'S SARCOMA IN UGANDA: GEOGRAPHIC AND ETHNIC DISTRIBUTION.

(E.) Taylor, J. F. (Makerere U. Med. Sch., Kampala, Uganda), P. G. Smith, D. Bull and M. C. Pike. *Brit J Cancer* 26:483-497, 1972.

Incidence patterns of Kaposi's sarcoma in Uganda were studied in a series of 339 patients recorded at Kampala between 1964-1968. The crude annual incidence for Kaposi's sarcoma in Uganda was 7.9 cases/10^6 population for both sexes with a male:female ratio of 13:7. Most patients were aged 35-44 yr. The male incidence rate increased steadily with age from late childhood, while the female incidence increase was less marked. Kaposi's sarcoma was more common in the western highland districts of Uganda than in other districts, and occurred more frequently among indigenous (Bantu) Ugandans than among non-indigenous inhabitants. No cases of Kaposi's sarcoma were seen among European or Indian residents of Uganda. Three patients with Kaposi's sarcoma also had other tumors; these tumors were Hodgkin's disease, lymphatic leukemia and carcinoma of the penis. The geographical distribution of Kaposi's sarcoma in Uganda was different from that of squamous cell carcinoma of the lower legs.

0568 PROBLEMS IN A RETROSPECTIVE EPIDEMIOLOGIC STUDY OF MALIGNANT TUMORS OF THE UPPER RESPIRATORY TRACT.

(Rus.) Balashov, V. A. (D. I. Ulyanov Kuibyshev Med. Inst., Kuibyshev, USSR.). *Zh Ushn Nos Gorl Bolezn* 32(3):19-25, 1972.

An epidemiologic study of malignant tumors of the upper respiratory tract carried out in Kuibyshev, USSR, is presented. From the medico-geographic point of view, Kuibyshev is a highly industrialized area with a high concentration of hazardous substance such as carcinogens. An analysis of the changes in the incidence of malignant tumors of the upper respiratory tract in Kuibyshev residents in recent years revealed fluctuations in types of cancers--laryngeal, nasal cavity and accessory sinuses. Laryngeal tumors showed a high incidence (78.2% in 1964 and 72.6% in 1968). Tumors of the nasal cavity and accessory sinuses amounted to 10.4% in 1964 and 13.7% in 1968. Tumors of the pharynx presented an average incidence of 11.4 and 13.7% in 1964 and 1968, resp. No correlation between geographic distribution and pathogenic factors was found: Pathogenic factors differed in individual patients. The individual factors were analyzed by computer from information supplied on questionnaires. A mathematical model consisting of these factors is given for malignant tumors of the upper respiratory tracts. The possibility of using the mathematical model to work out prophylactic measures is indicated. Consideration of the various aspects in an epidemiologic study proved to be suitable.

0569 CARCINOMA OF THE PENIS AND OF THE CERVIX.

(Dut.) Hemmes, G. D. (Oncol. Center, Utrecht U., Netherlands). *Ned Tydschr Geneeskd* 116(7):258-262, Feb. 1972.

The incidence of cervical carcinoma in women whose husbands suffered from carcinoma of the penis was studied. An epidemiological survey of carcinoma of the cervix and of carcinoma of the penis in

Netherlands during the 1958-1969 period re-
led the respective incidence rates of 1/7,200
ulation and 1/125,000. The average age of
ients with carcinoma of the cervix and carcinoma
the penis at the time of diagnosis was 51.5 yr
64 yr resp. A survey of 98 males with carcinoma
the penis and their wives and former wives (108
ales) revealed only one case of cervical carcino-
among such females, which is about the proportion
ained for the entire population. Thus, the
tribution by a carcinogenic smegma factor to
development of carcinoma of the cervix can be
ed out. Phimosis and pseudophimosis were
cursors of carcinoma of the penis in many cases.

0 THE GEOGRAPHICAL VARIATION OF CASES OF BONE
SARCOMA. *(Sw.)* Lindahl, O. (Orthopedic
gical Clinic, Linkoeping Regional Hosp., Sweden).
artidningen 69(24):2945-2948, 1972.

incidence of bone sarcoma in Sweden's Gaevleborg
nty (293,698 inhabitants) during the 1964-1969
iod was surveyed. The conditions considered for
survey were fibrosarcoma, periosteal sarcoma,
osarcoma, chondrosarcoma, malignant osteoclastoma,
ng's sarcoma, osteogenic sarcoma, not otherwise
cified (NOS) sarcomas, both malignant tumors and
plasms, malignant blastoma, osteolytic tumor and
eoblastic tumor. Two cases of fibrosarcoma, 6
es of chondrosarcoma, 3 cases of Ewing's sarcoma,
ases of osteogenic sarcoma and 1 case of NOS
coma were diagnosed during that period. These
cases correspond to an incidence rate of 8.8 cases/
00,000 population yearly. The bone sarcomas were
nificantly more frequent among males than among
ales. The distribution of the bone malignancies
ng the under 20, 20-40 and over 40 yr age groups
e 8, 4 and 9 cases, resp. Nine cases of the 21
ignant bone tumors were localized in the femur, 6
es in the tibia, 3 cases in the pelvis, 3 cases in
shoulder and 1 case in the rib. While no
nificant differences in the incidence rates between
an and rural populations were observed, the
quency of malignant bone sarcoma in communities
ch had ferrous metallurgy plants and suffered
m marked air pollution was five times the average
the whole country, the difference being
tistically significant. Air pollutants, and
ecially brown smoke from oxygen-blast converter
ration in such communities, are suspected as an
ological factor of malignant bone sarcomas.

71 AUTORADIOGRAPHIC ANALYSIS OF CELL KINETICS
IN HUMAN NORMAL EPIDERMIS AND BASAL CELL
CINOMA. *(E.)* Heenem, M. (Dept. Dermatology,
e U. Brussels, Belgium), G. Achten and P. Galand,
cer Res 33(1):123-127, 1973.

ation of S phase and labeling index were measured
basal cell carcinoma and in normal human epidermis
h an *in vitro* double labeling technique. Evalu-
on of the growth fraction in basal cell carcinoma
was performed after *in vivo* labeling with thymidine-^{3}H. The results indicate that growth fraction in this tumor is approximately 0.8, while indirect evidence suggests a growth fraction near 1 in normal skin. The main difference between the 2 tissues is a much longer S phase in basal cell carcinoma (19.2 hr) than in normal epidermis (10.3 hr), although both tissues exhibited a similar cell cycle time.

0572 INFECTIOUS MONONUCLEOSIS AND THE EMPIRICAL RISK OF CANCER. *(E.)* Miller, R. W. (Natl. Cancer Inst., Bethesda, Md.) and G. W. Beebe. *J Natl Cancer Inst* 50(2):315-321, 1973.

In a prospective study, 2437 U.S. veterans with infectious mononucleosis (IM) diagnosed during military service in World War II were compared with a similar number of controls as to the frequencies of death (1946-65) from leukemia-lymphoma, other cancers, or other causes. No significant differences were observed. There were five deaths from lymphoma *vs* four in the controls and *vs* 2.31 expected on the basis of U.S. national mortality rates. The comparison of five deaths observed *vs* 2.31 expected yielded a relative risk of 2.2 for which the 90% upper and lower confidence limits were 1.1 and 4.0. A retrospective study of 2659 World War II veterans who died of cancer, 1950-54 or 1959-63, and an equal number of matched controls also revealed no significant difference in the frequency with which IM was diagnosed during military service. Thus neither study demonstrated a relationship between clinical IM and cancer in adult males.

0573 FAMILIAL MALIGNANT MELANOMA. *(E.)* Wallace, D. C. (Queensland Inst. Med. Res., Australia), G. L. Beardmore and L. A. Exton. *Ann Surg* 177(1):15-20, 1973.

A study of the pattern of inheritance and of the clinical features and personal characteristics of the patients in 42 kindreds affected by malignant melanoma was carried out using data collected by the Queensland Melanoma Project. The pattern suggested the influence of a polygenic form of inheritance involving the additive contribution of an undetermined number of alleles at separate loci. In addition, the effects of a common environment within families appeared responsible for some of the familial tendency demonstrated. Chance occurrence among near relatives may have caused some families without a greater than normal tendency to develop malignant melanomas to be included in the series. Familial patients tended to be younger than patients with general melanoma; they had multiple primary lesions and were of Scottish or Irish descent. There was no suggestion that a separate form of malignant melanoma with a recognizable pattern of inheritance and separate clinical and histologic features was responsible for the familial instances.

0574 CASE CLUSTERING OF HODGKIN'S DISEASE IN A SMALL RURAL COMMUNITY, WITH ASSOCIATIONS AMONG CASES. *(E.)* Klinger, R. J. (Ohio State U. Hosp., Columbus) and J. P. Minton. *Lancet* (7796):168-173, 1973.

A study of the incidence of Hodgkin's disease in a small rural township in Ohio since 1960 revealed five diagnosed cases, four since 1968--an attack rate equivalent to 71/100,000, as compared with an expected rate of 4.3/100,000. Inquiries among patients, friends, and family members pointed to associations between patients similar to the patterns reported by investigators in Albany, N.Y. The linkings, which implicated eight contacts in addition to the five cases themselves, evolved around two core groups of high-school students. In three of the linked cases, tonsilectomy and adenoidectomy had been done before onset of Hodgkin's disease. This is the first space-time clustering of Hodgkin's disease to be reported.

0575 A COMPARATIVE GEO-PATHOLOGICAL STUDY OF LIVER CIRRHOSIS AND PRIMARY HEPATIC CANCER BETWEEN CAMBRIDGE AND TOKYO. *(E.)* Mori, W. (Tokyo Med. Dent. U., Japan) and M. Shah. *Gann* 63(6):765-771, 1972.

The incidence and interrelation of cirrhosis of the liver and liver cancer were examined and compared in 4,236 autopsy cases from Tokyo and 5,214 from Cambridge, U.K. Primary hepatic cancer incidence was 0.23% of autopsy cases in Cambridge and 2.0% of autopsies in Tokyo. In contrast to this marked difference, there was no remarkable difference between Tokyo and Cambridge in incidence of specific cirrhosis. However, nonspecific cirrhosis was more common in Tokyo than in Cambridge. A significant correlation between cirrhosis and liver cancer was seen only for some nonspecific cirrhosis (especially types A and B) and hepatocellular carcinoma. Cholangiocellular liver carcinoma showed no correlation with any kind of cirrhosis.

0576 EPIDEMIOLOGY OF BENIGN BREAST LESIONS: LACK OF ASSOCIATION WITH ORAL CONTRACEPTIVE USE. *(E.)* Sartwell, P. E. (Dept. Epid., John Hopkins U., Baltimore, Md.), F. G. Arthes and J. A. Tonascia. *N Engl J Med* 288(11):551-554, 1973.

Women with benign breast lesions were compared with matched hospital controls to determine whether the epidemiology of benign breast disease shares epidemiologic features with cancer of the breast. There were 306 patients with cystic disease and related conditions, 71 with fibroadenoma and 39 with miscellaneous other lesions. In the cystic-disease group there was a deficit of postmenopausal women and women whose first pregnancy was under age 25. A history of use of oral contraceptives or estrogens was given no more frequently by patients with cystic disease and fibroadenoma than by their respective controls. Fewer patients with cystic disease (14) than controls (34) had used oral contraceptives for more than one year. These findings imply that use of oral contraceptives or estrogens has no role in the occurrence of benign breast neoplasms. The reproductive histories of patients with benign and malignant breast neoplasms appear similar in some respects.

0577 CELL KINETICS OF AFRICAN CASES OF BURKITT LYMPHOMA. *(E.)* Iversen, U. (Uganda Cancer Inst., Kampala), O. H. Iversen, A. Z. Bluming, J. L. Ziegler and S. Kyalwasi. *Eur J Cancer* 8(3):305-308, 1972.

Small pieces of fresh biopsies of tumor tissue from 17 patients with African Burkitt lymphoma and three patients with other non-Burkitt undifferentiated lymphosarcomas were transferred to tissue culture medium with tritiated thymidine and Colcemid. Specimens were fixed after 1, 2, 3 and 4 hr resp., autoradiographed, and labelling indices determined. In two patients in stage IV, tritiated thymidine was given intravenously every 6 hr for 2 days. Biopsies were obtained 1 hr after the first injection, and then on the first and the second day, and the labelling indices were determined. In the same two patients the diameters of cutaneous tumor nodules were also measured for 3 days. The results show that in the Burkitt tumors analyzed, the mean cell "birth-rate" was 2.71% of all cells per hr, which gives a potential doubling time of 25.5 hr. The actual doubling time was 66 hr. The growth fraction was estimated to be 90-100% of all cells. The rate of cell loss was calculated to be about 70% of the rate of cell renewal. The rate of cellular proliferation in the three lymphomas of non-Burkitt type was 3.78% of all cells per hr, which gives a potential doubling time of 18.3 hr. The rapid rate of cell turn-over in the Burkitt tumor may kinetically explain the rapid response to cytostatic treatment, but it can not explain why Burkitt tumors seem to be more curable than other lymphomas. Immunological mechanisms may be responsible for this phenomenon.

See also:

* (Rev): 0001, 0010, 0014
* (Chem): 0059, 0102, 0182
* (Phys): 0206
* (Immun): 0429

578 DNA POLYMERASES OF MYELOMA: TEMPLATE AND PRIMER SPECIFICITIES OF TWO ENZYMES.
:.) Persico, F. J. (Inst. Microbiol., Rutgers
, New Brunswick, N.J.) and A. A. Gottlieb.
:ture New Biol 239(93):173-176, 1972.

IA polymerases of the murine myeloma tumor were
:parated by chromatography on DEAE-cellulose into
o fractions designated D-I and D-II. Fraction
-I was further separated into two DNA polymerase
:zymes: an enzyme using activated DNA as template
:d an enzyme using poly rA·oligo (dT)$_{12-18}$ as
mplate. The DNA-dependent enzyme exhibited
rtually no activity with poly rA·oligo (dT)$_{12-18}$
template, while the poly rA·oligo (dT)$_{12-18}$-
pendent enzyme did use activated DNA as template.
e poly rA·oligo (dT)$_{12-18}$ directed enzyme showed
preference for oligonucleotide primers. Evidence
s that poly rA·oligo (dT)$_{12-18}$-dependent myeloma
A polymerase was related to viral reverse trans-
iptase, but that it had been modified so as to
ter some of its functions.

79 MUTATION AND CANCER: NEUROBLASTOMA AND PHEOCHROMOCYTOMA. *(E.)* Knudson, A. G.,
. (Grad. Sch. Biomed. Sci., U. Texas, Houston)
d L. C. Strong. *Am J Hum Genet* 24(5):514-532, 1972.

ta on the incidence of neuroblastoma and pheochromo-
toma were examined; age at diagnosis, sex, multi-
icity of tumors and familial occurrence were
ven special attention. Statistical analysis of
ta suggested that risks of neuroblastoma and
eochromocytoma were increased for family members
a fraction of affected individuals. This fraction
s predisposed to either of the conditions by a
minant mutation which was tumor-specific. Pre-
sposed individuals did not develop tumors until
second event, thought also to be a mutation,
curred. In nonhereditary cases of neuroblastoma
pheochromocytoma, both mutational events were
ought to occur in somatic cells, and the
sulting tumors were always solitary. In hereditary
ses, one mutation was thought to occur in germinal
lls and to be inherited, and patients sometimes
veloped multiple tumors.

30 THE MEMBRANE POTENTIAL OF PRIMARY OVARIAN TUMOR CELLS *IN VITRO* AND ITS DEPENDENCE ON
CELL CYCLE. *(E.)* Redmann, K. (Med. Acad.
gdeburg, Germany), V. Müller, St. Tanneberger and
Kalkoff. *Acta Biol Med Germ* 28(5):853-856, 1972.

membrane potentials of primary human ovarian
mor cell cultures were determined by means of
roelectrodes and were related to the cell cycle.
t cells with visible nuclei, which were considered
be interphase cells, had a mean membrane poten-
l of -2.5 mV, a value far lower than those found
several permanent cell lines (-10.5 to -14.2 mV)
for cultured human embryo cells (-8.8 mV) Mitotic
ls (i.e., well delimited round cells loosely adherent to the culture surface) had a mean membrane potential of -6 mV. The potentials of the interphase and mitotic ovarian tumor cells remained constant throughout seven days of culture.

0581 OESTROGEN METABOLISM IN CULTURED HUMAN BREAST TUMOURS. *(E.)* Willcox, P. A. (Dept. Anat., U. Birmingham, England) and G. H. Thomas. *Brit J Cancer* 26(6):453-460, 1972.

Tissues from 25 human mammary tumors were grown in organ culture. Cultures were treated with (2,4,6,7-^{3}H)estradiol-17β or (2,4,5,7-^{3}H)estrone, and the interconversion of estradiol-17β and estrone by tumor cells was observed. All 25 tumors were able to interconvert the two estrogens. Benign tumors differed markedly from scirrhous tumors, both in concentrations of radioactivity taken up by cultured cells, and in ratios of estradiol-17β/estrone achieved in cultures. Among scirrhous tumors, there was no clear correlation between the estradiol-17β/estrone ratios and the morphology of the tumor tissue.

0582 TUMOR VARIATION IN FAMILIES WITH BREAST CANCER. *(E.)* Lynch, H. T. (Creighton U. Sch. Med., Omaha, Nebr.), A. J. Krush, H. M. Lemon, A. R. Kaplan, P. T. Condit and R. H. Bottomley. *J Am Med Assoc* 222(13):1631-1635, 1972.

The occurrence of cancer in different sites was observed in 577 members of 34 white American families; in each family two or more relatives had breast cancer. Adenocarcinoma of the colon was most frequently associated with breast cancer; colon cancer was seen in 12 families with a total of 256 members. Gastric, ovarian and endometrical cancer, and sarcomas, leukemias and brain tumors, were also seen in the breast cancer families.

0583 NUCLEAR DNA SEQUENCES PRESENT IN HUMAN LEUKEMIC CELLS AND ABSENT IN NORMAL LEUKOCYTES. *(E.)* Baxt, W. G. (Coll. Phys. Surg. Columbia Univ., New York, N.Y.) and S. Spiegelman. *Proc Natl Acad Sci USA* 69(12):3737-3741, 1972.

Virus-specific ^{3}H-labeled DNA prepared in an endogenous RNA-directed DNA polymerase reaction was used in DNA-DNA hybridization experiments to probe for virus-specific sequences in the genome of normal and leukemic human lymphocytes. DNA from both normal and leukemic lymphocytes contained virus-specific sequences. When these common DNA sequences were removed by exhaustive annealing with excess normal DNA, the remaining ^{3}H-DNA formed stable duplexes only with leukemic DNA, indicating that the leukemic cells contained virus-specific DNA sequences not found in normal lymphocytes. Evidence indicated that the ^{3}H-DNA sequences shared by normal and leukemic DNA were present in equal numbers in the genomes of leukemic and normal cells. These results are not consistent with the virogene-oncogene theory of carcinogenesis, which postulates the presence of at least one complete copy of oncogenic information in the genome of every normal cell. Rather, they seem to favor the provirus theory which postulates the presence of incomplete virus-specific sequences introduced into the genome upon infection of the cell by oncogenic viruses.

0584 TRYPTOPHAN METABOLISM AND RECURRENCE RATES AFTER MASTECTOMY IN PATIENTS WITH BREAST CANCER.

(E.) Bell, E. D. (Imp. Cancer Res. Fund, London, England), D. Tong, W. I. P. Mainwaring, J. L. Hayward and R. D. Bulbrook. *Clin Chim Acta* 42(1):445-447, 1972.

The relationship between tryptophan metabolism and recurrence rates was examined in 62 premenopausal and 66 menopausal and post-menopausal women with stage 1 or stage 2 primary breast cancer. All women were given a loading dose of 5 g L-tryptophan on the tenth day after mastectomy and the urinary excretion of kynurenine, xanthurenic acid, 3-hydroxy-anthranilic acid and 3-hydroxy-kynurenine was measured in an 8-hr specimen. It was found that premenopausal women who showed a low rate of tryptophan metabolism had a very high tumor recurrence rate (10 of 31) when compared to the recurrence rate in women who had larger amounts of urinary metabolites (2 of 31). No relation between tryptophan metabolizing ability and staging was observed. In menopausal and post-menopausal women, no correlation existed between tryptophan metabolism and recurrence rate.

0585 CHIMERISM IN LYMPHOID CELL CULTURE LINE DERIVED FROM LYMPH NODE OF MARMOSET INFECTED WITH *HERPESVIRUS SAIMIRI*.

(E.) Chu, E. W. (Natl. Cancer Inst., Bethesda, Md.) and A. S. Rabson. *J Natl Cancer Inst* 48(3):771-775, 1972.

Cytogenetic studies were performed on a lymphoid cell line (MLC-1) derived from mesenteric lymph nodes of a marmoset with a lymphoma-like disease induced by infection with *Herpesvirus saimiri*. Three months after start of culture, MLC-1 cultures contained a nearly equal distribution of cells with male and cells with female chromosomes. Ten months after culture initiation, the proportion of male to female cells in MLC-1 cultures remained unchanged, indicating no selective advantage of cells of either sex *in vitro*. The finding of cells with male and female karyotypes in MLC-1 cells eliminates the possibility of a monoclonal origin for this tumor cell line. Since marmosets are usually hematopoietic chimeras and those that are products of a twin pregnancy with a fraternal twin of the opposite sex have both male and female hematopoietic cells, it seemed that the monkey from which the MLC-line was derived was probably such a chimera.

0586 REGENERATION OF THE SURFACE GLYCOPROTEINS OF A TRANSPLANTABLE MOUSE TUMOR CELL AFTER TREATMENT WITH NEURAMINIDASE.

(E.) Hughes, R. C. (Harvard Med. Sch. Boston, Mass.) B. Sanford and R. W. Jeanloz. *Proc Nat Acad Sci USA* 69(4):942-945, 1972.

Ascitic fluid from strain A mice was drawn seven days after mice were inoculated with TA3 tumor cells; TA3 cells were treated with neuraminidase for the removal of at least 80% of total sialic acid present in the cells. Neuraminidase-treated TA3 cells were cultured with D-^{3}H-glucosamine and the regeneration of sialoglycoproteins was observed. Neuraminidase treatment did not affect the capacity of cells to use D-^{3}H-glucosamine and incorporate it into cellular glycoproteins. The rate of incorporation of glucosamine into glycoproteins released from TA3 cell surfaces, which is a measure of membrane glycoprotein synthesis, was found to be approximately linear. The similarity of glucosamine incorporation in neuraminidase-treated and -untreated TA3 cells suggested that these cells were effecting *de novo* synthesis of surface glycoproteins which contained sialic acid; in non-dividing TA3 cells, glycoprotein synthesis was accompanied by a relatively high rate of turnover of membrane glycoproteins. The rate of turnover of membrane glycoproteins that lacked sialic acid residues was the same as that occurring in non-dividing cells not treated with neuraminidase. The rapid synthesis of fully sialyated glycoproteins at the neuraminidase-treated TA3 cell surface made it unlikely that the observed rejection of treated TA3 cells by allogeneic mice was caused by induction of a primary immune response to altered TA3 cells.

0587 ALTERED REGULATION OF THE TRANSPORT OF RNA FROM NUCLEUS TO CYTOPLASM IN RAT HEPATOMA CELLS.

(E.) Shearer, R. W. (Dept. Path., U. Washington, Seattle) and E. A. Smuckler. *Cancer Res* 32(2):339-342, 1972.

RNA-DNA competition-hybridization techniques were used to study RNA transport from nucleus to cytoplasm in three Buffalo rat hepatomas (DB-1, DB-2A and DB-2E), in normal rat liver, in 19-day fetal liver and in adult liver regenerating 2.5 hr after 30% hepatectomy. RNA base sequences that were restricted to the nucleus in normal rat liver were found in the cytoplasm in the hepatomas, which had been induced by continuous feeding 0.06% of 3'-methyl-4-dimethylaminoazobenzene for 4-5 months. At least one-fourth of these RNA species were present in the cytoplasm of fetal and regenerating liver. No RNA's were restricted to the nucleus in tumor cells, suggesting that the mechanism of selective transport of RNA between nucleus and cytoplasm may be lost during malignant transformation. This migration of RNA out of tumor cell nucleus is an early effect of the carcinogen; it was apparent in a significant fraction of liver cells after only six days of azo dye feeding. •

588 RELEVANCE OF THE CYTOGENETIC STATUS IN ACUTE LEUKEMIA IN ADULTS. *(E.)* Gunz, . W. (Sidney Hosp., Australia), B. I. Bach, P. E. rossen, J. E. L. Mellor, S. Singh and P. C. Vincent. *Natl Cancer Inst* 50(1):55-61, 1973.

n 79 adults with acute leukemia, cytogenetic tudies of the blood and marrow were made to assess heir relationship to the patients' clinical con- ition. Of the patients, 30.4% had abnormal cell ines and 69.6%, only diploid ones. In 75% of he patients with abnormal lines, varying propor- ions of diploid cells were also present. Seventy- hree patients were treated subsequent to the nitial studies, and in 63% of them serial (2-21) ytogenetic examinations were done. Remissions ccurred in 35% of the treated patients with bnormal lines and in 48% of those with only diploid ells. The difference in remission rates was not tatistically significant. There was a significant o deletions of G-group chromosomes in the group of atients with cytogenetic abnormalities. However, either this nor any other specific chromosome bnormality had a significant relationship to he likelihood of remission induction, and neither ad the nature of the ploidy (hyperdiploidy, sudodiploidy, or hypodiploidy). Diploid cells ompletely replaced abnormal lines in 6 of 8 ntially aneuploid patients entering remission. ultiple serial specimens confirmed that 100% iploidy persisted throughout the remission eriods, with return of the original abnormal clones n terminal relapse. The frequent finding of ixtures of diploid with aneuploid or pseudodiploid ell populations in untreated patients and of he disappearance of abnormal cells during remission upports the view that improved therapeutic methods imed at the eradication of leukemic cells may ead to better clinical results.

589 TRANSCRIPTION FROM CHROMATINS OF HUMAN LYMPHOCYTIC LEUKEMIA CELLS AND NORMAL YMPHOCYTES. *(E.)* Sawada, H. (M. D. Anderson osp. Tumor Inst., Houston, Tex.), V. H. Gilmore nd G. F. Saunders. *Cancer Res* 33(2):428-434, 973.

hromatins prepared from leukemic lymphosarcoma ell(s), (LS), acute lymphoblastic leukemia cells, hronic lymphocytic leukemia cells (CLL) and ormal tissue culture lymphocytes (NC-37) were tudied with respect to their chemical compositions, hermal denaturation profiles, and template activi- ies. Acute lymphoblastic leukemia and LS chroma- ins had approximately two to four times the onhistone protein and RNA contents of CLL chroma- in. NC-37 chromatin showed increased amounts of onhistone protein and less histone content as ompared with leukemic lymphocytes. LS chromatin ad the highest template activity (10% of purified NA). The template activities of acute lympho- lastic chromatin (8%) and CLL-chromatin (5%) were lso higher than those of NC-37 chromatin (4%). ybridization competition experiments showed that the RNA products synthesized from acute lymphoblastic leukemia chromatin had additional RNA species which were not present in the transcripts of CLL and NC-37 chromatins. In contrast, the RNA products from NC-37 chromatin were similar to those from leukemic lymphocyte chromatins (acute lymphoblastic leukemia cell and CLL). The results also showed that leukemic lymphocyte chromatins, especially from LS and acute lymphoblastic leukemia, had more transcribable repetitive DNA sequences than did NC-37 chromatin. The data suggest that transcription of additional families of repetitive DNA sequences accompanies the transition to cancer in the hematopoietic system.

0590 NUCLEAR AND CYTOPLASMIC DNA SYNTHESIS DURING THE MITOTIC CYCLE OF HeLa CELLS. *(E.)* Volpe, P. (Int. Lab. Genetics Biophysics, Naples, Italy) and T. Eremenko. *Eur J Biochem* 32(2):227-232, 1973.

Experiments were conducted to determine the time of synthesis of nuclear and cytoplasmic DNAs during the mitotic cycle of HeLa S_3 cells synchronized with thymidine and grown in suspension. Nuclear DNA replicated only during the S-phase, but cytoplasmic DNA was synthesized with two peaks in the S- and G_2- phases. The first peak had its maximum 1 - 2 hr before that of nuclear DNA; the second occurred after that of nuclear DNA, approximately 9 hr after the removal of synchronizing thymidine from the culture medium. Newly synthesized nuclear and G_2-phase-cytoplasmic DNAs in CsCl gradients had buoyant densities of 1.698 and 1.688 g/cm^3, resp. The buoyant density of S-phase-cytoplasmic DNA was 1.701 g/cm^3, probably corresponding to the closed circular duplex form of mitochondrial DNA. The kinetics of synthesis of nuclear DNA is exponential, differing from that of S- and G_2-phase-cytoplasmic DNAs which show an asymptotic shape of [^{3}H]thymidine labelling. Ethidium bromide inhibited the synthesis *in vivo* of both S and G_2 peaks of cytoplasmic DNA, but did not appreciably influence the labelling of nuclear DNA.

0591 CYTOGENETIC ANALYSIS OF HUMAN ACUTE AND CHRONIC MYELOID LEUKEMIC CELLS CLONED IN AGAR CULTURE. *(E.)* Moore, M. A. S. (Royal Melbourne Hosp., Australia) and D. Metcalf. *Int J Cancer* 11(1):143-152, 1973.

Karyotypic analysis was performed on agar cultures of blood or bone marrow from 12 patients with acute or chronic myeloid or myelomonocytic leukemia in whom karyotypic markers were present. The granulocytic colonies and clusters which developed on culture were shown to be derived from representative cells of the leukemic population. In two patients with acute leukemia in remission, normal colonies with a normal karyotype were grown from marrow cells but in two patients with chronic myeloid leukemia in remission the Ph^1 abnormality persisted in colony cells. The agar culture technique appears to be ideal for following the emergence and disappearance of leukemic and normal granulopoietic populations in patients with these types of leukemia.

0592 EXPRESSION OF TUMORIGENIC POTENTIAL IN ISOLOGOUS HYBRID CLONES OF CHINESE HAMSTER CELLS. *(Fr.)* Blanchard, M. G. (Inst. Gustave-Roussy, Villejuif, France), G. Barski, B. Leon and D. Hemon. *Int J Cancer* 11(1):178-185, 1973.

Karyotypes and phenotypic characteristics of several clones, developed from a wild population of somatic hybrids obtained by crossing two Chinese Hamster cell lines, were analysed in detail. The crossed cell lines were (1) the DC-3F, sensitive to Actinomycin D (AD) and highly tumorigenic when checked by inoculation into the Syrian Hamster cheek pouch; and (2) the DC-3F/AD/Aza, resistant to AD and nontumorigenic. In the six clonal lines and their three derivatives, obtained secondarily from tumors, the resistance to AD was mostly associated with low or even entirely nonexpressed tumorigenic properties, whereas sensitivity to the toxic action of AD was, as a rule, linked with high malignancy. For all the studied clonal and derived lines a general tendency for chromosomal deletion from the "ideal," complete integration of the two parental karyotypes was observed. This deletion was frequently, but not always, more accentuated following passage through the animal and production of tumors. The relatively stable aspect of the Chinese Hamster karyotype, with most of the chromosomes easily identifiable, permitted a detailed analysis of the frequency of presence or deletion in the eight pairs and groups of autosomes, the X and M1 abnormal marker chromosomes. This analysis showed that, in general, a preferential deletion occurred in chromosomes Number 2, 4, 5, X, and M1. However, no link existed between the frequency of these individualized chromosomal deletions and either the malignancy or the resistance to AD of the hybrid clones.

0593 ENDOCRINE PROFILES AND BREAST CANCER. *(E.)* Argüelles, A. E. (Central Aeronautics Hosp., Buenos Aires, Argentina), U. L. Poggi, C. Saborida, C. Hoffman, M. Chekherdemian and O. Blandard. *Lancet* (7796):165-168, 1973.

Urinary estrone, estradiol, estriol, 17-oxosteroids, dehydroepiandrosterone, and protein-bound iodine-127 and plasma-11-hydroxycorticosteroids levels were studied in 115 women with breast cancer. The patients were divided into four groups (premenopausal or postmenopausal, stages 1/2 or 3/4). The estrogen excretion in the premenopausal patients in stages 1/2 was significantly lower than in the controls. The postmenopausal patients with advanced breast cancer showed a significant elevation of the estrone fraction. Total urinary 17-oxosteroids and dehydroepiandrosterone were below normal in the premenopausal patients, and this correlated fairly well with the low estrogen values. It is suggested that a high estrogen secretion might not be a necessary etiological factor in human breast cancer. Serum-protein-bound-iodine-127 levels were significantly reduced only in the premenopausal patients in stages 3/4, but more than 50% of all the cases had protein-bound-iodine levels below the lower limit of normal.

0594 TUMOR ANGIOGENESIS: IRIS NEOVASCULARIZATION AT A DISTANCE FROM EXPERIMENTAL INTRAOCULAR TUMORS. *(E.)* Gimbrone, M. A., Jr. (Children's Hosp. Med. Ctr., Boston, Mass.), S. B. Leapman, R. S. Cotran and J. Falkman. *J Natl Cancer Inst* 50(1):219-228, 1973.

Neovascularization occurred in the iris of the rabbit eye at a distance from intraocular tumors (Brown-Pearce epithelioma and V-2 carcinoma) implanted in the cornea, the anterior chamber, and the opposite leaf of the iris. Neovascularization was documented by 1) slit-lamp stereomicroscopic observations of the iris vasculature *in vivo*, 2) histologic sections showing increased small-vessel density and endothelial mitoses, 3) autoradiographic studies showing incorporation of ^{3}H-thymidine in endothelial cells, and 4) a distinctive topographic pattern of small-vessel growth in glycerin-cleared, flat-embedded preparations in which the entire microvascular tree of the iris was outlined with colloidal carbon. These observations indicated that cytoplasmic contact between tumor cell and responding vascular bed was not essential for tumor-induced angiogenesis *in vivo*. Neovascularization in this setting appeared to be mediated either directly by a humoral factor from viable tumor cells or through intermediary interaction with host tissues. Further, these studies experimentally correlated the clinical association of iris neovascularization with remote intraocular tumors.

0595 SERUM RIBONUCLEASES IN CANCER: RELATION TO TUMOR HISTOLOGY. *(E.)* Chretien, P. B. (Natl. Cancer Inst., Bethesda, Md.), W. Mathews, Jr. and P. L. Twomey. *Cancer* 31(1):175-179, 1973.

Serum alkaline ribonuclease (SRNase) levels were determined in 49 normal adults and 94 patients with clinically operable malignancies. The mean SRNase levels were higher in 22 patients with adenocarcinomas ($P < .003$) and 28 patients with squamous carcinomas ($P < .01$) than in controls. Within the adenocarcinoma and squamous cell carcinoma groups, mean SRNase levels were similar in patients with primary tumors only and those with metastases. The RNase levels of 21 patients with sarcomas and 23 patients with melanomas, however, did not differ from controls. The increased SRNase levels that occur early in the clinical evolution of adenocarcinomas and squamous carcinomas indicate that this change may have biological significance in the development of these malignancies. The correlation of increased SRNase levels with tumor histology may explain previous conflicting reports concerning abnormal levels of the enzyme in cancer patients.

0596 VARIATIONS IN CARBOHYDRATE METABOLISM-RELATED ENZYME ACTIVITIES OCCURRING IN NEUROBLASTOMA CELLS UNDER PROLIFERATION AND DIFFERENTIATION CONDITIONS. *(Fr.)* Tholey, G. (Natl. Sci. Res. Ctr. Strassbourg, France), J. Cieselski-Treska, B. Wurtz and P. Mandel. *C R Acad Sci* [*D*] *(Paris)* 275(15):1715-1718, 1972.

Aldolase, lactate dehydrogenase (LDH), malate dehydrogenase (MDH) and other carbohydrate metabolism-related enzyme activities were studied in neuroblastoma derived cell cultures. Observations were performed under cell proliferation conditions (Eagle's medium, modified by Dulbecco and enriched with 20% of calf serum) and under cell differentiation conditions (in the absence of calf serum in culture media after four weeks of cultivation under the above conditions). Data were then compared to findings achieved on cell extracts obtained directly from neuroblastomas *in vivo* which were maintained by transplantation in A/J mice. All enzyme activities were increased in the presence of calf serum: the highest activity was found for aldolase (225%). A minor decrease in aldolase, a 71% drop in cytoplasmic LDH, and an increase in mitochondrial MDH by 109% were observed under differentiation conditions after four days of cultivation in serum-deprived media. The minor variations in aldolase activity occurring upon withdrawl of serum from the culture media indicated that no considerable changes in the rate of glucose consumption occur during the differentiation process. But the drop in LDH and increase in MDH indicate that neuronal differentiation is characterized by enhanced Krebs cycle processes. The enhancement of respiration at the enzyme level occurring during the differentiation of neuroblasts is pointed out.

0597 RIBONUCLEOTIDE REDUCTASE OF A MAMMALIAN (RAT) TUMOR (ZAJDELA HEPATOMA): REDUCTION STEREOSPECIFICITY. *(Fr.)* David, S. (Biol. Compound Chem. Lab., Orsay, France) and F. Zajdela. *C R Acad Sci* [*D*] *(Paris)* 275(19):2187-2190, 1972.

Reduction reaction stereospecificity catalyzed by a raw preparation of ribonucleotide reductase extracted from an ascites tumor referred to as Zajdela hepatoma was studied. The original tumor was induced in a rat by chronic administration of dimethylaminoazobenzene; its 950th passage was used in this study. Its growth rate, ascitic evolution, easy i.p. transplantability, and high stability made the tumor comparable to the ascitic variety of Novikoff's hepatoma. The raw enzyme extract containing ribonucleotide reductase catalyzed the reduction of tritiated cytidine monophosphate (CMP) at the 2'C atom to the corresponding deoxycytidine phosphate. The reduction product was subjected to hydrolysis in the presence of alkaline phosphatase. The resulting deoxycytidine was then deaminated to tritiated deoxyuridine. Further breakdown studies provided evidence that rat Zajdela hepatoma ribonucleotide reductase catalyzes a reduction reaction with the preservation of the steric configuration of the substrate. Analogous steric specificity of ribonucleotide reductases was observed, so far, in bacterial RN reductases only.

0598 MAMMARY DUCT PROLIFERATION IN THE ELDERLY. A HISTOPATHOLOGIC STUDY. *(E.)* Kramer, W. M. (Med. Coll. Virginia, Richmond) and B. F. Rush, Jr. *Cancer* 31(1):130-137, 1973.

Both breasts of 70 women over 70 years of age dying from causes other than mammary cancer were studied microscopically at autopsy by multiple step sections. Intense focal intraductual proliferative activity was characteristic of almost half of the breasts of women in this age group. Proliferative intraductal changes tended to be bilateral and were frequently associated with adenosis and intraductal papillomas. Four cases (5.7%) showed intraductal carcinoma, and, in one of these (1.4%), there was a focus of microinvasion. All four cases showed both benign and atypical hyperplastic lesions in other areas of the breast. It is concluded that occult infiltrating breast carcinoma, unlike occult prostatic carcinoma, is an infrequent lesion in the elderly. Intraductal carcinoma, while somewhat more common, is unlikely to eventuate in clinical malignancy within the life span of these patients. Intraductal hyperplasia appears to be a common finding in the elderly and need not be viewed with alarm.

0599 QUANTITATIVE STUDY OF MELANOSOME AND MITOCHONDRIAL POPULATIONS IN PIGMENTED AND AMELANOTIC S-91 MOUSE MELANOMAS. *(E.)* Zara, H. D. (New York U. Med. Ctr., N.Y.) and H. B. Demopoulos. *Cancer Res* 33(1):47-50, 1973.

The relative numbers of mitochondria and melanosomes were enumerated in pigmented and amelanotic S-91 mouse melanomas. There were six times more mitochondria per sq µm of cytoplasm in amelanotic S-91 tumors than in melanotic S-91 tumors. This relative paucity of mitochondria in pigmented S-91 melanomas is significant in view of the identical *in vivo* growth rates of the two tumors and also because the two tumor types are so closely related in terms of common origin and ease of producing one from the other by serial selective transplantation. The respiratory rate of melanotic S-91 melanomas is only about 30% less than that of nonpigmented S-91 tumors. The 6-fold difference in mitochondrial numbers and the ultrastructural similarities of the mitochondria in the two tumor types suggest that the respiratory capacity of the melanotic S-91 melanocytes is attributable in part to melanosomes. These organelles contain high levels of tyrosinase activity, an enzyme that is coupled directly to molecular oxygen. This quantitative study of mitochondria and melanosome populations in these two closely related melanomas is harmonious with the concept that tyrosinase and melanosomes play a vital respiratory role in the relatively mitochondria-poor pigmented S-91 melanomas.

0600 FINE STRUCTURE OF PITUITARY TUMORS AND TRANSPLANTS IN MICE: EFFECTS ON THE PROSTATE. *(E.)* Petrea, I. (Yale U. Sch. Med., New Haven, Conn.) and W. U. Gardner. *Int J Cancer* 11(1):40-47, 1973.

Male and female mice from five inbred and six hybrid strains were inoculated s.c. or intramuscularly with two to four pituitary glands from isologous donors. The transplanted pituitary glands (TPG), primary pituitary tumors (PPT) which arose from them, and subsequent transplants (TPT) of these primaries were studied histologically. The TPG showed central necrosis and diminished secretory activity during the first month. Reappearance of secretory granules coincided with revascularization of the grafts and reappearance of somatotrophic and mammotrophic activity in the females. None of the TPG grew significantly during the first yr, but PPT appeared in 25% (27) of them after 400 days. The PPT contained extensive sinusoidal capillaries, and numerous heterogeneous secretory granules were present. Atypical mitochondria were numerous, the endoplasmic reticulum (ER) was dilated and the membranes were often discontinuous. Virus-like particles were not observed. A total of 79 TPT were examined from 42 to 708 days after transplantation of PPT. They initially grew slowly. By six months, the TPT were polynodular, with red and white nodules both of which had the same ultrastructural characteristics Synthesis of 100-300 Å long, 70 Å diameter fibrils varied inversely with the extent of granular secretory activity of the cells. Sixteen of the TPT in two hybrid strains contained cells with C-type particles in the periphery of the nuclei and in the ER. These cells showed the least granular secretory activity. The TPT were mammotrophic in females and prostatotrophic in males. Hypertrophic prostates contained compact, electron opaque, often laminated structures which were probably secretory granules. C-type particles were present in the prostates of the two virus-positive mouse strains.

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

AARONSON, S.A.
286
ABLASHI, D.V.
255
ABRAHAM, S.
553*
ACHTEN, G.
571
ADAM, M.
530*
ADAMS, R.A.
456*
ADAMSON, R.H.
122*
AHMED, A.K.
17*
AHMED, M.
464*
AKAMATSU, Y.
187*
AL-SARRAF, M.
470*
AL'TSHTEIN, A.D.
219
ALAM, B.S.
36
ALBERT, R.E.
132*, 148*
ALBRIGHT, J.
335*
ALEXANDER, P.
5
ALTANER, C.
223, 225, 275
ALTANEROVA, V.
225
ALTHOFF, J.
31, 89, 121*, 546*
ALVAREZ, J.
523*
ALVAREZ, Y.
523*
AMBROSE, K.R.
500*
AMCHENKOVA, A.M.
304*, 306*
ANDERER, F.A.
231
ANDERSEN, E.
387
ANDERSON, N.G.
500*
ANDERSON, P.
475*
ANDERSON, P.S., JR.
562
ANDZHAPARIDZE, O.G.
235
ANGSUBHAKORN, S.
32
ANSFIELD, F.J.
137*
AOKI, T.
257, 276, 295*
APFFEL, C.A.
360
APPLE, M.A.
346*
ARELLANO, M.G.
560
ARGUELLES, A.E.
593
ARIMORI, M.
277
ARMSTRONG, G.R.
255
ARTHES, F.G.
576
ASAL, N.R.
562
AVILA, L.
316*
BACH, B.I.
588
BACHMANN, A.
372
BADER, A.V.
287
BAEV, I.
209*
BAGSHAWE, K.D.
531*
BAHNA, L.
196*
BAHR, G.F.
229
BALANDIN, I.G.
306*
BALASHOV, V.A.
568
BALDA, B.-R.
313*
BALDWIN, R.W.
378
BALL, C.R.
162*
BALL, J.K.
65, 68
BANDLOW, G.
22*
BANERJEE, D.N.
554*
BANERJEE, M.R.
129*, 538
BANKHURST, A.D.
396
BANNASCH, P.
99, 115
BANSAL, S.C.
379
BARCLAY, T.H.C.
506*
BARRY, D.H.
35
BARRY, E.J.
153*
BARSKI, G.
262, 592
BARTELS, P.H.
229
BARTHOLOMAEUS, W.N.
411
BARTLETT, G.L.
501*
BARTON, B.P.
187*
BARTSCH, H.
172*
BASAVARAJ, H.K.
177*
BASERGA, R.
239, 343*
BASHAM, C.
416
BASILICO, C.
284
BASOMBRIO, M.A.
474*
BASS, L.R.
236, 246
BASSIN, R.H.
250
BAXT, W.G.
583
BEARDMORE, G.L.
573
BEATTIE, E.J., JR.
25
BEATTY, P.A.
199*
BECHET, J.M.
432*
BEDIGIAN, H.G.
267
BEEBE, G.W.
572
BEKTEMIROV, T.A.
305*
BELL, E.D.
584
BELLIVEAU, R.E.
443*
BENDER, E.
315*
BENDICH, A.
510*
BENEDICT, W.F.
96
BENJAMIN, S.A.
212*, 460*
BENOIT, A.
106
BENOVA, D.
209*
BENTON, B.D.A.
120*
BERMAN, L.D.
479*
BERNARD, C.
282
BERNSTEIN, I.D.
508*
BERTRAMS, J.
4
BETTINGER, J.
332*
BETTS, T.E.
59
BEVERLY, P.C.L.
395
BIAS, W.B.
475*
BIBBO, M.
229

BIERMAN, H.R.
556*
BIERWOLF, D.
315*
BILGER, L.
132*
BIRDSALL, N.J.M.
180*
BIRKMAYER, G.D.
313*
BISCHOFF, F.
558*, 559*
BISHOP, J.M.
263, 300*
BISTONI, F.
525*
BLACK, A.
126*
BLACK, H.S.
207
BLACK, M.M.
29, 506*
BLACK, P.J.
210*
BLAIR, H.A.
205
BLAIR, W.H.
177*
BLAKESLEE, J.R., JR.
504*
BLANCHARD, M.G.
592
BLANDARD, O.
593
BLASECKI, J.W.
393
BLAU, H.-J.
418
BLOEMENDAL, H.
308*
BLUMING, A.Z.
577
BLYUMKIN, V.N.
304*
BODEY, G.P., SR.
382, 385
BOETKER, N.K.
119*
BOGER, E.
135*
BOIRON, M.
282
BOLLAG, W.
57
BOLLENGIER, W.E.
44
BORRBERG, H.
25
BORCHERT, P.
171*
BORENFREUND, E.
510*
BORENSTEIN, D.
355
BORSOS, T.
494*
BORTIN, M.M.
457*
BOTTOMLEY, R.H.
466*, 582
BOURALI, M.-F.
261
BOUTWELL, R.K.
55
BOWEN, J.M.
332*
BOYD, R.E.
262
BRACHET, J.
437*
BRADA, Z.
184*
BRANCH, W.R.
352
BRAUN, M.
372
BREDCH, H.
31
BRESNICK, E.
88
BROOKS, A.L.
212*
BROSS, I.D.J.
218*
BROSS, I.J.
206
BROWN, E.R.
69, 141*
BROWN, G.B.
180*
BROWN, J.
6
BROWN, R.D.
142*
BRYAN, G.T.
137*, 145*, 190*
BRYSON, G.
558*, 559*
BUCK, C.A.
230, 279
BUCOVAZ, E.T.
152*
BULBA, S.
184*
BULBROOK, R.D.
584
BULL, D.
567
BURG, C.
391
BURGER, M.M.
273, 284, 441*
BURK, D.
136*
BURKE, P.J.
475*
BURKI, K.
88
BURNS, F.J.
132*, 148*
BUTLER, W.T.
382, 385
BYKOVSKIY, A.F.
241
BYKOVSKY, A.F.
305*
BYRNE, J.C.
265
BYRNE, M.
509*
CAIN, W.A.
459*
CALABRESI, P.
509*
CALCUTT, G.
94
CALVO, W.
302*
CAMARGO, M.E.
264
CAMBELOVA, J.
528*
CANNAT, A.
367, 404
CAPIZZI, R.L.
156*
CARDESA, A.
89, 121*, 546*
CARMODY, J.
17*
CARNES, W.H.
511*
CARRUTHERS, C.
465*
CARUBELLI, R.
459*
CARVAJAL, G.
39
CARVALHO, R.P.S.
264
CASPARY, E.A.
353
CASSAI, E.
221
CASSENS, R.G.
9
CASSETTA, D.
133*
CASSINGENA, R.
261
CASTO, B.C.
289, 329*
CASTRO, E.B.
202
CATALONA, W.J.
406
CATER, D.B.
535*, 536*
CAVALLO, T.
543
CAWLEY, J.
278
CAWLEY, L.P.
402
CEDERQVIST, C.
102
CEJKA, J.
439*, 470*
CERNA, I.
78
CHAN, E.W.
320*
CHANDRA, R.K.
408

CHAPMAN, W.H.
458*
CHARNEY, J.
268
CHARREL, J.
269
CHASSEAUD, L.F.
35
CHAUVEAU, J.
106
CHEKHERDEMIAN, M.
593
CHEN, K.-C.
177*
CHEPULIS, G.-K.S.
270
CHEVALIER, A.
450*
CHEVALIER, H.J.
58
CHEVALIER, H.-J.
111
CHISM, S.E.
451*, 484*
CHO-CHUNG, Y.S.
176*
CHO, H.Y.
248, 364
CHOPRA, D.P.
424*
CHOPRA, H.
345*
CHOPRA, H.C.
348*
CHOUDRY, K.
25
CHRETIEN, P.B.
369, 406, 595
CHU, E.W.
585
CHUAT, J.C.
282
CHURCHILL, W.H.
405
CHUTE, R.N.
213*
CIESELSKI-TRESKA, J.
596
CIKES, M.
377
CIOCCO, A.
563
CLAPP, N.K.
463*
CLARK, D.A.
483*
CLARK, H.F.
301*, 326*
CLARK, J.F.
542
CLARKE, H.D.
548*
CLAYSON, D.B.
105
CLIFFORD, P.
434*, 565
COGGIN, J.H., JR.
447*, 500*
COHEN, A.M.
517*
COHEN, S.M.
137*
COLE, P.
6
COLLER, J.A.
489*
COLNAGHI, M.I.
409
COMBER, P.
60
CONDIT, P.T.
582
CONNEY, A.H.
118*
CONSIGLI, R.A.
311*, 312*
CONSTANS, J.-P.
386
COON, H.G.
195*
COOPER, D.
87, 174*
CORPENING, B.J.
466*
CORREA, P.
122*
COTRAN, R.S.
543, 594
COUNSELL, R.
124*
COX, R.
191*
CRAWFORD, A.M.
28
CRAWFORD, L.V.
251
CREMER, N.
229
CRICHLOW, R.W.
489*
CRISS, W.E.
539, 540
CROCKER, T.T.
150*
CROSSEN, P.E.
588
CUDKOWICZ, G.
245, 476*
CUMMINGS, F.J.
509*
CURRIE, A.R.
28
CURRIE, G.A.
416
CUTLER, S.J.
506*
DALDORF, G.
264
DALGARD, D.W.
122*
DANIEL, B.G.
54, 159*
DANNA, K.J.
249
DAO, T.L.
87, 174*
DADUST, R.
555*
DAPKUS, D.C.
344*
DAVID, S.
597
DAVIDSOHN, I.
403, 486*
DAVIDSON, J.K.
62
DAVIES, R.E.
181*
DAVIS, R.C.
394
DAWSON, P.J.
322*
DE GROOT, F.G.
297*
DECKER, D.G.
38
DECKERS, P.J.
394
DECOSSE, J.J.
48, 495*
DEFENDI, V.
230, 239, 343*
DEINHARDT, F.
256, 454*
DELIMBETOVA, G.A.
304*
DELLA PORTA, G.
409
DELWICHE, R.
381
DEMISSIE, A.
423*
DEMOPOULOS, H.B.
599
DEMPO, K.
544
DENISOV, V.V.
200*
DENT, P.B.
258
DEROUEN, T.A.
36
DESHIELDS, L.H.
181*
DEUTSCH, E.
360
DEWHURST, F.
94
DEWYS, W.
496*
DHAR, P.
537*
DI LUZIO, N.R.
373
DI MAYORCA, G.
110
DICKINSON, J.P.
353
DIERLAM, P.
447*
DIETZ, W.
41
DILUZIO, N.R.
371

DIPAOLO, J.A.
329*
DIPPLE, A.
27, 146*
DIRINGER, H.
224, 231, 232
DMOCHOWSKI, L.
260, 332*
DOAK, R.L.
512*
DODGE, H.A.
181*
DOLIN, R.
291*
DONNER, M.
391
DONTENWILL, W.
58, 111
DORFMAN, R.F.
14*
DORSETT, B.
502*
DOUGHERTY, R.M.
412
DOUGLAS, D.R.
207
DROSDOWSKY, M.A.
79
DUCHESNE, J.
81
DUFF, R.
310*, 318*, 321*
DUNN, C.D.R.
46
DUNN, J.E., JR.
560
DUPUIS, C.
74
DVORAK, K.
495*
EATON, H.
198*
EBBESEN, P.
387
ECKHART, W.
410
ECKNER, R.J.
478*
EDSTROM, R.D.
490*
EDYNAK, E.M.
467*
EGGERS, A.E.
370
EIJSVOOGEL, V.P.
430*
EINHORN, N.
434*
EISENFELD, A.J.
44
EKELUND, L.
117*
ELDER, E.
248
ELIASSON, R.
272
ELLIOTT, J.
151*
ELLIS, V.L.
344*
ELLISON, R.R.
448*
ELSON, L.A.
59
ELVEBACK, L.R.
47, 429*
EMBLETON, M.J.
378
EMMINGER, A.
31
EREMENKO, T.
590
ERHARD, P.
288
ESSEX, M.
454*
ESTES, P.C.
375
EVANS, A.S.
264
EVANS, D.L.
426*
EVANS, R.
422*
EVRIN, P.-E.
435*
EXTON, L.A.
573
FAHMY, M.J.
70
FAHMY, O.G.
70
FALK, H.L.
194*
FALK, L.
256
FALKMAN, J.
594
FARAS, A.
300*
FARAS, A.J.
263
FARBER, E.
191*
FARRELLY, J.G.
323*
FASS, L.
342*
FAVRE, R.
303*
FEBVRE, H.
386
FEDER, S.
420
FEFER, A.
341*, 342*
FELLER, W.F.
16*
FENYO, E.M.
407
FEORINO, P.M.
314*
FERRER, J.F.
316*
FETHIERE, W.
547*
FIALKOW, P.J.
431*
FICKER, S.
498*
FIDLER, I.J.
520*
FIELD, E.J.
353
FIELDSTEEL, A.H.
322*
FILATOV, F.A.
305*
FILATOV, F.P.
304*
FINE, D.L.
338*
FINE, G.
548*
FINKEL, M.P.
325*
FINKELSTEIN, S.
429*
FINN, R.
442*
FIRMINGER, H.L.
108
FISCHGRUND, T.
417
FISCHINGER, P.J.
233, 250
FISH, S.A.
152*
FISHER, B.
444*
FISHER, E.R.
444*
FISHER, W.D.
125*
FLAKS, A.
105
FLAXMAN, B.A.
424*
FLEISCHMAN, J.B.
358
FLESHER, J.W.
147*
FOLKMAN, J.
543
FORMAN, J.
374
FORTEZA-VILA, J.
302*
FOX, M.
43
FRANKE, R.
84
FRANKEL, K.A.
204
FRANKIE, G.
199*
FRASER, L.B.
535*
FREARSON, P.M.
251
FREDRICKSON, T.N.
348*
FREEDMAN, S.O.
420, 428*

FREI, J.V.
26
FREIREICH, E.J.
382, 385
FRENKEL, K.
192*
FRENKEL, N.
221
FRIBERG, S., JR.
377
FRIED, A.H.
222
FRIEDMAN, M.
346*
FRIEDMAN, M.M.
307*
FRIEND, C.
245, 330*
FROST, P.
264
FUDENBERG, H.H.
455*
FUHRER, J.P.
279
FUJIMOTO, J.
52
FUJIMURA, S.
39
FUKUDA, S.
130*
FUKUTO, T.R.
126*
FURST, A.
133*
FURTH, J.
493*
FUSKA, M.
155*
FUTRELL, J.W.
380, 533*
GABELMAN, N.
283
GALAND, P.
571
GALLAGHER, C.H.
173*
GALLI, S.J.
456*
GALLO, R.C.
349*
GANDINI-ATTARDI, D.
358
GANTT, R.R.
339*
GARDNER, D.
148*
GARDNER, W.U.
44, 347*, 600
GARLAND, M.R.
123*
GARON, C.
257
GASSER, D.L.
417
GATELY, M.K.
355
GATES, O.
213*
GAZDAR, A.
345*
GELBOIN, H.V.
195*
GENTILE, R.
333*
GERBER, P.
238
GEVAUDAN, M.J.
269
GEVAUDAN, P.
269
GIBBARD, S.
90
GIBBS, W.N.
530*
GIELEN, J.E.
96
GIELKENS, A.L.J.
308*
GIESELER, A.
91
GILCHRIST, J.C.
199*
GILDEN, R.V.
364
GILMORE, V.H.
589
GIMBRONE, M.A., JR.
594
GIORDANO, R.
110
GIOVANELLA, B.C.
485*
GIRARD, M.
254
GIRARDI, A.J.
447*
GLASER, R.
321*
GLICK, M.C.
230
GLINSKA, H.
526*
GLYNN, J.P.
499*
GOLD, P.
401, 420
GOLDBLATT, P.J.
77
GOLDENBERG, D.M.
482*
GOLDFEDER, A.
216*
GOLDSCHMIDT, B.M.
192*
GOLDWATER, D.J.
462*
GOODMAN, H.M.
263
GOODWIN, W.L.
402
GOSSNER, W.
208
GOTHLIN, J.
117*
GOTTLIEB, A.A.
578
GOZ, B.
337*
GRABAR, P.
7
GRAFF, K.S.
491*
GRAFFI, A.
315*
GRAFFI, I.
315*
GRANDJEAN, C.
173*
GRASSO, P.
60
GRAZIANO, K.D.
477*
GREENBLATT, M.
49, 110
GREENSPAN, I.
69
GREY, H.M.
419
GRIMES, W.J.
299*
GRIMMER, G.
201*
GROFOVA, M.
220
GROSE, C.
314*
GROSS, L.
427*
GROSS, P.R.
293*
GROVER, P.L.
188*
GRUENSTEIN, M.
185*
GRUNBERGER, D.
39
GRUNDNER, G.
407
GULLINO, P.M.
176*
GUNZ, F.W.
588
GURTOO, H.L.
104
GUTMANN, H.R.
159*, 183*
GUTTERMAN, J.U.
382, 385, 481*
HACKER, B.
551*
HACKETT, A.J.
234
HACKETT, R.L.
169*
HADLER, H.I.
54, 159*
HALBERG, P.
387
HALL, T.C.
553*
HALTERMAN, R.
421
HALTERMAN, R.H.
519*

HAMILTON, J.M.
105
HANAFUSA, H.
280
HANNA, M.G., JR.
463*
HANSEN, H.J.
482*
HARAN-GHERA, N.
217*
HARDY, W.G.
215*
HARKE, H.-P.
111
HARLOZINSKA, A.
363
HARRIS, A.W.
396
HARRIS, C.C.
160*
HARRIS, D.
42
HARRIS, H.
361
HART, M.L.
511*
HARTMANN, J.F.
230
HASKELL, C.
346*
HASSON, J.
30
HATANO, M.
453*
HATTORI, K.
357
HAY, D.
242
HAYS, E.F.
259
HAYWARD, J.L.
584
HAYWARD, W.S.
280
HAZDRA, J.
141*
HAZDRA, J.J.
69
HECKER, E.
13
HEDRI, A.
21*
HEENEM, M.
571
HEIDELBERGER, C.
154*
HELLMAN, S.
440*, 451*, 484*
HELLSTROM, I.
433*, 458*
HELLSTROM, K.E.
433*, 458*
HEMMES, G.D.
569
HEMON, D.
592
HENDERSON, B.
480*
HENDERSON, B.E.
120*
HENLE, G.
252, 434*
HENLE, W.
252, 434*
HENNIG, W.
86
HENRIKSON, H.
117*
HEPPNER, G.
509*
HERBERMAN, R.B.
276, 295*, 327*, 507*
HERSH, E.
469*
HERSH, E.M.
382, 385, 481*
HESSE, J.
387
HEWETSON, J.
252
HIASA, Y.
163*
HIGGINS, P.J.
510*
HILDEBRANDT, A.
201*
HINDRINGER, B.
208
HIRANO, A.
30
HIRSCHHORN, K.
283
HIRSHAUT, Y.
515*
HLADKA, M.
223
HLAVAYOVA, E.
223, 275
HOFER, K.G.
374
HOFFMAN, C.
593
HOFFMAN, J.
61, 182*
HOLADAY, W.J.
204
HOLDEN, H.T.
471*
HOLMAN, L.
53
HOLMES, E.C.
384
HOLYCKE, E.D.
112
HONG, K.C.
155*
HOOD, J.E.
556*
HOOVER, E.A.
247
HOPP, M.
168*
HOPP, M.L.
63
HORN, R.C., JR.
548*
HOROSZEWICZ, J.S.
351*
HORTON, B.J.
103
HORTON, J.D.
103
HOSSFELD, D.K.
11
HSIUNG, G.D.
290*
HUDSON, P.M.
194*
HUEBNER, R.J.
157*, 364
HUGHES, R.C.
586
HULIN, N.
437*
HUMPHREY, R.
475*
HUNT, P.S.
112
HUNTER, B.
35
HURT, T.
338*
HYMAN, R.
383
ICHINOSE, H.
371
IKAWA, Y.
348*
IKUTA, F.
237
IL'IN, K.V.
241
IMASATO, K.
414
INGRAM, L.O.
125*
INOUE, M.
452*
IOACHIM, H.L.
502*
IORIO, A.
525*
IRVING, C.C.
143*
ISAKA, H.
544
ISKANDEROVA, V.R.
522*
ITO, K.
438*
ITO, N.
128*, 163*
ITZE, L.
71
IVERSEN, O.H.
577
IVERSEN, U.
577
JACKSON, F.E.
160*
JACKSON, J.
300*
JACKSON, J.P.
371

JACOB, J. 201*
JACOBI, J. 73
JACOBS, A.A. 425*
JACOBSON, M. 118*
JAEGERMAN, K. 526*
JAMRA, M. 264
JANES, J.M. 47
JANSON, V.K. 441*
JANSS, D.H. 143*
JARAMILLO, F.E. 36
JARRETT, O. 242
JEANLOZ, R.W. 586
JENSEN, B.L. 124*
JESSUP, J.M. 461*
JOEL, W. 466*
JOHNS, L. 266
JOHNSON, P.A. 276
JOHNSON, R.O. 145*
JONDORF, W.R. 78
JONES, E.L. 56, 92
JOSEPH, D. 323*
JOUBERT, F. 79
JULIAN, B. 339*
JURGELSKI, W. 194*
KAISER, N. 75
KALDERON, A.E. 547*
KALISS, N. 355
KALKOFF, W. 580
KAMAMOTO, Y. 163*
KAMATA, S. 113
KANEKO, A. 544
KANO, K. 397
KAPLAN, A.R. 582
KARPAS, A. 278
KARUSH, F. 399
KATSUTA, H. 33
KAUFMAN, D.G. 67
KAWASAKI, H. 414
KEAST, D. 411
KEITH, L. 69, 141*
KELLEN, J.A. 116*
KELLER, S. 502*
KENDE, M. 499*
KENNEDY, B.J. 490*
KENNEY, F.T. 323*, 334*
KERR, J.F.R. 28
KETCHAM, A.S. 369, 517*
KETCHEL, M.M. 374
KEYS, L. 499*
KHOURY, G. 265
KIANG, D.T. 490*
KIM, U. 465*
KIMOTO, E. 414
KINDER, D. 129*
KING, C.M. 144*
KINGSBURY, E.W. 62
KINOSHITA, N. 175*
KIRSTEN, W.H. 296*, 331*
KISELEVA, L.G. 270
KITAJIMA, T. 63, 168*
KITCHEN, D.A. 94
KITHIER, K. 439*, 470*
KIZER, D.E. 53
KLEIN, D. 475*
KLEIN, E. 431*
KLEIN, G. 361, 377, 407, 431*, 434*, 454*
KLIMISH, H.-J. 111
KLINGER, R.J. 574
KLOUDA, P.T. 531*
KLUBES, P. 78
KNIGHT, P. 310*
KNUDSON, A.G., JR. 579
KOCH, M.A. 224, 231, 232
KOCIBA, G.J. 247
KOLAR, G.F. 107
KOLAROVA, M. 529*
KOLLMORGEN, G.M. 459*
KOLODNY, G.M. 293*
KOLTSOV, V.D. 306*
KOMP, D.M. 298*
KONDRAT'YEVA, V.V. 24
KONINGS, R.N.H. 308*
KONISHI, Y. 163*
KORB, J. 281
KOSHEL, I.V. 522*
KOSUDA, L. 351*
KOTIN, P. 194*
KRAMARSKY, B. 268
KRAMCHANINOV, N.F. 561
KRAMER, W.M. 598
KRATKY, V. 155*
KRAUSS, S. 488*
KREIDER, J.W. 460*
KRIEGER, R.I. 126*
KRISHAN, A. 319*
KROES, R. 149*
KRUGER, F.W. 86, 98
KRUGER, G. 42
KRUSH, A.J. 582
KRYMSKAYA, L.L. 306*
KU, M.-M. 399
KULAS, H.-P.

224
KUMANISHI, T.
237
KUNTZMAN, R.
118*
KUNZ, W.
86
KUNZE, E.
97
KUPCHIK, H.Z.
537*
KURLAND, L.T.
38, 429*
KURLYANDSKIY, B.A.
197*
KUWAHARA, A.
50
KUWERT, E.
4
KWAPINSKI, J.B.G.
69, 141*, 356
KYALWASI, S.
577
KYLE, R.A.
429*
KYRIAZIS, A.P.
72
LACROIX, M.
81
LAFRENZ, U.
111
LAFUMA, J.
203
LAGEMAN, A.
41
LAIRD, H.M.
242
LAMON, E.
407
LANDON, J.C.
338*
LANIER, A.P.
38
LAPREVOTTE, I.
282
LARDIS, M.P.
467*
LARSEN, V.
102
LAU, T.
169*
LAUSCH, R.
518*
LAVRIN, D.
327*
LAVRIN, D.H.
507*
LAWLER, S.D.
531*
LAWRENCE, M.
335*
LEAPMAN, S.B.
594
LEBWOHL, D.J.
456*
LECOU, C.
527*
LEDINKO, N.
309*
LEE, D.M.
145*
LEE, P.W.
126*
LEE, R.
356
LEE, T.C.
180*
LEIS, H.P., JR.
29
LEMON, H.M.
165*, 582
LENGEL, C.R.
364
LENNETTE, E.H.
229
LEON, B.
592
LEONARD, E.J.
51, 494*
LEONG, S.S.
351*
LEPAGE, G.A.
8
LETHCO, E.J.
167*
LEUCHTENBERGER, C.
161*
LEUCHTENBERGER, R.
161*
LEVENBUK, I.S.
219
LEVENTHAL, B.G.
421, 519*
LEVIN, W.
118*
LEVINE, A.J.
273
LEVINE, A.S.
243
LEVINE, P.H.
298*, 387
LEVINSON, W.
300*
LEVINSON, W.E.
263
LEVY, R.
498*
LEWIS, A.M., JR.
243
LIEBELT, A.G.
88, 550*
LIEBELT, R.A.
550*
LIEBERMAN, M.W.
77, 146*
LIJINSKY, W.
158*, 189*
LILL, P.
403, 486*
LINA, P.H.C.
297*
LINDAHL, O.
570
LINDEN, G.
120*, 560
LIPMAN, M.
285
LITTMAN, B.
487*
LITWACK, M.
339*
LIU, M.
276
LLOYD, H.M.
73
LLOYD, W.
563
LOISILLIER, F.
400
LOMBARD, L.S.
139*, 140*
LOTLIKAR, P.D.
186*
LOTZOVA, E.
476*
LOVIG, C.A.
466*
LOWENFELS, A.B.
18*
LOWENTHAL, R.M.
395
LOWER, G.M., JR.
190*
LU, A.Y.H.
118*
LUCAS, S.J.
238
LUNGER, F.D.
301*
LUSHNIKOV, A.A.
306*
LUZ, A.
208
LYNCH, H.T.
582
MACDONALD, E.J.
1
MACDONALD, J.L.
473*
MACLEOD, D.F.
17*
MACMAHON, B.
6
MADDEN, R.E.
461*
MADISON, R.M.
62
MAGNUSSON, G.
271, 272
MAGOUS, R.
527*
MAINWARING, W.I.P.
584
MAKIURA, S.
163*
MALAVE, I.
397
MALEJKA-GIGANTI, D.
183*
MALONEY, B.D.
199*
MALONEY, T.
328*
MALONEY, W.C.

405
MANDEL, M.A.
48, 495*
MANDEL, P.
596
MANN, D.L.
421, 472*
MANNICK, J.A.
394
MANOJLOVSKI, B.
71
MANSKY, J.
496*
MANTEUIL, S.
254
MARCHEGIANI, M.
333*
MARCON, N.
381
MARCONI, P.
525*
MARCUCCI, L.
333*
MARCZYNSKA, B.
256
MARDINEY, M.R., JR.
468*
MARGGRAF, W.-D.
224, 231
MARKOE, A.M.
288
MARKOVIC, B.
564
MARTIN, M.A.
265
MARTIN, N.
521*
MARTIN, W.J.
473*
MARUGAMI, M.
128*
MASERA, P.
437*
MASHBITS, F.D.
197*
MASON, S.
396
MASSE, R.
203
MATHEWS, W., JR.
595
MATTISON, M.L.
442*
MATUSEVICH, L.L.
235
MAVLIGIT, G.
469*
MCBRIDE, C.
469*
MCCALL, J.T.
47
MCCLAIN, K.
331*
MCCLELLAN, R.O.
212*
MCCREDIE, K.B.
382, 385, 481*
MCDERMOTT, B.J.
551*
MCDONOUGH, E.
509*
MCGARRY, M.P.
324*
MCKINNELL, R.G.
344*
MCNAMEE, R.
373
MEADE, H.M.
492*
MEARES, J.D.
73
MEDVEDOVSKIY, A.G.
197*
MEGA, T.
128*
MEHTA, R.G.
538
MELIEF, C.J.M.
430*
MELLOR, J.E.L.
588
MELTZER, M.S.
51, 494*
MERRILL, C.L.
497*
METCALF, D.
15*, 591
METIVIER, H.
203
MEYER, G.
303*
MEYERS, P.
412, 471*
MICKEY, M.R.
480*
MIHAILOVICH, N.
71, 139*
MILGRAM, F.
397
MILHOLLAND, R.J.
75
MILLER-CRANKO, J.A.W.
307*
MILLER, D.G.
516*
MILLER, E.C.
171*, 172*
MILLER, E.F.
373
MILLER, F.
313*
MILLER, G.
285
MILLER, J.A.
171*, 172*
MILLER, R.C.
517*
MILLER, R.W.
572
MILO, G.E.
317*
MINARD, B.J.
402
MINTON, J.P.
574
MIRAND, E.A.
324*
MIROFF, G.
492*
MIRVISH, S.S.
49
MITCHELL, M.S.
462*
MITCHLEY, B.C.V.
27
MIYAKI, K.
83
MIYAMOTO, M.
32
MIZUNO, F.
362
MOHR, U.
31, 89, 121*
MOKYR, M.B.
462*
MOLNAR, H.
464*
MONTES DE OCA, F.
184*
MONTESANO, R.
67
MONTGOMERY, P.C.
520*
MONTGOMERY, P.O'B.
179*
MOORE, C.
127*
MOORE, D.H.
268
MOORE, M.A.S.
591
MOORE, T.
537*
MORAIS, R.
74
MORGAN, O.
530*
MORI, H.
360
MORI, M.
452*
MORI, W.
575
MORRIS, H.P.
542
MORRISON, J.C.
152*
MORRISON, W.C.
152*
MORTON, D.L.
384
MORTON, J.I.
366, 445*
MOSORA, F.
81
MOULIN, M.-C.
555*
MOUSSERON-CANET, M.
527*
MOUTTE, A.
386
MOY, P.
493*
MUKERJEE, D.

332*
MUKHERJI, B.
515*
MULLER, V.
580
MULLINS, G.
475*
MUMFORD, D.M.
485*
MUNTZING, J.
549*
MURAD, T.M.
95
MURAGISHI, H.
466*
MYERS, G.H.
533*
MYERS, G.H., JR.
380
NAGASAKI, H.
128*
NAGASAWA, H.
76
NAHAS, A.
553*
NAHMIAS, A.
221
NAMBA, M.
33
NATARAJAN, N.
206, 218*
NATHANS, D.
249
NATHANSON, L.
483*
NEBERT, D.W.
96
NECHAUD, B. DE
389
NELSON-REES, W.A.
157*
NEMIROVSKY, T.
390
NETTESHEIN, P.
158*
NEWBERNE, P.M.
66
NEWBERRY, B.H.
199*
NIELSEN, J.
102
NILSSON, K.
432*
NILSSON, T.
549*
NILSUWARN, N.
32
NISHIMOTO, A.
277
NOLAN, J.F.
120*
NOLIBE, D.
203
NOLLER, K.L.
38
NOMURA, S.
250
NOMURA, T.
114
NOONAN, K.D.
284
NORRIS, H.J.
403
NOVOTNA, L.
228
NOYES, W.F.
131*
NUNN, M.
327*
NUNN, M.E.
295*
NUTTER, R.L.
292*
O'CONNOR,D.M.
298*
O'CONNOR, T.E.
320*
O'CONOR, G.T.
298*
O'GRADY, J.F.
185*
O'NEILL, F.J.
350*
ODA, T.
240, 415
OETTGEN, H.F.
25
OGURA, H.
240
OHASHI, T.
277
OKAMOTO, E.
114
OKANO, H.
266
OKANO, T.
101
OKUNEWICK, J.P.
288
OLIVER, J.
26
ONDRUS, B.
528*
ONOE, T.
544
ORAVEC, C.
528*
ORDER, S.E.
440*, 451*, 484*
ORTEGO, H.J.
348*
OSATO, T.
362
OSOFSKY, L.
346*
OTH, D.
391
OTSUKA, H.
50
OTTEN, J.A.
375, 503*
OTTONEN, P.O.
65, 68
OXMAN, M.N.
410
OYASU, R.
63, 168*
PACE, J.L.
512*
PAGANELLI, J.
346*
PAGES, J.
254
PANEM, S.
296*
PAPENBURG, J.
115
PAPIRMEISTER, B.
156*
PARHAM, J.C.
180*
PARKER, G.A.
394
PATCH, C.T.
243
PATTERSON, L.T.
426*
PAUL, B.B.
425*
PAUL, J.S.
179*
PAVLOVSKY, A.
372
PEACOCK, A.C.
175*
PEARSON, G.
246
PEARSON, G.R.
236
PEARSON, L.
454*
PEEBLES, P.
345*
PELED, A.
217*
PENN, I.
376
PERDUE, J.F.
340*
PERLMUTTER, R.
112
PERRYMAN, L.E.
247
PERSAUD, C.
530*
PERSICO, F.J.
578
PETERSEN, K.
86
PETREA, I.
347*, 600
PETTENGILL, O.S.
358
PETUKHOVA, M.B.
304*
PETYAEV, M.M.
200*
PHILLIPS, B.
144*
PHILLIPS, E.
288
PIECZYNSKI, W.J.
329*
PIENTA, R.J.

338*
PIERONI, G.
269
PIESSENS, W.F.
405
PIKE, M.C.
567
PILERI, A.
437*
PINKERTON, H.
37
PINKERTON, P.H.
545*
PISANO, J.C.
371, 373
PITZURRA, M.
525*
PIZZAIA, L.M.
467*
PODANY, V.
196*
POGGI, U.L.
593
POIRIER, L.A.
74, 100
POLLACK, S.
388
POLLEY, M.J.
419
POMEROY, T.C.
215*
PONTEN, J.
432*
POST, J.
61, 182*
POTTER, V.R.
178*
POUND, A.W.
93
POUR, P.
89, 121*, 546*
POVLSEN, C.O.
431*
PRAGER, M.D.
497*
PREISLER, H.D.
330*
PRENDERGAST, R.A.
477*
PRESSMAN, D.
448*, 449*
PRICE, P.M.
283
PRITCHARD, D.J.
325*
QUAN, S.H.Q.
202
RABELLINO, E.M.
419
RABINOWITZ, A.D.
78
RABSON, A.S.
291*, 585
RABSTEIN, L.S.
34
RAGLAND, W.L.
512*
RAHA, C.R.
173*
RAICK, A.N.
134*
RAINERI, R.
55
RAMADAN, M.A.
91
RAMM, G.M.
80
RAO, A.R.
142*
RAO, K.V.N.
71
RAPP, F.
292*, 310*, 318*, 321*,
518*
RAPP, H.J.
487*, 501*
RASMUSSEN, R.E.
150*
RECKZEH, G.
58, 111
REDMANN, K.
580
REDMON, L.W.
236
REDMOND, C.K.
563
REES, J.A.
392
REICHARD, P.
271, 272
REICHLE, F.A.
185*
REICHLE, R.M.
185*
REIDBORD, H.E.
444*
REILLY, C.A., JR.
325*
REISFELD, R.A.
384
REISHER, J.I.
387
RENGER, H.C.
274, 284
REPPUCCI, P.
447*
REYNOLDS, R.K.
286
RHAESE, H.-J.
119*
RHIM, J.S.
157*, 248, 364
RICE, J.M.
62
RICH, R.
266
RICHARDS, J.F.
123*
RICHARDSON, H.I.
167*
RICHARDSON, M.E.
167*
RICHTER, C.
158*
RICHTER, R.
363
RICK, M.A.
266
RIMAN, J.
281
RIMM, A.A.
457*
RINTALA, A.
211*
RIVAT, C.
524*
ROBBINS, P.W.
294*
ROBERSON, L.
323*
ROBINS, R.A.
378
ROBINSON, W.E.
35
ROCCHI, G.
252
RODEY, G.E.
457*
ROE, F.J.C.
27
ROEDER, P.E.
521*
ROGENTINE, G.N.
491*
ROGENTINE, G.N., JR.
413
ROGERS, A.E.
66
ROGERS, S.
336*
ROIZMAN, B.
221
ROLLINGHOFF, M.
365
ROMEN, W.
99
ROPARTZ, C.
524*
ROSEMOND, G.P.
185*
ROSEN, F.
75
ROSEN, P.P.
202
ROSENBERG, S.A.
413, 498*
ROSS, G.D.
419
ROSS, S.
518*
ROSS, W.
99, 115
ROSSEN, R.D.
382, 385
ROSSI, G.B.
245, 333*
ROSSIER, E.
446*
ROUSE, B.T.
365, 541
ROVERA, G.
239, 343*
RUBIO, N.
400

RUDBERG, K.D.
467*
RUSH, B.F., JR.
598
RUSH, H.W.
563
RUTALA, W.
447*
RYAN, D.
118*
RYGAARD, J.
431*
SABBATH, M.
502*
SABINE, J.R.
103
SABORIDA, C.
593
SACCOMANNO, G.
557*
SADE, R.
543
SAFFIOTTI, U.
67, 160*
SAITO, Y.
534*
SAKALOVA, A.
528*
SAKAMOTO, C.K.
441*
SAKIYAMA, H.
294*
SALDEN, M.H.L.
308*
SALERNO, R.A.
34, 80
SALOMON, R.
175*
SALTZSTEIN, E.C.
157*
SALZMAN, N.P.
244
SAMPLE, W.F.
406
SANDER, J.
85
SANDLER, S.G.
298*
SANFORD, B.
586
SANFORD, B.H.
398
SANTAMARIA, C.S.
485*
SANTOS, G.W.
475*, 477*
SAPRIN, A.N.
24
SARKAR, N.H.
268
SARMA, P.
345*
SARTWELL, P.E.
576
SASSY, C.
450*
SATO, J.
33
SATO, Y.
101
SAUNDERS, G.F.
589
SAWADA, H.
589
SBARRA, A.J.
425*
SCANLON, M.D.
332*
SCHAFER, W.
233
SCHALLER, J.P.
317*
SCHAUER, A.
97
SCHECHTER, B.
498*
SCHEITHAUER, E.M.
155*
SCHER, W.
330*
SCHEURLEN, P.G.
12
SCHIDLOVSKY, G.
464*
SCHIOP-STANSLY, P.E.
320*
SCHLEGEL, J.U.
36
SCHMAHL, D.
98
SCHNEIDER, A.
161*
SCHNIEDERS, B.
86
SCHOENTAL, R.
90
SCHRAMM, T.
315*
SCHRECKER, A.W.
349*
SCHREIBER, H.
557*
SCHRCEDER, M.
193*
SCHUR, P.H.
405
SCHWABE, M.
208
SCHWEINSBERG, F.
85
SCHWEITZER, M.
430*
SEARLE, C.E.
56, 92
SEBRANEK, J.G.
9
SEBRING, E.D.
244
SEGAL, A.
193*
SEIDEL, H.-J.
302*
SEIFERT, E.
233
SEKELY, L.I.
183*
SEKI, S.
415
SELIGMANN, M.
404, 450*
SELKIRK, J.K.
154*
SELLAKUMAR, A.R.
67
SEN, L.
372
SENN, J.S.
545*
SENO, S.
452*
SEON, B.-K.
449*
SEVOIAN, M.
267
SEYDEL, H.G.
214*
SHAH, M.
575
SHEARER, R.W.
170*, 587
SHELLABARGER, C.J.
142*
SHELLEY, W.B.
64
SHEVLYAGIN, V.YA.
270
SHIGEMATSU, T.
260, 357
SHIN, H.S.
355
SHIN, M.L.
108
SHUBLADZE, A.K.
304*
SHULYAKOVSKAYA, T.S.
24
SHUSTER, J.
401, 420
SICKLES, E.A.
443*
SIEGEL, B.V.
366, 445*
SIEGLER, R.
335*
SIGEL, M.M.
471*
SILVER, H.K.B.
420
SILVERMAN, A.Y.
448*
SILVERMAN, M.
401
SIMKOVIC, D.
226
SIMON, R.M.
491*
SIMS, H.L.
348*
SIMS, P.
105, 188*
SIMSIMAN, R.C.
55
SINGER, S.J.
521*

SINGH, S. 588
SINHA, D. 87, 174*
SJOGREN, H.O. 379, 433*
SKLAREW, R.J. 61, 182*
SKORYNA, S.C. 155*
SKOTNIKOV, V.S. 200*
SKURZAK, H. 407
SLATTERY, S. 464*
SLEMMER, G. 552*
SMETANA, K. 281
SMIDA, J. 227
SMIDOVA, V. 227
SMITH, C. 338*
SMITH, G.L. 311*, 312*
SMITH, J.M. 160*
SMITH, L.D. 53
SMITH, P.G. 567
SMITH, W.J. 156*
SMITH, W.T. 92
SMOLAK, K. 526*
SMUCKLER, E.A. 170*, 587
SNYDER, K.L. 147*
SO, B.T. 164*
SOBEL, H. 20*
SOBHY, C.M. 152*
SOLLER, A. 110
SOLOVIEV, V.D. 305*
SOO, S.F. 398
SORENSON, G.D. 358
SOULE, H. 328*
SPIEGELMAN, S. 583
SPIELHOFF, R. 31
SPIELMANN, J. 97
SPORN, M.B. 160*, 349*
STARZL, T.E. 376
STEEVES, R.A. 478*
STEHELIN, D. 254
STEHLIN, J.S. 485*
STEJSKAL, R. 403, 486*
STEPANOVA, L.G. 235
STEPHENSON, H.R. 286
STEVEN, L.M., JR. 344*
STEWART, A. 10
STEWART, H.L. 167*
STOCK, N.D. 316*
STOKER, M.G.P. 2
STOLBACK, L. 509*
STONEHILL, E.H. 510*
STOTT, D.L. 436*
STRAUSS, R.R. 425*
STROBEL, G. 232
STROMBERG, K. 339*
STRONG, L.C. 579
STROUK, V. 407
STURM, M.M. 257, 276
STUTMAN, O. 505*
SUAREZ, H.G. 261
SUGA, K. 277
SUGAWARA, L. 362
SUMIE, H. 63, 168*
SUNDERMAN, F.W., JR. 169*
SUSSMAN, E. 42
SUTHERLAND, J.C. 468*
SVEC, F. 223
SVEC, J. 220, 228
SVEDMYR, A. 423*
SYLVESTER, S.S. 234
SYMES, M.O. 392
TABUCHI, K. 277
TAGASHIRA, Y. 130*
TAKAHASHI, G. 82
TAKAOKA, T. 33
TAKASUGI, M. 480*
TAKAYAMA, S. 113
TAKEMOTO, K.K. 257, 265, 364, 410
TANINO, T. 354
TANNEBERGER, ST. 580
TARANGER, L.A. 458*
TARMY, E.M. 45
TATARINOVA, YU.N. 306*
TAYLOR, J.F. 567
TAYLOR, J.M. 263
TELLER, M.N. 180*
TEMIN, H.M. 3
TENNANT, R.W. 323*, 334*, 463*
TENNEKOON, G.E. 198*
TENSER, R.B. 290*
TER MEULEN, V. 229
TERAO, M. 83
TERASAKI, P.I. 480*
TEVETHIA, S.S. 359, 368, 393
THEILEN, G. 454*
THOLEY, G. 596
THOMAS, C.B. 536*
THOMAS, G.H. 581
THOMFORD, N.R. 204
THOMSON, M.J. 123*
THOREN, M.M. 244
THURZO, V. 220, 228
TICARIC, S. 497*
TING, C.C. 507*
TITTLE, P.W. 127*

TOMII, S. 128*
TONASCIA, J.A. 576
TONG, D. 584
TOOLAN, H.W. 309*
TORMEY, D.C. 448*
TOTH, B. 40, 138*
TRAININ, N. 390
TRASS, T.C. 152*
TRAUTHEN, T. 110
TREGER, A. 135*
TRUJILLO, J.M. 332*
TSETLIN, E.M. 219
TSUMITA, T. 354
TSUYUGUCHI, I. 399
TUCKERMAN, E. 278
TUOMINEN, F.W. 323*, 334*
TUOMINEN, L. 211*
TURNER, H.C. 364
TURNER, W. 62, 246, 255
TWOMEY, P.L. 595
TYNDALL, R.L. 375, 503*
TYRRELL, D.A.J. 395
UCHINO, H. 438*
UCHIYAMA, M. 532*
URBAN, A. 526*
URIEL, J. 389, 400
UTSUMI, K. 452*
VAAGE, J. 513*
VAILLER, D. 391
VAITKEVICIUS, V.K. 470*
VALLADARES, Y. 523*
VAN DEN BERG, H.W. 162*
VAN DUUREN, B.L. 192*, 193*
VAN NOORD, M.J. 297*
VARET, B. 367
VARMUS, H.E. 263
VASS, W. 157*
VENITT, S. 45
VERNON, M.L. 248
VESELY, P. 253
VESSELINOVITCH, S.D. 71, 72, 139*, 140*
VINCENT, P.C. 588
VISEK, W.J. 293*
VLADIMIRSKAYA, E.B. 522*
VLASOVA, S.A. 24
VOLPE, P. 590
VON HAAM, E. 95
VRANA, M. 467*
WAGNER, J. 129*
WAGNER, J.E. 538
WALKER, J.E. 360
WALLACE, D.C. 573
WALLACE, W.C. 167*
WANG, I.Y. 150*
WARD, D.W. 442*
WARNER, G.A. 433*
WARNER, N.L. 365, 396, 521*, 541
WARREN, L. 230, 279
WARREN, S. 213*
WASSERMAN, M.B. 186*
WATSON, W.L. 19*
WATTENBERG, L.W. 166*
WAXMAN, S. 283
WEAVER, P.T. 120*
WEBSTER, G. 542
WECHSLER, W. 91
WEINSTEIN, I.B. 39
WEISBURGER, E.K. 151*
WEISBURGER, J.H. 149*, 151*
WEISS, R.A. 253
WELLER, M. 23*
WELLS, R.J.H. 541
WEST, S.B. 118*
WEXLER, H. 369
WHITEHEAD, V.M. 100
WHITMIRE, C.E. 34, 80
WHYBREW, W.D. 152*
WIBELL, L. 435*
WICKER, R. 261
WIDRIN, P. 346*
WIEBEL, F.J. 195*
WIED, G.L. 229
WIENER, F. 431*
WIERNIK, P.H. 443*
WILDENRADT, E. 346*
WILLCOX, P.A. 581
WILLIAMS, G.M. 151*
WILSON, S.H. 339*
WINNACKER, E.-L. 271, 272
WISLOCKI, P.G. 171*
WITSCHI, H. 77
WOGAN, G.N. 139*
WOLFE, L. 256, 454*
WONG, J.K.Y. 511*
WOODLIFF, H.J. 566
WOODSON, B. 300*
WORZALLA, J.F. 145*
WUNDERLICH, J.R. 473*
WURTZ, B. 596
WYBRAN, J. 455*
WYNDER, E.L. 164*
YAGER, J.D., JR. 178*

YAGI, M.J.
514*
YAGI, Y.
448*, 449*
YAMADA, O.
277
YAMADA, T.
33, 113, 453*
YAMAMOTO, N.
130*
YAMAMOTO, T.
237
YAMAZAKI, M.
83
YANAI, R.
76
YANKEE, R.A.
491*
YASUHIRA, K.
82
YIN, L.K.
489*
YOHN, D.S.
317*
YOKOTA, Y.
163*
YOSHIZU, H.
277
YOUN, J.K.
262
YUHAS, J.M.
463*
ZABEZHINSKIY, M.A.
109
ZACHO, A.
102
ZAJDELA, F.
597
ZAMCHECK, N.
381, 537*
ZARA, H.D.
599
ZARLING, J.M.
359, 368
ZASUKHINA, G.D.
235
ZBAR, B.
487*, 501*
ZEIGEL, R.F.
326*
ZEIGER, R.S.
175*
ZHDANOV, V.M.
241, 270, 305*
ZIEGLER, J.L.
577
ZIMMERMAN, H.M.
30

IC ACID
  CHORIOALLANTOIC MEMBRANE
    FERTILE HEN'S EGG, 60

ETOXY-N-2-ACETYLAMINOFLUORENE
  5'-ENDONUCLEASE ACTIVITY INHIBITION
    MITOCHONDRIA, RAT, 74

ETOXY-2-ACETYLAMINOFLUORENE
  RNA, TRANSFER
    E.COLI, 39

ETOXY-2-FLUORENYLACETAMIDE
  RNASE ACETYLATION, 153*

ETYLAMINOFLUORENE
  INDOLE
    URINARY BLADDER TUMOR, HAMSTER, 168*
  URINARY BLADDER TUMOR
    HAMSTER, 63
  ZYMBAL'S GLAND
    CARCINOGENESIS, RAT, 143*

NOMYCIN C
  VIRUS, AVIAN SARCOMA
    DNA SYNTHESIS INHIBITION, 346*
    SARCOMA FORMATION INHIBITION, 346*

NOMYCIN D
  BINDING CAPACITY
    LYMPHOCYTES, LEUKEMIA PATIENTS, 437*
  RESISTANCE
    SV40-TRANSFORMED HAMSTER CELLS, 261

OCARCINOMA
  CERVIX
    ESTROGEN TREATMENT, CLINICAL STUDY, 38
  VIRAL REPLICATION
    ENVIRONMENTAL CONTROL, FROG, 344*

OPATHY, MALIGNANT
  PICORNA VIRUS
    HUMAN, 269

NOCORTICAL CARCINOMA
  THYMIDINE KINASE ACTIVITY
    ESTROGEN, RAT, 123*
  THYMIDYLATE KINASE ACTIVITY
    ESTROGEN, RAT, 123*

TOXIN
  CHOLESTEROL SYNTHESIS
    PRECANCEROUS LIVER, RAT, 103

TOXIN B1
  COLON CARCINOMA
    VITAMIN A, RAT, 66
  DISTRIBUTION
    LIVER CELLS, CHICK EMBRYO, 83
  ALPHA FETOPROTEIN
    SERUM, RAT, 149*
  HEPATOMA
    INFANT MICE, 139*
  METABOLISM
    LIVER MICROSOME, RAT, 104

  PRIMARY LIVER CARCINOMA
    MONKEY, 122*
  TUMOR INDUCTION
    EPITHELIAL-LIKE CELLS, RAT LIVER, 151*

AGE
  CANCER INCIDENCE
    HUMAN, REVIEW, 20*

AGGLUTINATION
  MAMMARY TUMOR CELL
    SERUM, MOUSE, 354
  TUMOR CELL
    CONCANAVALIN A, MACROPHAGE ADHESION, 452*

ALKYLATION
  DNA
    EPOXIDES, MOLECULAR BASIS, 179*

2-AMINO-4-(5-NITRO-2-FURYL)THIAZOLE
  CARCINOGENICITY
    MOUSE, 190*

2-AMINOETHYLISOTHIURONIUM
  RADIATION PROTECTIVE EFFECT
    CHROMOSOME ABERRATIONS, RAT, 209*

AMMONIA
  SV40-TRANSFORMED FIBROBLASTS
    MOUSE, 293*

ANGIOGENESIS, TUMOR
  IRIS NEOVASCULARIZATION
    INTRAOCULAR TUMOR, RABBIT, 594
  VASOPROLIFERATIVE RESPONSE
    ULTRASTRUCTURE, RAT, 543

ANGIOSARCOMA
  1,1-DIMETHYLHYDRAZINE
    ULTRASTRUCTURE, MOUSE, 40

ANTIBODY
  ANTI-ALPHA2 MACROGLOBULIN ACTIVITY
    HUMAN IGG MYELOMA PROTEIN, 450*
  ANTINUCLEAR
    GRAFFI VIRUS, MOUSE, 367
    RAUSCHER LEUKEMIA VIRUS, MOUSE, 367
  EPSTEIN-BARR VIRUS-ASSOCIATED ANTIGEN
    RADIOTHERAPY, CANCER PATIENTS, 434*
  HERPES-LIKE VIRUS REPLICATION
    THYMUS CELLS, GUINEA PIG, 290*
  LEUKEMIA, ACUTE LYMPHOCYTIC
    HUMAN, 475*
  REAGINIC
    MELANOMA, MOUSE, 411
  VIRUS, AVIAN LEUKOSIS
    IMMUNOGLOBULIN ANALYSIS, CHICKEN, 412
  VIRUS, EPSTEIN-BARR
    CELLULAR IMMUNITY, HODGKIN'S DISEASE PATIENTS, 387
    CELLULAR IMMUNITY, LEUKEMIA PATIENTS, 387

ANTIGEN

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

- ACUTE LEUKEMIA ASSOCIATED
  - SEROLOGIC DETECTION, HUMAN, 472*
- ALLOGENEIC
  - CELL-MEDIATED IMMUNITY, HUMAN, 386
- CARCINOEMBRYONIC
  - COLON CARCINOMA, HUMAN, 428*
  - COLON CARCINOMA PATIENTS, 537*
  - COLONIC CARCINOMA, HUMAN-ANIMAL MODEL, 482*
  - PANCREATITIS PATIENTS, 381
  - RADIOIMMUNO-ELECTROPHORETIC PRECIPITATION, 489*
  - TUMOR TISSUES, CANCER PATIENTS, 470*
  - URINE, CANCER PATIENTS, 470*
- CARCINOEMBRYONIC, HUMAN
  - METABOLISM, RABBIT, DOG, 401
- CELL-SURFACE
  - LYMPHOMA, MOUSE, 377
- CROSSREACTIVE
  - ACUTE LEUKEMIA CELLS, HUMAN, 421
  - RAUSCHER LEUKEMIA VIRUS-INFECTED CELLS, HUMAN, 421
- FELINE ONCORNAVIRUS-ASSOCIATED CELL MEMBRANE
  - INDUCTION, HUMAN CELLS, 454*
- FETAL
  - PAPOVA VIRUS-INDUCED TUMORS, MOUSE, 507*
  - REVIEW, 5
- GASTRIC CARCINOMA
  - HUMAN, 360
- H-2
  - LYMPHOMA, MOUSE, 377
- HEPATOMA
  - IMMUNITY, RAT, 378
- HL-A SYSTEM
  - FREQUENCIES IN CANCER PATIENTS, 480*
  - HODGKIN'S DISEASE, 491*
  - REVIEW, 4
  - TROPHOBLASTIC NEOPLASMS, CLINICAL STUDY, 531*
- HUMAN LYMPHOID TUMOR CELLS
  - TRANSPLANTATION, HAMSTER, 456*
- IMMUNE RESPONSE
  - REVIEW, 15*
- IMMUNOGENICITY
  - DEAD TUMOR CELLS, MOUSE, 473*
- LEUKEMIA ASSOCIATED
  - IMMUNITY, LYMPHOCYTE TRANSFORMATION, HUMAN, 430*
- LEUKEMIA CELL
  - MOUSE, 363
- LYMPH NODE
  - CELL MIGRATION INHIBITION, HUMAN LYMPHOMAS, 372
- MURINE SARCOMA VIRUS ANTISERUM
  - POLYOMA VIRUS-TRANSFORMED CELLS, RAT, 364
- POLYOMA T SYNTHESIS
  - EMBRYO CELL CULTURE, MOUSE, 410
- SURFACE, MURINE SARCOMA VIRUS-SPECIFIC
  - TRANSFORMED CELLS, MOUSE, 407
- SV40 TUMOR-SPECIFIC
  - IMMUNITY, MOUSE, 393
  - MACROPHAGE MIGRATION INHIBITION, MOUSE, 393
- THY 1.2, SURFACE VARIANTS
  - LEUKEMIA CELLS, MOUSE, 383
- TRANSITORY CELL
  - LIVER, RAT, 389
- TRANSPLANTATION
  - IMMUNE RESPONSE, SOLID MURINE TUMORS, 391
  - MODIFICATION, HUMAN TUMOR CELLS, 527*
  - PLASMA CELL TUMOR, MOUSE, 365
  - POLYOMA VIRUS, HYBRID MOUSE CELLS, 361
- TUMOR
  - IMMUNOGENICITY,
    - RETICULOENDOTHELIAL CELLS, MOUSE, 513*
- TUMOR-ASSOCIATED
  - HODGKIN'S DISEASE, 484*
  - HODGKIN'S DISEASE PATIENTS, 440*
- TUMOR-FETAL
  - HODGKIN'S DISEASE, 451*
- TUMOR-SPECIFIC
  - DELAYED CUTANEOUS HYPERSENSITIVITY, GUINEA PIG, 384
  - FETAL TISSUES, HUMAN, 515*
  - HUMAN SARCOMAS, 515*
  - MOUSE EMBRYO, 510*
  - PLASMA MEMBRANE, HUMAN CELLS, 353
- TUMOR SPECIFIC CELL SURFACE
  - PAPOVA VIRUS-INDUCED TUMORS, MOUSE, 507*
- URINARY BLADDER TUMOR
  - IMMUNOLOGIC CLASSIFICATION, HUMAN, 486*
  - MOUSE, RAT, 458*
- VIRUS, AVIAN LEUKOSIS
  - ASCITES SARCOMA CELLS, MOUSE, 415
- VIRUS, AVIAN SARCOMA
  - ASCITES SARCOMA CELLS, MOUSE, 415
- VIRUS, CV
  - CANCER PATIENT SERA, 523*
- VIRUS, EPSTEIN-BARR
  - HEAT STABILITY, 423*
  - LYMPHOBLASTOID CELL LINES, HUMAN, 362
- VIRUS, GROSS LEUKEMIA
  - IMMUNOSUPPRESSION, RAT, 502*
- VIRUS-LIKE PARTICLE
  - LEUKEMIA, HUMAN, 356
- VIRUS, MAMMARY TUMOR
  - MOUSE MAMMARY TUMOR CELL CULTURE, 514*
- VIRUS, MASON-PFIZER MONKEY
  - MAMMARY TUMOR, MONKEY, 464*
- VIRUS, MURINE LEUKEMIA
  - MURINE SARCOMA VIRUS-TRANSFORMED CELLS, 233
- VIRUS-RELATED
  - LYMPHOMA, MOUSE, 409

ANTIGEN, SOLUBLE
- VIRUS, GROSS LEUKEMIA
  - PLASMA, RAT, 276
  - TUMOR CELLS, MOUSE, 295*

ANTILYMPHOCYTE SERUM
- IMMUNOGENICITY
  - VIRUS-INFECTED HUMAN LYMPHOCYTES, 446*

- SERUM
  - GRAFT-VS-HOST DISEASE
    - IMMUNOTHERAPY, MOLONEY LEUKEMIA, MOUSE, 499*
  - MYELOMA CELL
    - HUMAN, 402
  - VIRUS, FRIEND LEUKEMIA
    - LEUKEMOGENIC POTENTIAL, MOUSE, 333*
  - VIRUS, MURINE SARCOMA
    - ANTIGEN, RAT, 364
  - VIRUS, POLYOMA
    - ANTIGEN, RAT, 364

- ATIC AMINES
  - BACTERIAL INTERACTION, 130*
  - BACTERIOPHAGE INTERACTION, 130*

- HYDROCARBON HYDROXYLASE
  - INDUCTION
    - TISSUES, MOUSE, 88
  - METABOLISM
    - VITAMIN A, HAMSTER CELLS, 150*
  - REGULATION
    - MAMMALIAN CELL HYBRIDS, 195*
  - SKIN TUMOR
    - MOUSE, 96
  - STRUCTURE-ACTIVITY RELATIONSHIPS, 84

- STOS
  - ENVIRONMENTAL HAZARD
    - REVIEW, 17*
  - MESOTHELIOMA
    - ULTRASTRUCTURE, RAT, 108
  - OCCUPATIONAL HAZARD
    - REVIEW, 17*

- TES CELL
  - NOVIKOFF
    - ADENYLATE CHARGE CONTROL, 540

- TES TUMOR
  - GLYCOPROTEIN SYNTHESIS
    - NEURAMINIDASE, MOUSE, 586
  - IMMUNITY
    - LYMPH NODE CELLS, MOUSE, 535*
  - LYMPH NODE CELL
    - ELECTROPHORETIC MOBILITY, MOUSE, 536*
  - RESISTANCE
    - MOUSE, 525*
  - RIBONUCLEOTIDE REDUCTASE
    - STEREO SPECIFICITY, RAT, 597

- RBIC ACID
  - BETA-NAPHTHYLAMINE
    - URINARY BLADDER, RABBIT, 36

- PARAGINASE
  - LYMPHOCYTE BLASTOGENESIS
    - CELL SURFACE, RAT, 520*

- RADIATION PROTECTIVE EFFECT
  - CHROMOSOME ABERRATIONS, RAT, 209*

- ACYTIDINE
  - ROUS SARCOMA VIRUS-TRANSFORMED CELLS
    - DNA POLYMERASE, HAMSTER, 225

- BACILLUS CALMETTE-GUERIN
  - IMMUNE RESPONSE
    - POLYOMA TUMOR, RAT, 379
  - TUMOR CELL GROWTH
    - MUSCLE, GUINEA PIG, 487*
  - TUMOR IMMUNITY
    - GUINEA PIG, 501*

- BASAL CELL CARCINOMA
  - GROWTH
    - AUTORADIOGRAPHIC ANALYSIS, HUMAN, 571

- BAYOL
  - PLASMACYTOMA
    - ANTI-THYMOCYTE SERUM, MOUSE, 48

- BENZ(A)ANTHRACENE
  - CARCINOGENESIS
    - MOUSE, 27

- BENZENE
  - COCARCINOGENICITY
    - RAT, 197*

- BENZO(A)PYRENE
  - ADJUVANTS
    - CARCINOGENESIS, HAMSTER, 37
  - COCARCINOGENICITY
    - RAT, 197*
  - DERIVATIVES, 6-SUBSTITUTED
    - CARCINOGENICITY, MOUSE, 94
  - ELECTRON ACCEPTOR COMPLEX FORMATION
    - TEMPERATURE DEPENDENT KINETICS, 200*
  - EPOXIDES, K-REGION
    - METABOLIC FORMATION, 188*
  - GROWTH, TUMOR
    - CARTILAGE, MOUSE, 58
  - 6-HYDROXYMETHYLBENZO(A)PYRENE
    - TUMOR INDUCTION, RAT, 147*
  - METABOLITES, POST LYSOSOMAL, 173*
  - MUTAGENESIS
    - DROSOPHILA, 70
  - RENAL CARCINOGENESIS
    - MOUSE, 140*
  - RESPIRATORY SYSTEM TUMORS
    - HAMSTER, 67
    - ULTRASTRUCTURE, RAT, 160*
  - SQUAMOUS CELL CARCINOMA
    - RAT, 177*

- BENZO(RST)PENTAPHENE
  - CARCINOGENESIS
    - INHIBITION BY FLUORO DERIVATIVES, MOUSE, 135*

- N-BENZOYLOXY-N-METHYL-4-AMINOAZOBENZENE
  - 5'-ENDONUCLEASE ACTIVITY INHIBITION
    - MITOCHONDRIA, RAT, 74

- BERYLLIUM
  - LIVER
    - ULTRASTRUCTURAL CHANGES, RAT, 77

- BLOOD GROUP
  - ABO SYSTEM
    - ERYTHROCYTES, MAMMARY TUMOR PATIENTS, 526*

BLOOD VESSELS
- 1,1-DIMETHYLHYDRAZINE
  - CARCINOGENICITY, MOUSE, 138*

BONE
- OSTEOSARCOMA
  - ETIOLOGY, HUMAN, 325*
  - RADIATION, MOUSE, DOG, 205
  - RADIUM, MOUSE, 208
  - TRACE METALS, HUMAN, 47

BONE MARROW
- CELL POPULATION CHANGES
  - VIRAL LEUKEMOGENESIS, MOUSE, 335*
- CELL PROLIFERATION
  - CYTOTOXIC AGENTS, RAT, 46
- DEFECTIVE COLONY FORMATION
  - LEUKEMIA, HUMAN, 545*
- PARENTAL GRAFTS
  - RESISTANCE, HYBRID MICE, 476*
- THYMIC LYMPHOMA SUPPRESSION
  - MOUSE, 65

BONE SARCOMA
- INCIDENCE
  - SWEDEN, 570

BORDATELLA PERTUSSIS
- TUMOR IMMUNITY
  - GUINEA PIG, 501*

BRAIN
- METHYLNITROSOUREA
  - ULTRASTRUCTURE, RAT, 41

BRAIN TUMOR
- N-ETHYL-N-NITROSOUREA
  - RAT, 92
- VIRUS, ROUS SARCOMA
  - MORPHOLOGY, MOUSE, 237

BREAST
- SEE MAMMARY

BROMELAIN
- TRANSPLANTATION ANTIGENS
  - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

5-BROMODEOXYCYTIDINE
- ADENOVIRUS TRANSFORMATION
  - HAMSTER CELLS, 289

BROMODEOXYURIDINE
- ERYTHROID DIFFERENTIATION INHIBITION
  - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 330*
- HEMOGLOBIN SYNTHESIS INHIBITION
  - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 330*

5-BROMODEOXYURIDINE
- ADENOVIRUS TRANSFORMATION
  - HAMSTER CELLS, 289

7-BROMOMETHYL-12-METHYLBENZ(A)ANTHRACENE
- CARCINOGENESIS
  - MOUSE, 27

7-BROMOMETHYLBENZ(A)ANTHRACENE
- CARCINOGENESIS
  - MOUSE, 27
- DNA
  - E.COLI, 45
- DNA SYNTHESIS
  - LYMPHOCYTES, HUMAN, 146*

BURKITT'S LYMPHOMA
- ANTIGENIC PROPERTIES
  - ATHYMIC MOUSE, 431*
- GROWTH
  - ATHYMIC MOUSE, 431*
  - CELL KINETICS, 577
- LYMPHORETICULAR SYSTEM
  - REVIEW, 14*
- SIMULTANEOUS OCCURRENCE IN NEIGHBORS
  - CASE REPORTS, AMERICA, 298*

BUSULFAN
- CELL PROLIFERATION
  - BONE MARROW, RAT, 46

BUTANOL-BUTYLNITROSAMINE
- URINARY BLADDER TUMOR
  - RAT, 98

CADMIUM
- CARCINOGENESIS
  - INHIBITION BY ZINC, RAT, 133*

CARBAMYL PHOSPHATE
- N-HYDROXY-2-ACETYLAMINOFLUORENE
  - NUCLEIC ACID INTERACTION, 186*
  - PROTEIN INTERACTION, 186*

CARBOHYDRATE
- CELL CONTENT
  - VIRUS TRANSFORMATION, HAMSTER, CHICK, 230
- METABOLISM
  - NEUROBLASTOMA CELLS, 596
- SURFACE GLYCOPROTEINS
  - VIRUS-TRANSFORMED CELLS, 279

CARBON TETRACHLORIDE
- COCARCINOGENICITY
  - RAT, 197*

CELL
- ROSETTE FORMING
  - IMMUNITY, CANCER PATIENTS, 455*

CELL CYCLE
- IMMUNOGLOBULIN CONTENT
  - MYELOMA CELL, MOUSE, 358
- MEMBRANE POTENTIAL
  - OVARIAN TUMOR, HUMAN, 580

CELL, EPIDERMAL
- MITOTIC INHIBITION
  - PSORIASIS LESIONS, HUMAN, 424*

CELL PROLIFERATION
- BONE MARROW
  - CYTOTOXIC AGENTS, RAT, 46
- 2-FLUORENYLACETAMIDE-INDUCED HEPATOMA
  - RAT, 148*
- METHYLCHOLANTHRENE-INDUCED SARCOMA,

- 182*
    - RAT, 61

IUM 144
- CHROMOSOME ABERRATIONS
    - LIVER CELLS, HAMSTER, 212*

VIX
- ADENOCARCINOMA
    - ESTROGEN TREATMENT, CLINICAL STUDY, 38
- SQUAMOUS CELL CARCINOMA
    - IMMUNOLOGY, CLINICAL STUDY, 403
- TUMOR INCIDENCE
    - NETHERLANDS, 569

MERISM
- LYMPH NODE
    - HERPESVIRUS SAIMIRI, MONKEY, 585

HLORO-7-BROMOMETHYLBENZ(A)ANTHRACENE
- CARCINOGENESIS
    - MOUSE, 27

HLOROBENZYLIDENEMALONONITRILE
- ESTERASE ACTIVITY
    - SKIN, MOUSE, 35

ORPROMAZINE
- HEXAMETHYLMELAMINE METABOLISM
    - RAT, HAMSTER, 145*

LESTEROL OXIDE
- ULTRAVIOLET CARCINOGENESIS
    - MOUSE, 207

LESTEROL SYNTHESIS
- AFLATOXIN
    - PRECANCEROUS LIVER, RAT, 103
- N-2-FLUORENYLACETAMIDE
    - PRECANCEROUS LIVER, RAT, 103

NDROMA
- HAFNIUM
    - MOUSE, 64
- ZIRCONIUM
    - MOUSE, 64

RIOALLANTOIC MEMBRANE
- ACETIC ACID
    - FERTILE HEN'S EGG, 60
- CROTON OIL
    - FERTILE HEN'S EGG, 60
- FORMALDEHYDE
    - FERTILE HEN'S EGG, 60
- PHENOL
    - FERTILE HEN'S EGG, 60
- TOBACCO SMOKE CONDENSATE
    - FERTILE HEN'S EGG, 60

RIOCARCINOMA
- CARCINOEMBRYONIC ANTIGEN
    - CASE REPORT, 443*
- HUMAN CHORIONIC GONADOTROPHIN
    - CASE REPORT, 443*
- REGAN ISOENZYME
    - CASE REPORT, 443*

RIONIC TUMOR
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - RAT, 32
- 4-NITROQUINOLINE-1-OXIDE
    - RAT, 32

CHROMATIN
- TRANSCRIPTION
    - HUMAN LYMPHOCYTIC LEUKEMIA CELLS, 589

CHROMOSOME
- HYBRIDIZATION
    - RD114-VIRUS RNA, HUMAN, 283

CHROMOSOME ABERRATIONS
- CERIUM 144
    - LIVER CELLS, HAMSTER, 212*
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - CARCINOGENESIS, MOUSE, 68
- NEOPLASTIC DISEASE
    - HUMAN, REVIEW, 11
- PRASEODYMIUM 144
    - LIVER CELLS, HAMSTER, 212*
- RADIATION, X-RAY
    - RAT, 209*
- VIRUS, HERPES SIMPLEX
    - CELL CYCLE ANALYSIS, HUMAN, 350*

ALPHA-CHYMOTRYPSIN
- TRANSPLANTATION ANTIGENS
    - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

COCARCINOGENICITY
- BENZENE
    - RAT, 197*
- BENZO(A)PYRENE
    - RAT, 197*
- CARBON TETRACHLORIDE
    - RAT, 197*

COLLAGENASE
- TRANSPLANTATION ANTIGENS
    - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

COLON CARCINOMA
- ABH ISOANTIGENS
    - SYNTHETIC, DEGRADATIVE ENZYMES, HUMAN, 490*
- AFLATOXIN B1
    - VITAMIN A, RAT, 66
- CARCINOEMBRYONIC ANTIGEN
    - CLINICAL STUDY, 537*
    - HUMAN, 428*
    - HUMAN TUMOR-ANIMAL MODEL, 482*
- MAMMARY TUMOR
    - FAMILIAL OCCURRENCE, UNITED STATES, 582

CONCANAVALIN A
- AGGLUTINATION, TUMOR CELL
    - MACROPHAGE ADHESION, 452*
- BINDING
    - SV40 TEMPERATURE-SENSITIVE MUTANT, MOUSE CELL, 284
- VIRUS, FRIEND LEUKEMIA ,
    - FRIEND DISEASE IMMUNITY, MOUSE, 258

- COPPER
  - POLAROGRAPHIC MAXIMUM
    - POLYCYCLIC HYDROCARBONS, 196*

- CORTISONE ACETATE
  - CYCLIC IMMUNOSUPPRESSION
    - RADIATION CARCINOGENESIS, MOUSE, 215*

- CROTON OIL
  - CHORIOALLANTOIC MEMBRANE
    - FERTILE HEN'S EGG, 60

- CYCLAMATE
  - URINARY BLADDER TUMOR
    - RAT, 98, 167*

- CYCLOPHOSPHAMIDE
  - HOST RESPONSE
    - SYNGENIC TUMOR TRANSPLANTS, HAMSTER, 518*
  - MAMMARY TUMOR
    - INCIDENCE, RAT, 112

- CYTOSINE ARABINOSIDE
  - VIRUS, HERPES SIMPLEX
    - KIDNEY CELLS, RABBIT, HUMAN, 292*

- CYTOXAN
  - GRAFT-VS-HOST DISEASE
    - IMMUNOTHERAPY, MOLONEY LEUKEMIA, MOUSE, 499*

- DDD
  - PRECURSORS
    - ACID HYDROLYSIS PRODUCTS, 124*

- DDT
  - PRECURSORS
    - ACID HYDROLYSIS PRODUCTS, 124*

- DEOXYCYTIDINE KINASE
  - HERPES SIMPLEX INFECTION
    - HAMSTER CELL LINES, 337*

- 2,7-DIACETYLAMINOFLUORENE
  - LEUKEMIA, CHRONIC MYELOGENOUS
    - RAT, 113

- DIBENZ(A,H)ANTHRACENE
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 289

- DIBENZ(A,H)ANTHRACENE-5,6-EPOXIDE
  - ISOLATION AND CHARACTERIZATION
    - SKIN, MOUSE, 154*

- N-DIBUTYLNITROSAMINE
  - RESPIRATORY SYSTEM TUMORS
    - NURSING HAMSTERS, OFFSPRING, 31

- DIBUTYLNITROSAMINE
  - URINARY BLADDER TUMOR
    - ANGIOGRAPHY, RAT, 117*
    - RNA METABOLISM, RAT, 97

- DIBUTYRYL CYCLIC AMP
  - GLYCOLIPID SYNTHESIS
    - TRANSFORMED HAMSTER CELLS, 294*

- DIETHYLAMINOETHYL-DEXTRAN
  - SARCOMA
    - MOUSE, 62

- DIETHYLNITROSAMINE
  - FOLATE DEFICIENCY
    - RAT, 100
  - FORMIMINOGLUTAMIC ACID EXCRETION
    - RAT, 100
  - HEPATOCELLULAR CARCINOMA
    - RAT, 109
  - LIVER TISSUE
    - FREE RADICAL CONCENTRATION, RAT, 24

- N-DIETHYLNITROSAMINE
  - RESPIRATORY SYSTEM TUMORS
    - NURSING HAMSTERS, OFFSPRING, 31

- DIETHYLSTILBESTROL
  - ADENOCARCINOMA OF CERVIX
    - CLINICAL STUDY, 38

- DIETHYLSTILBESTROL DIPROPIONATE
  - PITUITARY GLAND
    - MITOTIC INDEX, RAT, 73

- DIHYDRO-AFLATOXIN B1
  - DISTRIBUTION
    - LIVER CELLS, CHICK EMBRYO, 83

- 3,2'-DIMETHYL-4-AMINO-DIPHENYL
  - URINARY BLADDER TUMOR
    - HAMSTER, 164*

- 9,10-DIMETHYL-1,2-BENZANTHRACENE
  - DNA BINDING
    - MOUSE, 175*

- DIMETHYL SULFOXIDE
  - CELL DIVISION STIMULATION
    - PLASMA MEMBRANE, BACTERIA, 125*

- 14C-DIMETHYLAMINOAZOBENZENE
  - METABOLITE FIXATION
    - LIVER PROTEINS, RAT, 106

- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - LYMPHOMA
    - SUPPRESSION, MOUSE, 65
  - MAMMARY TUMOR
    - RAT, 142*
    - SHOCK STRESS, RAT, 199*
    - ULTRASTRUCTURE, RAT, 95
  - SKIN TUMOR
    - MOUSE, 96
  - CARCINOGENICITY ALTERATION BY LIGHT
    - MOUSE, 181*
  - CHORIONIC TUMOR
    - RAT, 32
  - CHROMOSOME ABERRATIONS
    - CARCINOGENESIS, MOUSE, 68
  - CORTICOSTERONE BIOSYNTHESIS
    - RAT, 79
  - DEOXYCORTICOSTERONE BIOSYNTHESIS
    - RAT, 79
  - ELECTRON ACCEPTOR COMPLEX FORMATION
    - TEMPERATURE DEPENDENT KINETICS, 200*

HYDROXYMETHYL DERIVATIVES
    CARCINOGENESIS, MOUSE, 105
KIDNEY CELL TRANSFORMATION
    C-TYPE RNA VIRUS ACTIVATION, RAT, 248
LYMPHOMA
    SPLENECTOMY, MOUSE, 187*
MAMMARY TUMOR
    SPLENECTOMY, MOUSE, 187*
PROSTATE
    HUMAN, 131*
TRANSFORMATION
    FELINE EMBRYO CELLS, 157*
TRANSPLACENTAL CARCINOGENESIS
    RAT, 116*
TUMOR INDUCTION
    EPITHELIAL-LIKE CELLS, RAT LIVER, 151*

-DIMETHYLHYDRAZINE
ANGIOSARCOMA
    ULTRASTRUCTURE, MOUSE, 40
BLOOD VESSELS
    CARCINOGENICITY, MOUSE, 138*
KIDNEY
    CARCINOGENICITY, MOUSE, 138*
LUNG
    CARCINOGENICITY, MOUSE, 138*

METHYLMYLERAN
CELL PROLIFERATION
    BONE MARROW, RAT, 46

METHYLNITROSAMINE
FORMATION BY INTESTINAL BACTERIA
    RAT, 78
RNA METHYLATION
    LIVER, MOUSE, 86
TRANSFORMATION, MALIGNANT
    HAMSTER CELLS, 110
TUMOR INDUCTION
    EPITHELIAL-LIKE CELLS, RAT LIVER, 151*

4-DINITROPHENOL
HUMAN DIPLOID CELL TRANSFORMATION
    LEUKEMIA-LIKE VIRUS, 235

PHENYL HYDANTOIN
LYMPHORETICULAR SYSTEM
    MOUSE, 42

SODIUM ETHYLENEDIAMINETETRAACETATE
TRANSPLANTATION ANTIGENS
    SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

A
ALKYLATION
    EPOXIDES, MOLECULAR BASIS, 179*
BINDING
    9,10-DIMETHYL-1,2-BENZANTHRACENE, MOUSE, 175*
7-BROMOMETHYLBENZ(A)ANTHRACENE
    E.COLI, 45
ETHYLATION
    ETHIONINE CARCINOGENESIS, RAT, MOUSE, 191*
N-HYDROXY-2-FLUORENYLACETAMIDE
    LIVER, RAT, 144*
MUTAGENESIS
    ETHYL METHANESULFONATE, 119*
    METHYL METHANESULFONATE, 119*
BETA-PROPIOLACTONE
    SKIN, MOUSE, 193*
TRANSCRIPTION-CONTROL REGION
    ADENOVIRUS 2-SV40 HYBRID, 243
VIRUS-SPECIFIC
    HYBRIDIZATION, HUMAN LEUKEMIC CELLS, 583

DNA POLYMERASE
    INHIBITION BY METHYLATED POLYNUCLEOTIDES
        VIRUS, MURINE LEUKEMIA, 323*
    INHIBITION BY THIOSEMICARBAZONE
        VIRUS, ROUS SARCOMA, 300*
    PRENEOPLASTIC NODULES
        MAMMARY GLAND, MOUSE, 538
    RNA TRANSCRIPTION
        E.COLI, 263
        VIRUS, ROUS SARCOMA, 263
    ROUS SARCOMA VIRUS-TRANSFORMED CELLS
        5-AZACYTIDINE, HAMSTER, 225
    TEMPLATE SPECIFICITY
        MYELOMA, MOUSE, 578
    VIRUS, RAUSCHER LEUKEMIA
        ACTIVE-SITE-DIRECTED INHIBITORS, 349*

DNA REPLICATION
    SEMICONSERVATIVE
        POLYOMA VIRUS, MOUSE, 272
    VIRUS, POLYOMA
        FIBROBLASTS, MOUSE, 271
    VIRUS, SV40
        BIDIRECTIONAL, 249
        GROWTH CYCLE ANALYSIS, 254
        INITIATION SITE, 244

DNA SYNTHESIS
    7-BROMOMETHYLBENZ(A)ANTHRACENE
        LYMPHOCYTES, HUMAN, 146*
    CYTOPLASMIC
        MITOTIC CYCLE, HELA CELLS, 590
    INHIBITION
        METHYLAZOXYMETHANOL ACETATE, 162*
    3-METHYLCHOLANTHRENE
        MAMMARY NODULE, MOUSE, 129*
    NUCLEAR
        MITOTIC CYCLE, HELA CELLS, 590
    PHYTOHEMAGGLUTININ
        PHAGOCYTE, 425*
        SPLEEN CELLS, LEUKEMIC MOUSE, 425*
    PRENEOPLASTIC NODULES
        MAMMARY GLAND, MOUSE, 538
    RNA-DIRECTED
        REVIEW, 3
    SKIN TUMOR
        URETHANE, MOUSE, 93
    SV40 INFECTION
        HAMSTER CELLS, 343*

DNA TRANSCRIPTION
    VIRUS, HERPES SIMPLEX
        CERVICAL CANCER TISSUE, HUMAN, 221
    VIRUS, SV40
        TRANSFORMED CELLS, MONKEY, 265

- ENVIRONMENTAL HAZARD
  - ASBESTOS
    - REVIEW, 17*

- ENZYME
  - SEE ALSO NAMES OF
  - ARYL HYDROCARBON HYDROXYLASE
    - REGULATION, MAMMALIAN CELL HYBRIDS, 195*
    - SKIN TUMORS, MOUSE, 96
    - STRUCTURE-ACTIVITY RELATIONSHIPS, 84
    - VITAMIN A, HAMSTER CELLS, 150*
  - ARYL HYDROCARBON HYDROXYLASE INDUCTION
    - TISSUES, MOUSE, 88
  - DEOXYCYTIDINE KINASE
    - HERPES SIMPLEX INFECTION, HAMSTER CELL LINES, 337*
  - EPOXIDE HYDRASE
    - SKIN TUMORS, MOUSE, 96
  - PATTERN
    - HEPATOMA, RAT, 544
  - RIBONUCLEASE
    - VIRUS, MURINE LEUKEMIA, 331*
  - RNASE ACETYLATION
    - N-ACETOXY-2-FLUORENYLACETAMIDE, 153*
  - THYMIDYLATE BIOSYNTHETIC
    - MAMMARY TUMOR, MOUSE, 553*

- EPIDEMIOLOGY
  - CANCER INCIDENCE
    - CALIFORNIA, 560
  - MAMMARY TUMOR
    - REVIEW, 1

- EPOXIDE HYDRASE
  - SKIN TUMOR
    - MOUSE, 96

- EPOXIDES
  - DNA ALKYLATION
    - MOLECULAR BASIS, 179*

- EPOXIDES, K-REGION
  - BENZO(A)PYRENE
    - METABOLIC FORMATION, 188*

- ERYTHROLEUKEMIA
  - VIRUS, RAUSCHER LEUKEMIA
    - CULTURE LINE ESTABLISHMENT, MOUSE, 348*

- ERYTHROPOIETIN
  - VIRUS, FRIEND LEUKEMIA
    - POLYCYTHEMIC MICE, 324*

- ESTRADIOL
  - INTERSTITIAL CELL TUMORS
    - MOUSE, 44

- ESTRIOL
  - MAMMARY TUMOR INHIBITION
    - RAT, 165*

- ESTROGEN
  - ADRENOCORTICAL CARCINOMA
    - THYMIDINE KINASE ACTIVITY, RAT, 123*
    - THYMIDYLATE KINASE ACTIVITY, RAT, 123*
  - ALPHA FETOPROTEIN
    - INTRACELLULAR LOCALIZATION, RAT LIVER, 400
  - MAMMARY TUMOR
    - PARITY, CLINICAL STUDY, 29
    - RAT, 87, 174*
    - THYMIDINE KINASE ACTIVITY, RAT, 123*
    - THYMIDYLATE KINASE ACTIVITY, RAT, 123*
  - METABOLISM
    - CULTURED MAMMARY TUMORS, HUMAN, 581
  - PITUITARY GLAND
    - MITOTIC INDEX, RAT, 73

- ETHANOL
  - CELL DIVISION STIMULATION
    - PLASMA MEMBRANE, BACTERIA, 125*

- ETHIDIUM BROMIDE
  - RNA SYNTHESIS INHIBITION
    - MELANOMA, HAMSTER, 313*

- ETHIONINE
  - CARCINOGENESIS
    - DNA ETHYLATION, RAT, MOUSE, 191*
    - 1,10-PHENANTHROLINE, RAT LIVER, 184*

- DL-ETHIONINE
  - HEPATOMA
    - HYPOPHYSECTOMY, RAT, 163*

- ETHYL METHANESULFONATE
  - MUTAGENESIS
    - DNA, 119*

- ETHYL NITROSOUREA
  - CARCINOGENESIS
    - NEONATAL MARSUPIAL, 194*

- N-ETHYL-N-NITROSOUREA
  - NERVOUS SYSTEM TUMORS
    - MOUSE, 56
  - BRAIN TUMOR
    - RAT, 92

- ETHYLATION
  - DNA
    - ETHIONINE CARCINOGENESIS, RAT, MOUSE, 191*
  - MUTAGENESIS
    - DNA, 119*

- ETHYLNITROSOUREA
  - HEPATOMA
    - TRANSPLANTABILITY, MOUSE, 72
  - KIDNEY
    - CARCINOGENESIS, MOUSE, 71
  - NERVE TUMOR
    - ULTRASTRUCTURE, RAT, 30
  - NERVOUS SYSTEM TUMORS
    - TRANSPLANTATION, RAT, 91

- ETHYLPHENYLPROPIOLATE
  - SKIN TUMOR PROMOTION

    - MOUSE, 134*

- INTRAOCULAR TUMOR
  - IRIS NEOVASCULARIZATION, RABBIT, 594

RIC OXIDE
- RESPIRATORY SYSTEM TUMORS
  - HAMSTER, 67
- SQUAMOUS CELL CARCINOMA
  - RAT, 177*

HA FETOPROTEIN
- AFLATOXIN B1
  - SERUM, RAT, 149*
- EXPERIMENTAL INDUCTION
  - ADULT RABBIT, 352
- N-2-FLUORENYLACETAMIDE
  - SERUM, RAT, 149*
- HEPATOCARCINOGENS
  - SERUM, RAT, 149*
- INTRACELLULAR LOCALIZATION
  - ESTROGENS, RAT LIVER, 400
- 2'-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - SERUM, RAT, 149*
- 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - LIVER, RAT, 389
  - SERUM, RAT, 149*
- ONTOGENY
  - FETAL, NEONATAL RABBIT, 352
- RADIOIMMUNOASSAY
  - HUMAN, 420

ROBLAST
- TRANSFORMATION
  - TUMOR VIRUSES, REVIEW, 2
- TRANSFORMATION, MALIGNANT
  - FLUORENYLHYDROXAMIC ACIDS, RAT, 183*

ROSARCOMA
- IMMUNITY
  - TUMOR SIZE, MOUSE, 394
- INDUCTION
  - IMMUNOSUPPRESSED MOUSE, 398
- MACROPHAGE CONTENT
  - SYNGENEIC RAT, MOUSE, 422*
- 3-METHYLCHOLANTHRENE
  - IMMUNITY, GROWTH, MOUSE, 461*

LUORENYLACETAMIDE
- DOSE FRACTIONATION
  - CELL SURVIVAL, RAT LIVER, 132*
  - HYPERPLASTIC RESPONSE, RAT LIVER, 132*
- HEPATOMA
  - CELL PROLIFERATION, RAT LIVER, 148*

-FLUORENYLACETAMIDE
- CHOLESTEROL SYNTHESIS
  - PRECANCEROUS LIVER, RAT, 103
- ALPHA FETOPROTEIN
  - SERUM, RAT, 149*
- HEPATOMA
  - RNA TRANSPORT ALTERATION, RAT, 170*

FLUORENYLHYDROXAMIC ACID
- TRANSFORMATION, MALIGNANT
  - FIBROBLASTS, RAT, 183*

FOOD
- NITROSAMINES
  - REVIEW, 9

FORMALDEHYDE
- CHORIOALLANTOIC MEMBRANE
  - FERTILE HEN'S EGG, 60

FREUND'S ADJUVANT
- GROWTH, TUMOR
  - GUINEA PIG, 51, 494*
- RADIATION LEUKEMIA VIRUS-INDUCED LYMPHOMA
  - MOUSE, 511*
- TUMOR IMMUNITY
  - GUINEA PIG, 501*
- VIRUS, RAUSCHER LEUKEMIA
  - POTENTIATION, MOUSE, 366

FRIEND DISEASE
- IMMUNITY
  - CONCANAVALIN A, MOUSE, 258
  - PHYTOHEMAGGLUTININ, MOUSE, 258
  - PLANT LECTINS, MOUSE, 258

GAMMAGLOBULIN
- LEUKEMIA
  - CHILDREN, 418

GASTRIC CARCINOMA
- ANTIGEN
  - HUMAN, 360
- TOBACCO SMOKING
  - CLINICAL STUDY, 102

GLIOMA
- IMMUNE RESPONSE
  - LYMPHOCYTE STIMULATION, HUMAN, 370

GLIOMA, CEREBRAL
- TUBERCULOSIS
  - ASSOCIATION, CLINICAL STUDY, 442*

GLYCOLIPID
- TRANSFORMED CELL
  - FRIEND LEUKEMIA VIRUS, MOUSE, 232
  - SV40, MOUSE, 232

GLYCOLIPID SYNTHESIS
- DIBUTYRYL CYCLIC AMP
  - TRANSFORMED HAMSTER CELLS, 294*

GLYCOPROTEIN
- CARBOHYDRATE CONTENT
  - VIRUS-TRANSFORMED CELLS, 279
- MAMMARY TUMOR AGENT
  - PROPERTIES, 492*

GLYCOPROTEIN, MUCOSAL
- RECTAL CARCINOMA
  - IMMUNOHISTOLOGICAL STUDIES, HUMAN, 414

GLYCOPROTEIN SYNTHESIS
- ASCITES TUMOR

    NEURAMINIDASE, MOUSE, 586

GLYCOSYLTRANSFERASE
  SIALIC ACID LEVELS
    POLYOMA VIRUS-TRANSFORMED HAMSTER CELLS, 299*
    SARCOMA VIRUS-TRANSFORMED HAMSTER CELLS, 299*
    SV40-TRANSFORMED MOUSE CELLS, 299*

GRAFT-VS-HOST DISEASE
  ANTISERUM
    IMMUNOTHERAPY, MOLONEY LEUKEMIA, MOUSE, 499*
  CYTOXAN
    IMMUNOTHERAPY, MOLONEY LEUKEMIA, MOUSE, 499*

GRAFT-VS-HOST REACTION
  LEUKEMIA
    MOUSE, 457*

GRANULAR CELL
  MYOBLASTOMA
    TISSUES, HUMAN, 546*

GRANULOCYTOPOIESIS
  VIRULENT LEUKEMOGENIC VIRUS PRODUCTION
    SPLEEN CULTURES, MOUSE, 328*

GROWTH
  BASAL CELL CARCINOMA
    AUTORADIOGRAPHIC ANALYSIS, HUMAN, 571
  BURKITT'S LYMPHOMA
    ATHYMIC MOUSE, 431*
    CELL KINETICS, 577
  GOMPERTZIAN TUMOR
    SYSTEMIC FACTORS SPECIFICITY, MOUSE, 496*
  IMMUNITY
    FIBROSARCOMAS, MOUSE, 461*
  MAMMARY TUMOR
    HUMAN PLACENTAL LACTOGEN, RAT, 76
  3-METHYLCHOLANTHRENE CARCINOGENESIS
    IMMUNODEPRESSION, MOUSE, 392
  PLATING EFFICIENCY
    METHYLNITROSOUREA, MOUSE, 26
  SV40-TRANSFORMED CELL
    LIPID CONTENT, 224
  TUMOR CELL
    BCG, GUINEA PIG, 487*

GROWTH, TUMOR
  BENZO(A)PYRENE
    CARTILAGE, MOUSE, 58
  FREUND'S ADJUVANT
    GUINEA PIG, 51, 494*

HAFNIUM
  CHONDROMA
    MOUSE, 64

HEMANGIOMA
  NITROSODIMETHYLAMINE
    MOUSE, 50

HEPATOCARCINOGENESIS
  NITROSOMORPHOLINE
    RAT, 115

HEPATOCELLULAR CARCINOMA
  DIETHYLNITROSAMINE
    RAT, 109

HEPATOMA
  SEE ALSO LIVER
  AFLATOXIN B1
    INFANT MICE, 139*
  ANTIGEN
    IMMUNITY, RAT, 378
  ENZYME PATTERN
    RAT, 544
  DL-ETHIONINE
    HYPOPHYSECTOMY, RAT, 163*
  ETHYLNITROSOUREA
    TRANSPLANTABILITY, MOUSE, 72
  2-FLUORENYLACETAMIDE
    CELL PROLIFERATION, RAT LIVER, 148*
  N-2-FLUORENYLACETAMIDE
    RNA TRANSPORT ALTERATION, RAT, 170*
  1-HYDROXYSAFROLE
    RAT, 171*
  3'-METHYL-DIMETHYLAMINOAZOBENZENE
    RNA TRANSPORT ALTERATION, RAT, 170*
  ALPHA-NAPHTHYLISOTHIOCYANATE
    HYPOPHYSECTOMY, RAT, 163*
  NITROSOMORPHOLINE
    RAT, 99
  POLYCHLORINATED BIPHENYLS
    MOUSE, 128*
  RNA STAINING
    CYTOPHOTOMETRIC MEASUREMENTS, 555*
  RNA TRANSPORT
    ALTERED REGULATION, RAT CELLS, 587

HEPATOMA, MORRIS
  ADENYLATE CHARGE CONTROL, 539

HERPES ZOSTER
  LYMPHOMA, MALIGNANT
    CLINICAL STUDY, 516*

HETEROTRANSPLANTATION
  TUMORS
    HAMSTER, HUMAN, 528*

HEXAMETHYLMELAMINE
  METABOLISM
    HEPATIC MICROSOMES, RAT, HAMSTER, 145*

HISTONE
  VIRUS, MOLONEY SARCOMA
    ONCOGENICITY STIMULATION, MOUSE, 306*

HODGKIN'S DISEASE
  ANTIGEN
    HL-A SYSTEM, 491*
    TUMOR-ASSOCIATED, 484*
  ANTIGENS, TUMOR-ASSOCIATED
    HUMAN, 440*
  ANTIGENS, TUMOR-FETAL
    IMMUNOELECTROPHORETIC ANALYSIS,

451*
DRUG-INDUCED VIRUS PRODUCTION
  LYMPHOCYTIC CELLS, HUMAN, 319*
EPSTEIN-BARR VIRUS ANTIBODY
  CELLULAR IMMUNITY, HUMAN, 387
IMMUNOLOGY
  REVIEW, 12
INCIDENCE
  OHIO, 574
LYMPHORETICULAR SYSTEM
  REVIEW, 14*

RMONE
 SEE ALSO NAMES OF
 ADENOVIRUS TRANSFORMATION
  HAMSTER CELLS, 317*
 MAMMARY TUMOR REGRESSION
  RAT, 176*

ALURONIDASE
 INHIBITOR
  BLOOD SERUM, CANCER PATIENTS, 529*
 TRANSPLANTATION ANTIGENS
  SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

BRIDIZATION
 RD114-VIRUS RNA
  CHROMOSOME, HUMAN, 283

DROCARBON, POLYCYCLIC
 CARCINOGENIC ACTIVITY
  POLAROGRAPHIC MAXIMUM OF COPPER, 196*
 CARCINOGENIC INHIBITION
  ETHOXYQUIN, RAT, 166*
  PHENOLIC ANTIOXIDANTS, RAT, 166*
 INCORPORATION INTO PROTEIN
  MECHANISMS, 152*
 SOIL CONTENT
  ICELAND, 201*
 VIRAL TRANSFORMATION
  HAMSTER CELLS, 329*

DROCARBON, POLYCYCLIC AROMATIC
 ARYL HYDROCARBON HYDROXYLASE
  STRUCTURE-ACTIVITY RELATIONSHIPS, 84

DROCORTISONE
 ANTIGENIC ALTERATION
  LYMPHOBLASTIC LEUKEMIA CELLS, MOUSE, 459*

HYDROXY-ACETYLAMINOFLUORENE
 MITOCHONDRIA
  CARCINOGENICITY, RAT, 159*

HYDROXY-N-ACETYLAMINOFLUORENE
 MITOCHONDRIA
  RAT, 54

HYDROXY-2-ACETYLAMINOFLUORENE
 NUCLEIC ACID INTERACTION
  CARBAMYL PHOSPHATE, 186*
 PROTEIN INTERACTION
  CARBAMYL PHOSPHATE, 186*

HYDROXY-2-AMINOFLUORENE
 5'-ENDONUCLEASE ACTIVITY INHIBITION
  MITOCHONDRIA, RAT, 74

N-HYDROXY-2-FLUORENYLACETAMIDE
 DNA
  LIVER, RAT, 144*

N-HYDROXY-N-2-FLUORENYLACETAMIDE
 TUMOR INDUCTION
  EPITHELIAL-LIKE CELLS, RAT LIVER, 151*

HYDROXYLAMINE, AROMATIC
 CARCINOGENIC ACTIVATION
  ENZYMATIC O-ACETYLATION, 172*

7-HYDROXYMETHYL-12-METHYLBENZ(A)ANTHRACENE
 TERATOGENESIS
  CELL DEATH, RAT, 28

6-HYDROXYMETHYLBENZO(A)PYRENE
 BENZO(A)PYRENE
  TUMOR INDUCTION, RAT, 147*

3-HYDROXYPROPANE SULFONIC ACID
 REACTION WITH ADENOSINE, 192*
 REACTION WITH DNA, 192*
 REACTION WITH GUANOSINE, 192*

1-HYDROXYSAFROLE
 HEPATOMA
  RAT, 171*

HYPERSENSITIVITY
 CELLULAR
  MAMMARY TUMOR, HUMAN, 506*
 DELAYED CUTANEOUS
  TUMOR-SPECIFIC ANTIGEN, GUINEA PIG, 384
 IMMUNITY
  INFLAMMATORY RESPONSE, GUINEA PIG, 508*

IMMUNE COMPLEX DISEASE
 LEUKEMIA
  ONCORNAVIRUS RELATED ANTIGEN, HUMAN, 468*
 LYMPHOMA
  ONCORNAVIRUS RELATED ANTIGEN, HUMAN, 468*

IMMUNE RESPONSE
 ANTI-TUMOR REACTIVITY
  FETAL TISSUES, HAMSTER, MOUSE, 500*
 ANTIGEN
  REVIEW, 15*
 BACILLUS CALMETTE-GUERIN
  POLYOMA TUMOR, RAT, 379
 GENETIC CONTROL
  SPLEEN CELLS, MOUSE, 417
 GLIOMA
  LYMPHOCYTE STIMULATION, HUMAN, 370
 MODIFIED LYMPHOMA CELLS
  TUMOR-HOST RELATIONSHIP, MOUSE, 497*
 NEWCASTLE DISEASE VIRUS-INFECTED TUMOR CELLS
  MOUSE, 395

- REGIONAL LYMPHATICS
  - GUINEA PIG, 380
- SERUM MEDIATED INHIBITION
  - AUTOLOGOUS TUMOR CELLS, HUMAN, 416
- TRANSPLANTATION ANTIGENS
  - SOLID MURINE TUMORS, MOUSE, 391
- VIRUS, RNA TUMOR
  - MOUSE, 463*
- X-IRRADIATED TUMOR CELLS
  - MOUSE, 395

IMMUNITY
- ANTITUMOR CELL-MEDIATED
  - ALLOGENEIC ANTIGENS, HUMAN, 386
- ASCITES TUMOR
  - LYMPH NODE CELLS, MOUSE, 535*
- CELL DEATH
  - MOUSE, 374
- CELL MEDIATED
  - LYMPHOCYTE BLASTOGENESIS, HUMAN, 469*
- FRIEND DISEASE
  - CONCANAVALIN A, MOUSE, 258
  - PHYTOHEMAGGLUTININ, MOUSE, 258
  - PLANT LECTINS, MOUSE, 258
- GROWTH
  - FIBROSARCOMAS, MOUSE, 461*
- HEPATOMA-SPECIFIC ANTIGENS
  - RAT, 378
- HYPERSENSITIVITY
  - INFLAMMATORY RESPONSE, GUINEA PIG, 508*
- IMMUNE SERA
  - MOLONEY VIRUS-INDUCED LYMPHOMA, 236
  - MOLONEY VIRUS-INDUCED SARCOMA, 236
- LEUKEMIA
  - GUINEA PIG, 427*
- LEUKEMIA ASSOCIATED ANTIGENS
  - LYMPHOCYTE TRANSFORMATION, HUMAN, 430*
- LUNG TUMOR PRODUCTION
  - TUMOR CELL INOCULATION ROUTE, MOUSE, 369
- LYMPHOCYTE PROTEIN SYNTHESIS
  - GUINEA PIG, MOUSE, 498*
- LYMPHOCYTE STIMULATION
  - GLIOMA, HUMAN, 370
- MAMMARY TUMOR
  - ANTIBODY-CELL INTERACTIONS, MOUSE, 505*
- METHYLCHOLANTHRENE INDUCED TRANSPLANTED SARCOMA
  - GUINEA PIG, 517*
- PLASMA CELL TUMOR
  - TRANSPLANTATION ANTIGENS, MOUSE, 365
- REGIONAL LYMPH NODE
  - MAMMARY TUMOR, MOUSE, 444*
- ROSETTE FORMING CELL
  - CANCER PATIENTS, 455*
- SUPPRESSION
  - DEAD TUMOR CELL ANTIGENS, MOUSE, 473*
- SV40 TUMOR-SPECIFIC ANTIGENS
  - MACROPHAGE MIGRATION INHIBITION, MOUSE, 393
- TRANSPLANTATION
  - SV40-TRANSFORMED CELLS, MOUSE, 359, 368
- TUMOR
  - BCG, GUINEA PIG, 501*
  - BORDATELLA PERTUSSIS, GUINEA PIG, 501*
  - FREUND'S ADJUVANT, GUINEA PIG, 501*
  - SEA STAR EXTRACT, MOUSE, 477*
- TUMOR ALLOGRAFT
  - REGIONAL LYMPHATICS, GUINEA PIG, 533*
- TUMOR SIZE
  - FIBROSARCOMA, MOUSE, 394
- TUMOR-SPECIFIC
  - MALIGNANT MELANOMA, CLINICAL STUDY, 509*
  - POLY I:C, MOUSE, 460*
- TUMOR TRANSPLANT
  - POLY I:C, RAT, HAMSTER, 228
- VIRUS, ROUS SARCOMA
  - AVIAN LEUKOSIS VIRUSES, CHICKEN, 471*

IMMUNITY, CELLULAR
- EPSTEIN-BARR VIRUS ANTIBODY
  - CHRONIC LYMPHATIC LEUKEMIA, HUMAN, 387
  - HODGKIN'S DISEASE, HUMAN, 387
- MALIGNANT, MELANOMA
  - HUMAN, 483*

IMMUNODEPRESSION
- 3-METHYLCHOLANTHRENE CARCINOGENESIS
  - TUMOR GROWTH, MOUSE, 392

IMMUNOGENICITY
- ANTIGEN
  - DEAD TUMOR CELLS, MOUSE, 473*
- MAMMARY TUMOR
  - GLYCOCALYX DISSOCIABILITY, 465*
  - METASTASIZING CAPACITY, 465*
  - SURFACE ANTIGEN SOLUBILITY, 465*
- TUMOR ANTIGENS
  - LIVER RETICULOENDOTHELIAL CELLS, MOUSE, 513*
- VIRUS-INFECTED HUMAN LYMPHOCYTES
  - ANTILYMPHOCYTE SERUM, 446*

IMMUNOGLOBULIN
- AVIAN LEUKOSIS VIRUS ANTIBODY
  - CHICKEN, 412
- LEUKEMIA
  - CHILDREN, 418
- LUNG CANCER
  - BRONCHIAL SECRETIONS, HUMAN, 495*
- LYMPHOCYTE BLASTOGENIC RESPONSE
  - LEUKEMIA CELLS, HUMAN, 382, 385
- MONOCLONAL IGA AND IGM
  - SERUM, CASE REPORT, 448*, 449*
- MYELOMA CELL
  - CELL CYCLE, MOUSE, 358
- SERUM LEVELS
  - LYMPHOBLASTIC LEUKEMIA PATIENTS, CHILDREN, 408
- SURFACE
  - BOVINE LYMPHOSARCOMA CELLS, 399
  - LYMPHOCYTES, LEUKEMIA PATIENTS, 419
- SYNTHESIS

    - LYMPHOBLASTOID CELL LINES, HUMAN, 432*
  - THYMOMAS
    - MOUSE, 396

- MUNOGLOBULIN IGD
  - PURIFICATION
    - IMMUNE ADSORPTION, HUMAN, 524*

- MUNOGLOBULIN IGG
  - LIGHT-CHAIN POOL
    - PLASMACYTOMAS, MOUSE, 436*
  - MYELOMA, MULTIPLE
    - ULTRASTRUCTURE, HUMAN, 357

- MUNOLOGY
  - ADENOVIRUS 12-INDUCED TUMORS
    - RESISTANCE, MOUSE, 479*
  - HODGKIN'S DISEASE
    - REVIEW, 12
  - LEUKEMIA, MYELOMONOCYTIC
    - CHILDREN, 404
  - SQUAMOUS CELL CARCINOMA OF CERVIX
    - CLINICAL STUDY, 403
  - TUMOR ETIOLOGY
    - REVIEW, 7

- MUNOSUPPRESSION
  - FIBROSARCOMA INDUCTION
    - MOUSE, 398
  - GROSS LEUKEMIA VIRUS ANTIGENS
    - RAT, 502*
  - MALIGNANT TUMOR DEVELOPMENT
    - ORGAN TRANSPLANT RECIPIENTS, 376
  - MAREK'S DISEASE
    - SERUM PROTEIN CHANGES, CHICKEN, 426*

- PLANTATION
  - LIVING SOLID-STATE SURFACE IMPLANTS
    - RAT, 558*
  - SOLID-STATE IMPLANTS
    - HISTOLOGIC QUIESCENCE, RAT, 559*

- DOLE
  - 2-ACETYLAMINOFLUORENE
    - URINARY BLADDER TUMOR, HAMSTER, 168*

- FECTIOUS MONONUCLEOSIS
  - CANCER RISK
    - MALES, UNITED STATES, 572
  - SERUM
    - MURINE LYMPHOMA CELLS, 397

- TESTINE
  - SMALL-BOWEL TUMORS
    - HUMAN, REVIEW, 18*

- TESTINE, LARGE
  - CARCINOMA
    - RADIOTHERAPY, CERVICAL CARCINOMA PATIENTS, 202

- IODO-2'-DEOXYURIDINE
  - RESISTANCE
    - LYMPHOMA CELLS, 43

- IODODEOXYURIDINE
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 289

- KAPOSI'S SARCOMA
  - INCIDENCE
    - UGANDA, 567

- KARYOTYPE
  - TUMORIGENIC POTENTIAL
    - HYBRID CLONES, HAMSTER, 592

- KIDNEY
  - 1,1-DIMETHYLHYDRAZINE
    - CARCINOGENICITY, MOUSE, 138*
  - ENDEMIC NEPHROPATHY
    - INCIDENCE, YUGOSLAVIA, 564
  - ETHYLNITROSOUREA
    - CARCINOGENESIS, MOUSE, 71

- KIDNEY TUMOR
  - BENZO(A)PYRENE
    - MOUSE, 140*

- LARYNX
  - CARCINOMA
    - ONCORNA VIRUS, HUMAN CELLS, 241

- LEUKEMIA
  - BONE MARROW
    - DEFECTIVE COLONY FORMATION, HUMAN, 545*
  - CELL ANTIGENS
    - MOUSE, 363
  - GRAFT-VS-HOST REACTION
    - MOUSE, 457*
  - IMMUNE COMPLEX DISEASE
    - ONCORNAVIRUS RELATED ANTIGEN, HUMAN, 468*
  - IMMUNITY
    - GUINEA PIG, 427*
  - IMMUNITY, MACROPHAGE-MEDIATED
    - HYPERIMMUNE ISOANTIBODY, MOUSE, 462*
  - IMMUNOGLOBULIN ALTERATIONS
    - CHILDREN, 418
  - INCIDENCE
    - AUSTRALIA, 566
  - INDUCTION
    - LYMPHOID CELL INOCULATION, MOUSE, 390
  - LYMPHOBLASTIC CELLS
    - ANTIGENIC ALTERATION, MOUSE, 459*
  - LYMPHOCYTE BLASTOGENIC RESPONSE
    - IMMUNOGLOBULIN, HUMAN CELLS, 382, 385
  - MACROPHAGE RECOGNITION FACTOR
    - RAT, 373
  - 3-METHYLCHOLANTHRENE
    - SPLENECTOMY, MOUSE, 34
    - THYMECTOMY, MOUSE, 34
  - MIXED LEUKOCYTE CULTURE REACTIVITY
    - HUMAN LEUKEMIC CELLS, 519*
  - RADIATION, X-RAY
    - CHILDREN, 218*
  - SERUM GAMMAGLOBULIN CONTENT
    - CHILDREN, 418
  - SUSCEPTIBILITY
    - INTRAUTERINE X-IRRADIATION, CHILDREN, 206

- VIRUS, C-TYPE PARTICLE
  - HAMSTER, 315*
  - LYMPHOID CELL LINES, CATTLE, 316*
- VIRUS-LIKE PARTICLE
  - ANTIGEN, HUMAN, 356
- VIRUS-SPECIFIC DNA
  - HYBRIDIZATION, HUMAN, 583

LEUKEMIA, ACUTE
- CYTOGENETIC STATUS
  - CLINICAL STUDY, ADULTS, 588
- LYMPHOCYTE TRANSFORMATION
  - PHYTOHEMAGGLUTININ, CHILDREN, 522*

LEUKEMIA, ACUTE LYMPHOBLASTIC
- SERUM IMMUNOGLOBULIN LEVELS
  - CHILDREN, 408

LEUKEMIA, ACUTE LYMPHOCYTIC
- ANTIBODY, CYTOTOXIC
  - HUMAN, 475*

LEUKEMIA, CHRONIC LYMPHATIC
- EPSTEIN-BARR VIRUS ANTIBODY
  - CELLULAR IMMUNITY, HUMAN, 387

LEUKEMIA, CHRONIC MYELOGENOUS
- 2,7-DIACETYLAMINOFLUORENE
  - RAT, 113

LEUKEMIA, HUMAN
- ANTIGEN
  - VIRUS-LIKE PARTICLE, 356

LEUKEMIA, MYELOID
- CYTOGENETIC ANALYSIS
  - HUMAN CELLS, 591

LEUKEMIA, MYELOMONOCYTIC
- IMMUNOLOGICAL ABNORMALITIES
  - CHILDREN, 404

LEUKEMOGENESIS
- N-(4(5-NITRO-2-FURYL)-2-THIAZOLYL) ACETAMIDE
  - EFFECT OF P-HYDROXYACETANILIDE, MOUSE, 137*
  - EFFECT OF SODIUM SULFATE, MOUSE, 137*

LEUKOCYTE
- REACTIVITY
  - LEUKEMIA CELLS, HUMAN, 519*
- SURFACE CHANGES
  - PLANT LECTINS, HUMAN LEUKEMIC CELLS, 488*

LIPID
- SV40-TRANSFORMED CELL
  - CELL GROWTH, MOUSE, 224

LIPOSARCOMA
- ULTRASTRUCTURE
  - CASE REPORTS, 547*

LIVER
- BERYLLIUM
  - ULTRASTRUCTURAL CHANGES, RAT, 77
- CANCER INCIDENCE
  - ENGLAND, 575
  - TOKYO, 575
- CIRRHOSIS INCIDENCE
  - ENGLAND, 575
  - TOKYO, 575
- HEPATOCARCINOGENESIS
  - NITROSOMORPHOLINE, RAT, 115
- HEPATOCELLULAR CARCINOMA
  - DIETHYLNITROSAMINE, RAT, 109
- HEPATOMA
  - AFLATOXIN B1, INFANT MICE, 139*
  - CELL PROLIFERATION, RAT, 148*
  - ENZYME PATTERN, RAT, 544
  - 1-HYDROXYSAFROLE, RAT, 171*
  - POLYCHLORINATED BIPHENYLS, MOUSE, 128*
  - RNA STAINING, CYTOPHOTOMETRIC MEASUREMENTS, 555*
  - RNA TRANSPORT ALTERATION, RAT, 170*
  - TRANSPLANTABILITY, MOUSE, 72
- HEPATOMA, MORRIS
  - ADENYLATE CHARGE CONTROL, 539
- HEPATOMA-SPECIFIC ANTIGENS
  - IMMUNITY, RAT, 378
- 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - AMP DEAMINASE INTERACTION, RAT, 53
- 3'-METHYL-DIMETHYLAMINOAZOBENZENE
  - RAT, 178*
- MICROSOMAL ALDRIN EPOXIDATION
  - BIPYRIDYLIUM COMPOUNDS, RAT, 126*
- MICROSOMAL CYTOCHROME P450
  - SUBSTRATE SPECIFICITIES, RAT, 118*
- MICROSOME
  - AFLATOXIN B1 METABOLISM, RAT, 104
- NITROSOMORPHOLINE-INDUCED HEPATOMA
  - RAT, 99
- NOVIKOFF ASCITES CELLS
  - ADENYLATE CHARGE CONTROL, 540
- PRIMARY CARCINOMA
  - AFLATOXIN B1, MONKEY, 122*
- RNA TRANSPORT
  - ALTERED REGULATION, RAT HEPATOMA CELLS, 587

LUNG
- BRONCOGENIC CARCINOMA
  - EXFOLIATIVE CYTOLOGY, RAT, HAMSTER, 557*
- 1,1-DIMETHYLHYDRAZINE
  - CARCINOGENICITY, MOUSE, 138*
- TUMOR IMMUNITY
  - TUMOR CELL INOCULATION ROUTE, MOUSE, 369

LUNG CANCER
- HUMAN, REVIEW, 19*
- IMMUNOGLOBULIN
  - BRONCHIAL SECRETIONS, HUMAN, 495*
- MORTALITY
  - OKLAHOMA, 562
- PLUTONIUM DIOXIDE
  - MONKEY, 203
- TOBACCO SMOKING
  - HUMAN, 59

LYMPH NODE
- ANTIGEN
  - CELL MIGRATION INHIBITION, HUMAN

    - LYMPHOMAS, 372
  - CHIMERISM
    - HERPESVIRUS SAIMIRI, MONKEY, 585
  - IMMUNOGLOBULIN SYNTHESIS
    - HUMAN, 432*
  - MULTIPLE LYMPHOBLASTOID LINE ESTABLISHMENT
    - HUMAN, 432*

MPH NODE CELL
  - ASCITES TUMOR
    - ELECTROPHORETIC MOBILITY, MOUSE, 536*
    - IMMUNITY, MOUSE, 535*

MPH NODE, REGIONAL
  - MAMMARY TUMOR
    - IMMUNITY, MOUSE, 444*

MPHATIC SYSTEM, REGIONAL
  - IMMUNE RESPONSE
    - GUINEA PIG, 380
  - TUMOR ALLOGRAFT IMMUNITY
    - GUINEA PIG, 533*

MPHOCYTE
  - ACTINOMYCIN BINDING CAPACITY
    - CHRONIC LYMPHOCYTIC LEUKEMIA PATIENTS, 437*
  - AUTOLOGOUS
    - CYTOTOXICITY, CULTURED HUMAN TUMORS, 485*
  - BLASTOGENESIS
    - L-ASPARAGINASE, CELL SURFACE, RAT, 520*
    - CELL-MEDIATED IMMUNITY, HUMAN, 469*
  - BLASTOGENIC RESPONSE
    - AUTOLOGOUS LEUKEMIA CELLS, HUMAN, 481*
  - COMPLEMENT RECEPTOR SITES
    - LEUKEMIA PATIENTS, 419
  - MAREK'S DISEASE
    - PATHOGENESIS, CHICKEN, 541
  - MELANOMA CELL DESTRUCTION
    - NEGRO, 433*
  - PROTEIN SYNTHESIS
    - CELL-MEDIATED IMMUNITY, GUINEA PIG, MOUSE, 498*
  - PYRIMIDINE BIOSYNTHESIS CONTROL
    - HUMAN, 438*
  - REACTIVITY
    - CANCER PATIENTS, 406
  - ROSETTE-FORMING ABILITY
    - LEUKEMIA PATIENTS, 419
  - SARCOMA INHIBITION
    - MOUSE, 25
  - SERUM
    - CYTOTOXICITY, HUMAN, 413
  - SERUM INHIBITORY EFFECT
    - AUTOLOGOUS LEUKEMIA CELLS, HUMAN, 481*
  - STIMULATION
    - EPSTEIN-BARR VIRUS, HUMAN, 238
  - SURFACE IMMUNOGLOBULIN
    - LEUKEMIA PATIENTS, 419
  - SURFACE IMMUNOGLOBULINS
    - LYMPHOPROLIFERATIVE DISEASES, CLINICAL STUDY, 405
  - TRANSFORMATION
    - IMMUNITY, LEUKEMIA ASSOCIATED ANTIGENS, HUMAN, 430*
    - PHYTOHEMAGGLUTININ, LEUKEMIC CHILDREN, 522*

LYMPHOMA
  - ANTIGENS, VIRUS-RELATED
    - MOUSE, 409
  - CELL MIGRATION INHIBITION
    - LYMPH NODE ANTIGENS, HUMAN, 372
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - SUPPRESSION, MOUSE, 65
    - SPLENECTOMY, MOUSE, 187*
  - IMMUNE COMPLEX DISEASE
    - ONCORNAVIRUS RELATED ANTIGEN, HUMAN, 468*
  - MOLONEY VIRUS-INDUCED
    - IMMUNE SERA, MOUSE, 236
  - SUPPRESSION, ANTIBODY-MEDIATED
    - MACROPHAGE PARTICIPATION, MOUSE, 355
  - VIRUS, FRIEND LEUKEMIA
    - ADOPTIVE CHEMOIMMUNOTHERAPY, MOUSE, 342*
  - VIRUS, MOLONEY
    - ALLOGENEIC CHEMOIMMUNOTHERAPY, MOUSE, 341*
  - VIRUS, RADIATION LEUKEMIA
    - FREUND'S ADJUVANT, MOUSE, 511*
  - VIRUS SYNTHESIS
    - RIBOSOMES, MOUSE, 347*

LYMPHOMA, MALIGNANT
  - HERPES ZOSTER
    - CLINICAL STUDY, 516*
  - HYPERINFECTION
    - S. STERCORALIS, CASE REPORTS, 530*

LYMPHOMAGENESIS
  - RADIATION, COBALT 60
    - THYMIC GRAFTS, MOUSE, 259

LYMPHOPROLIFERATIVE DISEASE
  - LYMPHOCYTE SURFACE IMMUNOGLOBULINS
    - CLINICAL STUDY, 405

LYMPHORETICULAR SYSTEM
  - BURKITT'S LYMPHOMA
    - REVIEW, 14*
  - DIPHENYL HYDANTOIN
    - MOUSE, 42
  - HODGKIN'S DISEASE
    - REVIEW, 14*

LYMPHOSARCOMA
  - SURFACE IMMUNOGLOBULINS
    - BOVINE CELLS, 399
  - VIRUS, FELINE LEUKEMIA
    - CAT, 247

LYSOLECITHIN
  - CELL DIVISION STIMULATION
    - PLASMA MEMBRANE, BACTERIA, 125*

MACROPHAGE
  - FIBROSARCOMAS
    - SYNGENEIC RAT, MOUSE, 422*
  - RECOGNITION FACTOR

    - LEUKEMIA, RAT, 373

MAMMARY DUCT
- PROLIFERATION
    - HISTOPATHOLOGY, ELDERLY, 598

MAMMARY GLAND
- CELLULAR COMPOSITION
    - PREMALIGNANT TISSUES, MOUSE, 552*
- PRENEOPLASTIC NODULES
    - DNA POLYMERASE ACTIVITY, MOUSE, 536
    - DNA SYNTHESIS, MOUSE, 538
- RNA SYNTHESIS
    - PRENEOPLASTIC NODULES, MOUSE, 554*

MAMMARY TUMOR
- BENIGN BREAST LESIONS
    - EPIDEMIOLOGY, CLINICAL STUDY, 576
- BODY FAT
    - RAT, 127*
- CELL AGGLUTINATION
    - SERUM, MOUSE, 354
- CELLULAR HYPERSENSITIVITY
    - HUMAN, 506*
- COLON CARCINOMA
    - FAMILIAL OCCURRENCE, UNITED STATES, 582
- CYCLOPHOSPHAMIDE
    - INCIDENCE, RAT, 112
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - RAT, 142*
    - SHOCK STRESS, RAT, 199*
    - ULTRASTRUCTURE, RAT, 95
    - SPLENECTOMY, MOUSE, 187*
- ENDOCRINE DYSFUNCTION
    - HUMAN, REVIEW, 23*
- ENDOCRINE PROFILES
    - CLINICAL STUDY, 593
- EPIDEMIOLOGY
    - REVIEW, 1
- ESTROGEN
    - PARITY, CLINICAL STUDY, 29
    - RAT, 87, 174*
- ESTROGEN METABOLISM
    - ORGAN CULTURE, HUMAN, 581
- ETIOLOGY
    - HUMAN, REVIEW, 6
- ETIOLOGY, VIRAL
    - HUMAN, REVIEW, 8
- GROWTH
    - HUMAN PLACENTAL LACTOGEN, RAT, 76
- HORMONAL FACTORS
    - MOUSE, 550*
- IMMUNITY
    - ANTIBODY-CELL INTERACTIONS, MOUSE, 505*
- IMMUNOGENICITY
    - GLYCOCALYX DISSOCIABILITY, 465*
    - METASTASIZING CAPACITY, 465*
    - SURFACE ANTIGEN SOLUBILITY, 465*
- INHIBITION BY ESTRIOL
    - RAT, 165*
- LYMPH NODE, REGIONAL
    - IMMUNITY, MOUSE, 444*
- MASON-PFIZER MONKEY VIRUS ANTIGENS
    - MONKEY, 464*
- MUSCLE ACTIVITY
    - RAT, 127*
- OVARIECTOMY
    - PORTACAVAL SHUNT, RAT, 185*
- PHYTOHEMAGGLUTININ
    - INCIDENCE, RAT, 112
- PROLACTIN
    - RAT, 87, 174*
- RADIATION, GAMMA
    - HUMAN, MOUSE, RAT, 213*
- RADIATION, X-RAY
    - HUMAN, MOUSE, RAT, 213*
- RECURRENCE RATE
    - TRYPTOPHAN METABOLISM, HUMAN, 584
- REGRESSION, HORMONE DEPENDENT
    - RAT, 176*
- THYMIDINE KINASE ACTIVITY
    - ESTROGEN, RAT, 123*
- THYMIDYLATE BIOSYNTHETIC ENZYMES
    - MOUSE, 553*
- THYMIDYLATE KINASE ACTIVITY
    - ESTROGEN, RAT, 123*
- VIRUS-INDUCED
    - HUMAN, REVIEW, 22*
- VIRUS, RNA
    - MOUSE, HUMAN, REVIEW, 16*

MAREK'S DISEASE
- IMMUNOSUPPRESSION
    - SERUM PROTEIN CHANGES, CHICKEN, 426*
- LYMPHOCYTE
    - PATHOGENESIS, CHICKEN, 541
- PLASMA MEMBRANE PROTEINS
    - TUMOR CELLS, CHICKEN, 512*

MAREK'S DISEASE TUMOR CELLS
- PLASMA MEMBRANE
    - PROTEIN, 512*

MELANOMA
- CELL DESTRUCTION
    - LYMPHOCYTES, NEGRO, 433*
- MELANOSOME
    - QUANTITATIVE STUDY, MOUSE, 599
- MITOCHONDRIA
    - QUANTITATIVE STUDY, MOUSE, 599
- REAGINIC ANTIBODY PRODUCTION
    - MOUSE, 411
- RNA SYNTHESIS INHIBITION
    - ETHIDIUM BROMIDE, HAMSTER, 313*

MELANOMA, MALIGNANT
- FAMILIAL INCIDENCE
    - AUSTRALIA, 573
- IMMUNITY
    - CLINICAL STUDY, 509*
- IMMUNITY, CELLULAR
    - HUMAN, 483*

MELANOSOME
- MELANOMA
    - QUANTITATIVE STUDY, MOUSE, 599

MEMBRANE
- POTENTIAL
    - OVARIAN TUMOR CELLS, HUMAN, 580

MESONEPHROMA
- OVARY
    - CLINICAL STUDY, 546*

- SOTHELICMA
  - ASBESTOS
    - ULTRASTRUCTURE, RAT, 108

- TABOLISM
  - AFLATOXIN B1
    - LIVER MICROSOME, RAT, 104
  - ARYL HYDROCARBON HYDROXYLASE
    - VITAMIN A, HAMSTER CELLS, 150*
  - CARBOHYDRATE
    - NEUROBLASTOMA CELLS, 596
  - CARCINOEMBRYONIC ANTIGEN, HUMAN
    - RABBIT, DOG, 401
  - ESTROGEN
    - CULTURED MAMMARY TUMORS, HUMAN, 581
  - HEXAMETHYLMELAMINE
    - HEPATIC MICROSOMES, RAT, HAMSTER, 145*
  - PHOSPHOLIPID
    - SV40-TRANSFORMED CELL, MOUSE, 231
  - RNA
    - URINARY BLADDER TUMOR, RAT, 97
  - TRYPTOPHAN
    - MAMMARY TUMOR RECURRENCE RATE, HUMAN, 584

- TALS
  - BERYLLIUM
    - ULTRASTRUCTURAL CHANGES, LIVER, RAT, 77
  - HAFNIUM
    - CHONDROMA, MOUSE, 64
  - ZIRCONIUM
    - CHONDROMA, MOUSE, 64

- TALS, TRACE
  - OSTEOSARCOMA
    - HUMAN, 47
  - STRONTIUM
    - METABOLIC EFFECTS, HYDRA, 155*

- -METHYL-4-DIMETHYLAMINOAZOBENZENE
  - ALPHA FETOPROTEIN
    - SERUM, RAT, 149*

- -METHYL-DIMETHYLAMINOAZOBENZENE
  - HEPATOMA
    - RNA TRANSPORT ALTERATION, RAT, 170*
  - LIVER
    - RAT, 178*

- -METHYL-4-DIMETHYLAMINOAZOBENZENE
  - AMP DEAMINASE INTERACTION
    - LIVER, RAT, 53
  - ALPHA FETOPROTEIN
    - LIVER, RAT, 389
    - SERUM, RAT, 149*

- THYL METHANESULFONATE
  - MUTAGENESIS
    - DNA, 119*

- THYLATION
  - MUTAGENESIS
    - DNA, 119*
  - RNA
    - DIMETHYLNITROSAMINE, LIVER, MOUSE, 86

- METHYLAZOXYMETHANOL ACETATE
  - ENZYMIC HYDROLYSIS
    - DNA SYNTHESIS INHIBITION, 162*

- 7-METHYLBENZ(A)ANTHRACENE
  - CARCINOGENESIS
    - MOUSE, 27

- METHYLCHOLANTHRENE
  - INDUCED TRANSPLANTED SARCOMA
    - HOST IMMUNITY, GUINEA PIG, 517*
  - SARCOMA
    - CELL PROLIFERATION PATTERNS, 182*
    - CELL PROLIFERATION, RAT, 61
    - ISOTOPIC 13C/12C RATIO, RAT, 81

- 3-METHYLCHOLANTHRENE
  - FIBROSARCOMA
    - IMMUNITY, GROWTH, MOUSE, 461*
  - DISTRIBUTION
    - MACROAUTORADIOGRAPHIC STUDY, MOUSE, 82
  - DNA SYNTHESIS
    - MAMMARY NODULE, MOUSE, 129*
  - HEXAMETHYLMELAMINE METABOLISM
    - RAT, HAMSTER, 145*
  - LEUKEMIA
    - SPLENECTOMY, MOUSE, 34
    - THYMECTOMY, MOUSE, 34
  - SARCOMA
    - INHIBITION, MOUSE, 25
  - TUMOR GROWTH
    - IMMUNODEPRESSION, MOUSE, 392
  - TUMOR INDUCTION
    - C-TYPE RNA VIRUS INFECTION, RAT, 80

- 20-METHYLCHOLANTHRENE
  - ELECTRON ACCEPTOR COMPLEX FORMATION
    - TEMPERATURE DEPENDENT KINETICS, 200*

- METHYLNITROSOUREA
  - BRAIN
    - ULTRASTRUCTURE, RAT, 41
  - PLATING EFFICIENCY
    - EMBRYO CELLS, MOUSE, 26

- N-METHYLNITROSOUREA
  - RESPIRATORY SYSTEM TUMORS
    - ULTRASTRUCTURE, RAT, 160*

- BETA2-MICROGLOBULIN
  - ISOLATION FROM URINE
    - KIDNEY TRANSPLANT PATIENTS, 439*
    - MULTIPLE MYELOMA PATIENTS, 439*
  - RADIOIMMUNOASSAY
    - CLINICAL STUDY, 435*

- MICROSOME
  - ALDRIN EPOXIDATION
    - BIPYRIDYLIUM COMPOUNDS, RAT LIVER, 126*

- MITOCHONDRIA
  - N-HYDROXY-ACETYLAMINOFLUORENE
    - CARCINOGENICITY, RAT, 159*

- N-HYDROXY-N-ACETYLAMINOFLUORENE
    - RAT, 54
  - MELANOMA
    - QUANTITATIVE STUDY, MOUSE, 599
  - RNA TUMOR VIRUS PRODUCTION
    - CHICK CELL, 287
    - MOUSE CELL, 287

- MITOSIS
  - INHIBITION
    - EPIDERMAL CELLS, HUMAN PSORIASIS LESIONS, 424*

- MORTALITY
  - LUNG CANCER
    - OKLAHOMA, 562
  - NEOPLASMS, MALIGNANT
    - STEEL WORKERS, UNITED STATES, 563

- MOUSE
  - RESISTANCE
    - ADENOVIRUS 12-INDUCED TUMORS, 479*

- MUCOPOLYSACCHARIDE
  - PLASMA MEMBRANE
    - VIRUSES, TRANSFORMED CELLS, 260

- MUTAGEN, CHEMICAL
  - DETECTION SYSTEM
    - MOUSE, 156*

- MUTAGENESIS
  - BENZO(A)PYRENE
    - DROSOPHILA, 70
  - DNA
    - ETHYL METHANESULFONATE, 119*
    - METHYL METHANESULFONATE, 119*

- MUTATION
  - NEUROBLASTOMA
    - PREDISPOSITION, HUMAN, 579
  - PHEOCHROMOCYTOMA
    - PREDISPOSITION, HUMAN, 579

- MYCOBACTERIUM BOVIS
  - TUMOR CELL GROWTH
    - MUSCLE, GUINEA PIG, 487*
  - TUMOR IMMUNITY
    - GUINEA PIG, 501*

- MYELOFIBROSIS
  - GRANULOCYTOPOIETIC STIMULANT
    - SERUM, HUMAN, 556*

- MYELOMA
  - DNA POLYMERASE
    - TEMPLATE SPECIFICITY, MOUSE, 578
  - IMMUNOGLOBULIN CONTENT
    - CELL CYCLE, MOUSE, 358

- MYELOMA CELL
  - ANTISERUM
    - HUMAN, 402

- MYELOMA, MULTIPLE
  - IMMUNOGLOBULIN IGG
    - ULTRASTRUCTURE, HUMAN, 357
  - BETA2-MICROGLOBULIN ISOLATION
    - URINE, PATIENTS, 439*

- MYOBLASTOMA
  - GRANULAR CELL
    - TISSUES, HUMAN, 546*

- BETA-NAPHTHYLAMINE
  - ASCORBIC ACID
    - URINARY BLADDER, RABBIT, 36

- ALPHA-NAPHTHYLISOTHIOCYANATE
  - HEPATOMA
    - HYPOPHYSECTOMY, RAT, 163*

- NASAL TUMORS
  - 3,4,5-TRIMETHOXYCINNAMALDEHYDE
    - RAT, 90

- NASOPHARYNGEAL CARCINOMA
  - INCIDENCE
    - KENYA, 565

- NEOPLASMS
  - TROPHOBLASTIC
    - HL-A ANTIGENS, CLINICAL STUDY, 531*

- NEOPLASMS, MALIGNANT
  - MORTALITY
    - STEEL WORKERS, UNITED STATES, 563
  - PSYCHOSOMATIC FACTORS
    - HUMAN, REVIEW, 21*

- NEOPLASTIC DISEASE
  - CHROMOSOME ABERRATIONS
    - HUMAN, REVIEW, 11
  - ETIOLOGY
    - HUMAN, REVIEW, 13

- NERVE TUMOR
  - ETHYLNITROSOUREA
    - ULTRASTRUCTURE, RAT, 30

- NERVOUS SYSTEM TUMORS
  - N-ETHYL-N-NITROSOUREA
    - MOUSE, 56
  - TRANSPLANTATION
    - RAT, 91

- NEURAMINIDASE
  - GLYCOPROTEIN SYNTHESIS
    - ASCITES TUMOR, MOUSE, 586

- NEUROBLASTOMA
  - CARBOHYDRATE METABOLISM
    - ENZYME ACTIVITIES, 596
  - MUTATION
    - PREDISPOSITION, HUMAN, 579

- NICKEL CARBONYL
  - CARCINOGENICITY
    - RAT, 169*

- N-(4(5-NITRO-2-FURYL)-2-THIAZOLYL) ACETAMIDE
  - LEUKEMOGENICITY
    - EFFECT OF P-HYDROXYACETANILIDE, MOUSE, 137*
    - EFFECT OF SODIUM SULFATE, MOUSE, 137*

-NITROQUINOLINE-1-OXIDE
- ALIPHATIC AMINO ACID INTERACTION, 101
- CHORIONIC TUMOR
  - RAT, 32
- HISTONE INTERACTION, 101
- TRANSFORMATION, MALIGNANT
  - ELECTROPHORETIC MOBILITY, RAT, 33
  - MOUSE, 52

ITROSAMINES
- FOOD
  - REVIEW, 9
- FORMATION
  - TERTIARY AMINES, NITRITE, 189*
  - TERTIARY AMINES, NITRITE, RAT, 85
- RESPIRATORY SYSTEM TUMORS
  - NURSING HAMSTERS, OFFSPRING, 31
- TRANSFORMATION, MALIGNANT
  - HAMSTER CELLS, 110

-NITROSO-BUTANOL-(4)-BUTYLAMINE
- URINARY BLADDER
  - CARCINOGENESIS, RAT, 107

-NITROSO-DIBUTYLAMINE
- URINARY BLADDER
  - CARCINOGENESIS, RAT, 107

ITROSODIMETHYLAMINE
- HEMANGIOMA
  - MOUSE, 50

-NITROSOHEXAMETHYLENEIMINE
- RESPIRATORY SYSTEM TUMORS
  - HAMSTER, MOUSE, 89

-NITROSOHEXAMETHYLENIMINE
- RESPIRATORY SYSTEM TUMORS
  - HAMSTER, 121*

ITROSOMETHYLUREA
- TRANSFORMATION, MALIGNANT
  - HAMSTER CELLS, 110

-NITROSOMETHYLUREA
- TUMOR INDUCTION
  - EPITHELIAL-LIKE CELLS, RAT LIVER, 151*

ITROSOMORPHOLINE
- HEPATOCARCINOGENESIS
  - RAT, 115
- HEPATOMA
  - RAT, 99

CCUPATIONAL HAZARD
- ASBESTOS
  - REVIEW, 17*
- MALIGNANT NEOPLASMS
  - MORTALITY, STEEL WORKERS, U.S., 563

STEOSARCOMA
- ETIOLOGY
  - HUMAN, 325*
- METALS, TRACE
  - HUMAN, 47
- PLUTONIUM
  - MOUSE, DOG, 205
- RADIUM
  - MOUSE, 208
  - MOUSE, DOG, 205
- STRONTIUM
  - MOUSE, DOG, 205

OVARY
- MESONEPHROMA
  - CLINICAL STUDY, 548*
- TUMOR CELL
  - MEMBRANE POTENTIAL, HUMAN, 580

PANCREATIN
- TRANSPLANTATION ANTIGENS
  - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

PAPAIN
- TRANSPLANTATION ANTIGENS
  - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

PAPILLOMA, SQUAMOUS
- VIRUS DETECTION
  - CHAFFINCH, 297*

PENIS
- TUMOR INCIDENCE
  - NETHERLANDS, 569

PHAGOCYTE
- PHYTOHEMAGGLUTININ
  - DNA SYNTHESIS, 425*

1,10-PHENANTHROLINE
- ETHIONINE CARCINOGENESIS
  - LIVER, RAT, 184*

PHENOBARBITAL
- HEXAMETHYLMELAMINE METABOLISM
  - RAT, HAMSTER, 145*

PHENOL
- CHORIOALLANTOIC MEMBRANE
  - FERTILE HEN'S EGG, 60

PHEOCHROMOCYTOMA
- MUTATION
  - PREDISPOSITION, HUMAN, 579

PHOSPHODIESTERASE
- CYCLIC AMP
  - LIVER, RAT, 542

PHOSPHOLIPID
- METABOLISM
  - SV40-TRANSFORMED CELL, MOUSE, 231

PHYTOHEMAGGLUTININ
- DNA SYNTHESIS
  - PHAGOCYTE, 425*
  - SPLEEN CELLS, LEUKEMIC MOUSE, 425*
- LYMPHOCYTE TRANSFORMATION
  - ACUTE LEUKEMIC CHILDREN, 522*
- MAMMARY TUMOR
  - INCIDENCE, RAT, 112
- VIRUS, FRIEND LEUKEMIA
  - FRIEND DISEASE IMMUNITY, MOUSE, 258

- VIRUS, RAUSCHER LEUKEMIA
  - MOUSE, 445*

PIPERAZINE
- SODIUM NITRITE
  - DOSE-RESPONSE STUDIES, MOUSE, 49

PITUITARY
- IMMUNOHISTOCHEMICAL STAINING
  - NEOPLASTICS LESIONS, MOUSE, RAT, 493*

PITUITARY GLAND
- DIETHYLSTILBESTROL DIPROPIONATE
  - MITOTIC INDEX, RAT, 73
- ESTROGEN
  - MITOTIC INDEX, RAT, 73

PITUITARY TUMOR
- TRANSPLANTATION
  - ULTRASTRUCTURE, MOUSE, 600

PLASMA
- AUTOLOGOUS LYMPHOCYTES
  - CYTOTOXICITY, CULTURED HUMAN TUMORS, 485*

PLASMA CELL TUMOR
- TRANSPLANTATION ANTIGENS
  - IMMUNITY, MOUSE, 365

PLASMA MEMBRANE
- MUCOPOLYSACCHARIDE
  - VIRUSES, TRANSFORMED CELLS, 260
- PROTEIN
  - CHICKEN LYMPHOID CELLS, 512*
  - MAREK'S DISEASE TUMOR CELLS, 512*
- TUMOR-SPECIFIC ANTIGEN LOCALIZATION
  - HUMAN CELLS, 353
- VIRUS-TRANSFORMED CELLS
  - CYTOCHEMISTRY, CHICKEN, 340*

PLASMACYTOMA
- BAYOL
  - ANTI-THYMOCYTE SERUM, MOUSE, 48
- IMMUNOGLOBULIN IGG ASSEMBLY
  - LIGHT-CHAIN POOL, MOUSE, 436*

PLUTONIUM
- OSTEOSARCOMA
  - MOUSE, DOG, 205

PLUTONIUM DIOXIDE
- LUNG CANCER
  - MONKEY, 203

POLLUTION, WATER
- TUMOR FREQUENCY
  - FISH, 69, 141*

POLYADENYLIC ACID
- VIRUS, MOLONEY LEUKEMIA
  - SYNTHESIS SUPPRESSION, 334*

POLYCHLORINATED BIPHENYLS
- HEPATOCARCINOGENICITY
  - MOUSE, 128*
- HEPATOMA
  - MOUSE, 128*

POLYINOSINIC:POLYCYTIDYLIC ACID
- AVIAN SARCOMA VIRUS
  - TUMOR TRANSPLANT IMMUNITY, RAT, HAMSTER, 228
- IMMUNITY, TUMOR-SPECIFIC
  - MOUSE, 460*
- ROUS SARCOMA VIRUS REPLICATION
  - CHICKEN CELLS, 220

PRASEODYMIUM 144
- CHROMOSOME ABERRATIONS
  - LIVER CELLS, HAMSTER, 212*

PROCHLORPERAZINE
- HEXAMETHYLMELAMINE METABOLISM
  - RAT, HAMSTER, 145*

PROLACTIN
- MAMMARY TUMOR
  - RAT, 87, 174*

BETA-PROPIOLACTONE
- DNA
  - SKIN, MOUSE, 193*

PROSTATE
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - HUMAN, 131*
- HYPERPLASTIC
  - ENZYME ACTIVITY, HUMAN, 549*
- PITUITARY TUMOR
  - ULTRASTRUCTURE, MOUSE, 600

PROTEIN
- FETAL, BETA MIGRATING
  - HUMAN TUMORS, 467*
- HUMAN IGG MYELOMA
  - ANTI-ALPHA2 MACROGLOBULIN ACTIVITY, 450*
- MONOCLONAL
  - INCIDENCE, MINNESOTA POPULATION, 429*
- NUCLEAR ACIDIC
  - SV40 INFECTION, MONKEY, HAMSTER, 239
- PLASMA MEMBRANE
  - CHICKEN LYMPHOID CELLS, 512*
  - MAREK'S DISEASE TUMOR CELLS, 512*
- POLYCYCLIC HYDROCARBON INCORPORATION
  - MECHANISMS, 152*
- POLYOMA VIRUS
  - MOUSE EMBRYO CULTURES, 251

PROTEIN, MYELOMA
- AFFINITY LABELING
  - NITROPHENYL LIGANDS, MOUSE, 521*

PROTEIN SYNTHESIS
- LYMPHOCYTE
  - CELL-MEDIATED IMMUNITY, GUINEA PIG, MOUSE, 498*

PURINE
- ONCOGENIC DERIVATIVES
  - REACTION BLOCKING BY METHYL SUBSTITUENTS, 180*

RADIATION
- ATOMIC BOMB SURVIVORS

    - LEUKEMIA INCIDENCE, JAPAN, 210*
    - LYMPHOMA INCICENCE, JAPAN, 210*
  - PLUTONIUM
    - OSTEOSARCOMA, MOUSE DOG, 205
  - RADIUM
    - OSTEOSARCOMA, MOUSE, 208
    - OSTEOSARCOMA, MOUSE DOG, 205
  - STRONTIUM
    - OSTEOSARCOMA, MOUSE DOG, 205
  - THYROID CARCINOMA
    - CASE REPORTS, 204

- ADIATION, CERIUM 144
  - CHROMOSOME ABERRATIONS
    - HAMSTER, 212*

- ADIATION, COBALT 60
  - VIRAL LYMPHOMAGENESIS
    - THYMIC GRAFTS, MOUSE, 259

- ADIATION, GAMMA
  - MAMMARY TUMOR
    - HUMAN, MOUSE, RAT, 213*

- ADIATION, PRASEODYMIUM 144
  - CHROMOSOME ABERRATIONS
    - HAMSTER, 212*

- ADIATION, UV
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 289
  - CARCINOGENESIS
    - CHOLESTEROL OXIDE, MOUSE, 207
  - DEFECTIVE SV40-ADENO 7 HYBRID
    - TRANSFORMATION POTENTIAL, HAMSTER, 310*
    - TUMOR INDUCTION, HAMSTER, 310*
  - VIRUS, HERPES SIMPLEX
    - ULTRASTRUCTURE, TRANSFORMED HAMSTER CELLS, 321*

- ADIATION, WHOLE-BODY
  - LEUKEMOGENIC VIRUS RELEASE
    - MOUSE, 217*

- ADIATION, X-RAY
  - CARCINOGENESIS
    - HUMAN, REVIEW, 10
    - TUMOR-RESISTANT MOUSE, 216*
  - CHROMOSOME ABERRATIONS
    - RAT, 209*
  - IMMUNE RESPONSE
    - TUMOR CELLS, MOUSE, 395
  - INTRAUTERINE
    - LEUKEMIA SUSCEPTIBILITY, CHILDREN, 206
  - LEUKEMIA
    - CHILDREN, 218*
  - MAMMARY TUMOR
    - HUMAN, MOUSE, RAT, 213*
  - THYROID CARCINOMA
    - ADULTS, 214*

- ADIUM
  - OSTEOSARCOMA
    - MOUSE, 208
    - MOUSE, DOG, 205

- ECTAL CARCINOMA
  - MUCOSAL GLYCOPROTEIN
    - IMMUNOHISTOLOGICAL STUDIES, HUMAN, 414

- RESISTANCE
  - ADENOVIRUS 12-INDUCED TUMORS
    - MOUSE, 479*
  - ASCITES TUMOR
    - MOUSE, 525*
  - 5-IODO-2'-DEOXYURIDINE
    - LYMPHOMA CELLS, 43
  - PARENTAL BONE MARROW GRAFTS
    - HYBRID MICE, 476*
  - TUMOR TRANSPLANTATION
    - SPLEEN, RAT, RABBIT, 534*

- RESPIRATORY SYSTEM
  - CARCINOGENESIS
    - INFECTION, RAT, 158*

- RESPIRATORY SYSTEM TUMORS
  - BENZO(A)PYRENE
    - HAMSTER, 67
    - ULTRASTRUCTURE, RAT, 160*
  - N-DIBUTYLNITROSAMINE
    - NURSING HAMSTERS, OFFSPRING, 31
  - N-DIETHYLNITROSAMINE
    - NURSING HAMSTERS, OFFSPRING, 31
  - FERRIC OXIDE
    - HAMSTER, 67
  - N-METHYLNITROSOUREA
    - ULTRASTRUCTURE, RAT, 160*
  - NITROSAMINES
    - NURSING HAMSTERS, OFFSPRING, 31
  - N-NITROSOHEXAMETHYLENEIMINE
    - HAMSTER, MOUSE, 89
  - N-NITROSOHEXAMETHYLENIMINE
    - HAMSTER, 121*

- RESPIRATORY SYSTEM, UPPER
  - TUMOR INCIDENCE
    - USSR, 568

- RETICULOSIS
  - VIRUS, C-TYPE PARTICLE
    - RAT, 315*

- RETICULUM CELL SARCOMA
  - SHEEP ERYTHROCYTE INOCULATION
    - MOUSE, 375

- RETINOIC ACID
  - TUMOR PROPHYLAXIS
    - SKIN, MOUSE, 57

- RIBONUCLEASE
  - SERUM LEVELS
    - TUMOR HISTOLOGY, CLINICAL STUDY, 595
  - VIRUS, MURINE LEUKEMIA, 331*

- RIBONUCLEOTIDE REDUCTASE
  - ASCITES TUMOR
    - STEREO SPECIFICITY, RAT, 597

- RIBOSOME
  - VIRUS SYNTHESIS
    - LYMPHOMA, MOUSE, 347*

RIFAMPICIN
- ADENOVIRUS 12 TUMOR DEVELOPMENT
  - HAMSTER, 309*

RNA
- DENSITY
  - SV40, MONKEY CELLS, 222
- METABOLISM
  - URINARY BLADDER TUMOR, RAT, 97
- METHYLATION
  - DIMETHYLNITROSAMINE, LIVER, MOUSE, 86
- ONCOGENIC VIRAL
  - TRANSLATION, E.COLI, 308*
- RD114-VIRUS
  - HYBRIDIZATION, HUMAN CHROMOSOMES, 283
- STAINING
  - HEPATOMAS, CYTOPHOTOMETRIC MEASUREMENTS, 555*
- TRANSPORT
  - ALTERED REGULATION, RAT HEPATOMA CELLS, 587
- TRANSPORT ALTERATION
  - HEPATOMA, RAT, 170*
- VIRUS, AVIAN SARCOMA
  - CHICKEN EMBRYO CELLS, 280

RNA, MESSENGER
- EUKARYOTIC
  - TRANSLATION, E.COLI, 308*

RNA REPLICATION
- VIRUS, ROUS SARCOMA
  - CHICK CELLS, 240

RNA SYNTHESIS
- INHIBITION BY ETHIDIUM BROMIDE
  - MELANOMA, HAMSTER, 313*
- MAMMARY GLAND
  - PRENEOPLASTIC NODULES, MOUSE, 554*

RNA TRANSCRIPTION
- DNA POLYMERASE
  - E.COLI, 263
  - VIRUS, ROUS SARCOMA, 263

RNA, TRANSFER
- N-ACETOXY-2-ACETYLAMINOFLUORENE
  - E.COLI, 39
- POLYAMINE BINDING
  - MOUSE LEUKEMIC CELLS, E.COLI, 551*

SARCOMA
- DIETHYLAMINOETHYL-DEXTRAN
  - MOUSE, 62
- METHYLCHOLANTHRENE
  - CELL PROLIFERATION PATTERNS, 182*
  - CELL PROLIFERATION, RAT, 61
  - ISOTOPIC 13C/12C RATIO, RAT, 81
- 3-METHYLCHOLANTHRENE
  - INHIBITION, MOUSE, 25
- MOLONEY VIRUS-INDUCED
  - IMMUNE SERA, MOUSE, 236

SCAR
- POST-BURN CARCINOMA
  - CASE REPORTS, 211*
- SMALLPOX VACCINATION
  - MALIGNANCY, CASE REPORT, 307*

SEROTONIN
- RADIATION PROTECTIVE EFFECT
  - CHROMOSOME ABERRATIONS, RAT, 209*

SERUM
- GRANULOCYTOPOIETIC STIMULANT
  - MYELOFIBROSIS PATIENTS, 556*
- HUMORAL RECOGNITION FACTOR
  - CARCINOMA PATIENTS, 371
- HYALURONIDASE INHIBITOR
  - CANCER PATIENTS, 529*
- IMMUNOGLOBULIN LEVELS
  - LYMPHOBLASTIC LEUKEMIA PATIENTS, CHILDREN, 408
- INFECTIOUS MONONUCLEOSIS
  - MURINE LYMPHOMA CELLS, 397
- LYMPHOCYTE
  - CYTOTOXICITY, HUMAN, 413
- MONOCLONAL IGA AND IGM
  - IMMUNOFLUORESCENT IDENTIFICATION, CASE REPORT, 448*
  - LIGHT-CHAIN IDENTIFICATION, CASE REPORT, 449*
- PHYSIOLOGIC SIMILARITIES
  - PREGNANT AND TUMOR-BEARING ANIMALS, 503*
- RIBONUCLEASE
  - TUMOR HISTOLOGY, CLINICAL STUDY, 595
- SEROLOGIC SIMILARITIES
  - PREGNANT AND TUMOR-BEARING ANIMALS, 503*
- TUMOR-BEARING MICE
  - CYTOTOXICITY, LYMPH NODE CELLS, 388

SIALIC ACID
- GLYCOSYLTRANSFERASE ACTIVITY
  - POLYOMA VIRUS-TRANSFORMED HAMSTER CELLS, 299*
  - SARCOMA VIRUS-TRANSFORMED HAMSTER CELLS, 299*
  - SV40-TRANSFORMED MOUSE CELLS, 299*

SKIN
- ALLOGRAFT REACTION
  - MOUSE, 532*

SKIN CANCER
- INCIDENCE
  - USSR, 561

SKIN TUMOR
- ARYL HYDROCARBON HYDROXYLASE
  - MOUSE, 96
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - MOUSE, 96
- EPOXIDE HYDRASE
  - MOUSE, 96
- ETHYLPHENYLPROPIOLATE
  - MOUSE, 134*
- TOBACCO SMOKE CONDENSATE
  - MOUSE, 111
- URETHANE
  - DNA SYNTHESIS, MOUSE, 93

SODIUM NITRITE

PIPERAZINE
- DOSE-RESPONSE STUDIES, MOUSE, 49

DIUM OLEATE
- CELL DIVISION STIMULATION
  - PLASMA MEMBRANE, BACTERIA, 125*

DIUM TETRAPHENYL BORON
- TRANSPLANTATION ANTIGENS
  - SV40 INDUCED TUMOR CELLS, HAMSTER, 504*

LEEN
- TUMOR TRANSPLANTATION RESISTANCE
  - RAT, RABBIT, 534*

UAMOUS CELL CARCINOMA
- BENZO(A)PYRENE
  - RAT, 177*
- CERVIX
  - IMMUNOLOGY, CLINICAL STUDY, 403
- FERRIC OXIDE
  - RAT, 177*
- STOMACH
  - ACID BURNS, CASE REPORTS, 198*

ILBESTROL
- UROGENITAL TRACT CARCINOMA
  - INCIDENCE, ADOLESCENTS, 120*

OMACH
- GASTRIC CANCER ANTIGEN
  - HUMAN, 360
- GASTRIC CARCINOMA
  - TOBACCO SMOKING, CLINICAL STUDY, 102
- SQUAMOUS CELL CARCINOMA
  - ACID BURNS, CASE REPORTS, 198*

RONTIUM
- METABOLIC EFFECTS
  - HYDRA, 155*
- OSTEOSARCOMA
  - MOUSE, DOG, 205

RATOGENESIS
- 7-HYDROXYMETHYL-12-METHYLBENZ(A) ANTHRACENE
  - CELL DEATH, RAT, 28
- URETHANE
  - MOUSE, 114

-O-TETRADECANOYL-PHORBOL-13-ACETATE
- HISTONE PHOSPHORYLATION
  - MOUSE, 55

TRAHYDRODESOXOAFLATOXIN B1
- DISTRIBUTION
  - LIVER CELLS, CHICK EMBRYO, 83

IOSEMICARBAZONE
- VIRUS, ROUS SARCOMA
  - DNA POLYMERASE INHIBITION, 300*

YMOCYTE
- GLUCOCORTICOID RECEPTORS
  - RAT, MOUSE, 75

MOMA
- IMMUNOGLOBULIN SYNTHESIS
  - MOUSE, 396

THYROID CARCINOMA
- RADIATION
  - CASE REPORTS, 204
- RADIATION, X-RAY
  - ADULTS, 214*

TOBACCO
- CIGARETTE SMOKE
  - FIBROBLASTIC LUNG CELLS, HUMAN, 161*
- CIGARETTES
  - CHARCOAL ADSORPTIVE CAPACITY, 136*
  - CHARCOAL DETERIORATION, 136*
  - EHRLICH ASCITES CELL FERMENTATION TEST, 136*
  - WATER ELUTION TEST, 136*
- MARIJUANA SMOKE
  - FIBROBLASTIC LUNG CELLS, HUMAN, 161*
- SUGAR CONTENT, SMOKE ACIDITY
  - LUNG CANCER MORTALITY, HUMAN, 59

TOBACCO SMOKE CONDENSATE
- CHORIOALLANTOIC MEMBRANE
  - FERTILE HEN'S EGG, 60
- SKIN TUMOR
  - MOUSE, 111

TOBACCO SMOKING
- GASTRIC CARCINOMA
  - CLINICAL STUDY, 102

TRANSFORMATION
- ADENOVIRUS
  - HAMSTER CELLS, 289
- DEFECTIVE SV40-ADENO 7 HYBRID
  - UV RADIATION, HAMSTER, 310*
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - FELINE EMBRYO CELLS, 157*
- FIBROBLAST
  - TUMOR VIRUSES, REVIEW, 2
- HUMAN DIPLOID CELLS
  - 2,4-DINITROPHENOL, 235
  - LEUKEMIA-LIKE VIRUS, 235
- LYMPHOCYTE
  - IMMUNITY, LEUKEMIA ASSOCIATED ANTIGENS, HUMAN, 430*
  - PHYTOHEMAGGLUTININ, LEUKEMIC CHILDREN, 522*
- SPLEEN CELLS
  - FRIEND LEUKEMIA VIRUS INFECTION, MOUSE, 245
- SV40 TRANSFORMED MOUSE FIBROBLASTS
  - VIRUS, MURINE SARCOMA, 274
- TRANSFORMED STATE MAINTENANCE
  - VIRUS, POLYOMA, 273
  - VIRUS, SV40, 273
- UV-IRRADIATED HERPES VIRUS
  - ULTRASTRUCTURE, HAMSTER CELLS, 321*
- VIRUS
  - CARBOHYDRATE CONTENT, HAMSTER, CHICK CELLS, 230
- VIRUS, ADENO 12
  - HORMONAL INFLUENCE, HAMSTER CELLS, 317*

- VIRUS, ADENO SA7
  - POLYCYCLIC HYDROCARBONS, HAMSTER CELLS, 329*
- VIRUS, AVIAN SARCOMA
  - LIVER CELLS, RAT, 275
- VIRUS, EPSTEIN-BARR
  - INFECTIVITY, HUMAN CELL, MONKEY CELL, 285
- VIRUS, FELINE SARCOMA
  - MAMMALIAN CELL CULTURES, 320*
- VIRUS, HERPES SIMPLEX
  - HAMSTER CELL LINES, 318*
- VIRUS, MASON-PFIZER MONKEY
  - RHESUS CELLS, 338*
- VIRUS, MURINE LEUKEMIA
  - FOCUS ASSAY, MOUSE CELLS, 234
- VIRUS, MURINE SARCOMA
  - LIVER CELLS, RAT, 275

TRANSFORMATION, MALIGNANT
- DIMETHYLNITROSAMINE
  - HAMSTER CELLS, 110
- FLUORENYLHYDROXAMIC ACID
  - FIBROBLASTS, RAT, 183*
- 4-NITROQUINOLINE-1-OXIDE
  - ELECTROPHORETIC MOBILITY, RAT, 33
  - MOUSE, 52
- NITROSOMETHYLUREA
  - HAMSTER CELLS, 110

TRANSPLACENTAL CARCINOGENESIS
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - RAT, 116*
- URETHANE
  - MOUSE, 114

TRANSPLANTATION
- ANTIGEN
  - IMMUNE RESPONSE, SOLID MURINE TUMORS, 391
  - MODIFICATION, HUMAN TUMOR CELLS, 527*
  - PLASMA CELL TUMOR, MOUSE, 365
  - POLYOMA VIRUS, HYBRID MOUSE CELLS, 361
- ETHYLNITROSOUREA-INDUCED HEPATOMA
  - MOUSE, 72
- HEPATOMA CELLS, RAT
  - HYDROCORTISONE TREATED HAMSTERS, 466*
  - X-IRRADIATED HAMSTERS, 466*
- HERPESVIRUS SAIMIRI-INFECTED TUMOR TISSUES
  - MONKEY, 256
- IMMUNITY
  - SV40-TRANSFORMED CELLS, MOUSE, 359, 368
- NERVOUS SYSTEM TUMORS
  - RAT, 91
- PITUITARY TUMOR
  - ULTRASTRUCTURE, MOUSE, 600

3,4,5-TRIMETHOXYCINNAMALDEHYDE
- NASAL TUMORS
  - RAT, 90

TRYPTOPHAN
- METABOLISM
  - MAMMARY TUMOR RECURRENCE RATE, HUMAN, 584

TUBERCULOSIS
- GLIOMA, CEREBRAL
  - ASSOCIATION, CLINICAL STUDY, 442*

TUMOR VIRUS
- SEE VIRUS

URETHANE
- SKIN TUMOR
  - DNA SYNTHESIS, MOUSE, 93
- TERATOGENESIS
  - MOUSE, 114
- TRANSPLACENTAL CARCINOGENESIS
  - MOUSE, 114

URINARY BLADDER
- BETA-NAPHTHYLAMINE
  - ASCORBIC ACID, RABBIT, 36
- N-NITROSO-BUTANOL-(4)-BUTYLAMINE
  - CARCINOGENESIS, RAT, 107
- N-NITROSO-DIBUTYLAMINE
  - CARCINOGENESIS, RAT, 107

URINARY BLADDER TUMOR
- 2-ACETYLAMINOFLUORENE
  - HAMSTER, 63
- 2-ACETYLAMINOFLUORENE/INDOLE ADMINISTRATION
  - HAMSTER, 168*
- ANTIGEN
  - MOUSE, RAT, 458*
- BUTANOL-BUTYLNITROSAMINE
  - RAT, 98
- CYCLAMATE
  - RAT, 98, 167*
- DIBUTYLNITROSAMINE
  - ANGIOGRAPHY, RAT, 117*
  - RNA METABOLISM, RAT, 97
- 3,2'-DIMETHYL-4-AMINO-DIPHENYL
  - HAMSTER, 164*
- IMMUNOLOGIC CLASSIFICATION
  - HUMAN, 486*
- INCIDENCE
  - YUGOSLAVIA, 564

UROGENITAL TRACT CARCINOMA
- STILBESTROL
  - INCIDENCE, ADOLESCENTS, 120*

VINBLASTINE
- CELL PROLIFERATION
  - BONE MARROW, RAT, 46

VIRUS
- ADENOVIRUS 2-SV40 HYBRID
  - DNA TRANSCRIPTION-CONTROL REGION, 243
- DEFECTIVE SV40-ADENO 7 HYBRID
  - TRANSFORMATION POTENTIAL, HAMSTER, 310*
  - TUMOR INDUCTION, HAMSTER, 310*
- LEUKEMOGENIC VIRUS RELEASE
  - WHOLE-BODY IRRADIATION, MOUSE, 217*
- TRANSFORMATION
  - CARBOHYDRATE CONTENT, HAMSTER, CHICK CELLS, 230

RUS, ADENO
  TRANSFORMATION
    HAMSTER CELLS, 289

RUS, ADENO 12
  DIFFERENTIAL HYPOTHERMIA
    TUMOR CELLS, ULTRASTRUCTURE, HAMSTER, 277
  INDUCED TUMORS
    RESISTANCE, MOUSE, 479*
  TRANSFORMATION
    HORMONAL INFLUENCE, HAMSTER CELLS, 317*
  TUMOR DEVELOPMENT
    RIFAMPICIN, HAMSTER, 309*

RUS, ADENO-ASSOCIATED
  HERPESVIRUS SAIMIRI
    LYMPHOID CELLS, MONKEY, 291*

RUS, ADENO MONKEY
  ONCOGENICITY
    MOUSE, RAT, 219

RUS, ADENO SA7
  TRANSFORMATION
    POLYCYCLIC HYDROCARBONS, HAMSTER CELLS, 329*

RUS, AVIAN LEUKOSIS
  ANTIBODY
    IMMUNOGLOBULIN ANALYSIS, CHICKEN, 412
  ANTIGEN
    ASCITES SARCOMA CELLS, MOUSE, 415

RUS, AVIAN MYELOBLASTOSIS
  LOCALIZATION OF CONSTITUENTS, 339*
  RIBOSOME-LIKE PARTICLES
    ULTRASTRUCTURE, 281

RUS, AVIAN SARCOMA
  ANTIGEN
    ASCITES SARCOMA CELLS, MOUSE, 415
  DNA SYNTHESIS INHIBITION
    ACTINOMYCIN C, 346*
  ENVELOPE ANTIGENIC PROPERTIES
    CHICK EMBRYO FIBROBLASTS, 226
  HOST RANGE
    CHICK EMBRYO FIBROBLASTS, 226
  INTERFERENCE
    CHICK EMBRYO FIBROBLASTS, 226
  MORPHOLOGICAL REVERTANT
    TRANSFORMED MOUSE CELL, 286
  RNA
    CHICKEN EMBRYO CELLS, 280
  SARCOMA FORMATION INHIBITION
    ACTINOMYCIN C, 346*
  TRANSFORMATION
    LIVER CELLS, RAT, 275
  TUMOR TRANSPLANT IMMUNITY
    POLY I:C, RAT, HAMSTER, 228
  TUMORIGENICITY
    RODENTS, 227

US, C-TYPE
  3-METHYLCHOLANTHRENE-INDUCED TUMORS
    RAT, 80

VIRUS, C-TYPE PARTICLE
  CELL-VIRUS INTERACTION
    ULTRASTRUCTURE, REPTILIAN CELL LINE, 301*
  LEUKEMIA
    HAMSTER, 315*
  LYMPHOID CELL LINES
    LEUKMEIC CATTLE, 316*
  RETICULOSIS
    RAT, 315*

VIRUS, C-TYPE RNA
  ACTIVATION
    TRANSFORMED KIDNEY CELLS, RAT, 248

VIRUS, CV
  ANTIGEN
    CANCER PATIENT SERA, 523*

VIRUS, EPSTEIN-BARR
  ANTIBODY
    CELLULAR IMMUNITY, HODGKIN'S DISEASE PATIENTS, 387
    CELLULAR IMMUNITY, LEUKEMIA PATIENTS, 387
  ANTIGEN
    HEAT STABILITY, 423*
    LYMPHOBLASTOID CELL LINES, HUMAN, 362
  GUILLAIN-BARRE SYNDROME
    CLINICAL STUDY, 314*
  INFECTION
    INCIDENCE, BRAZIL, 264
    QUANTITATIVE ASPECTS, 351*
  INFECTION, LYMPHOBLASTOID CELL LINES
    ANTIGEN EXPRESSION, 252
    COLONY FORMATION, 252
  LYMPHOCYTE STIMULATION
    HUMAN, 238
  TRANSFORMATION
    INFECTIVITY, HUMAN CELL, MONKEY CELL, 285

VIRUS, FELINE LEUKEMIA
  HOST RANGE
    CAT, HUMAN, 242
  LYMPHOSARCOMA
    CAT, 247

VIRUS, FELINE SARCOMA
  INFECTION
    MAMMALIAN CELL CULTURES, 320*
  TRANSFORMATION
    MAMMALIAN CELL CULTURES, 320*

VIRUS, FRIEND LEUKEMIA
  ANTISERUM
    LEUKEMOGENIC POTENTIAL, MOUSE, 333*
  CONCANAVALIN A
    FRIEND DISEASE IMMUNITY, MOUSE, 258
  DEFECTIVE
    PATHOLOGIC PROPERTIES, MOUSE, 322*
  ERYTHROID DIFFERENTIATION INHIBITION
    BROMODEOXYURIDINE, MOUSE CELL, 330*
  ERYTHROPOIETIN
    POLYCYTHEMIC MICE, 324*

- HEMOGLOBIN SYNTHESIS INHIBITION
  - BROMODEOXYURIDINE, MOUSE CELL, 330*
- INFECTION
  - SPLEEN CELL TRANSFORMATION, MOUSE, 245
  - SPLEEN CELLS, MOUSE, 304*
- LYMPHOMA
  - ADOPTIVE CHEMOIMMUNOTHERAPY, MOUSE, 342*
- PHYTOHEMAGGLUTININ
  - FRIEND DISEASE IMMUNITY, MOUSE, 258
- PLANT LECTINS
  - FRIEND DISEASE IMMUNITY, MOUSE, 258
- SPLEEN FOCUS-FORMING
  - ANTIGEN ALTERATION, HUMAN LEUKEMIC TISSUE, 478*
- TRANSFORMED CELL
  - GLYCOLIPID CONTENT, MOUSE, 232

VIRUS, GRAFFI
- ANTINUCLEAR ANTIBODY INDUCTION
  - MOUSE, 367

VIRUS, GROSS LEUKEMIA
- ANTIGEN
  - IMMUNOSUPPRESSION, RAT, 502*
- SOLUBLE ANTIGENS
  - PLASMA, RAT, 276
  - TUMOR CELLS, MOUSE, 295*

VIRUS, HERPES-LIKE
- INFECTION
  - THYMUS CELLS, GUINEA PIG, 290*

VIRUS, HERPES SAIMIRI
- ADENOVIRUS-ASSOCIATED VIRUS
  - LYMPHOID CELLS, MONKEY, 291*
- INFECTION
  - TRANSPLANTATION, MONKEY, 256

VIRUS, HERPES SIMPLEX
- CHROMOSOME ABERRATIONS
  - CELL CYCLE ANALYSIS, HUMAN, 350*
- CYTOSINE ARABINOSIDE
  - KIDNEY CELLS, RABBIT, HUMAN, 292*
- DNA TRANSCRIPTION
  - CERVICAL CANCER TISSUE, HUMAN, 221
- INFECTION
  - DEOXYCYTIDINE KINASE ACTIVITY, 337*
- RADIATION, UV
  - ULTRASTRUCTURE, TRANSFORMED HAMSTER CELLS, 321*
- TRANSFORMATION
  - HAMSTER CELL LINES, 318*

VIRUS, IGUANA HERPES
- DENSE MATERIAL
  - INTRA-ENVELOPE INCORPORATION, 326*

VIRUS, LEUKEMIA
- ISOLATION
  - HUMAN CELL CULTURE, 305*

VIRUS, MAMMARY TUMOR
- ANTIGEN
  - MOUSE MAMMARY TUMOR CELL CULTURE, 514*
- BIOLOGICAL CHARACTERISTICS
  - MOUSE, 268
- PHYSICAL CHARACTERISTICS
  - MOUSE, 268

VIRUS, MAREK'S DISEASE
- INFECTION
  - SUSCEPTIBILITY, HAMSTER CELL CULTURES, 267

VIRUS, MASON-PFIZER MONKEY
- ANTIGEN
  - MAMMARY TUMOR, MONKEY, 464*
- TRANSFORMATION
  - RHESUS CELLS, 338*

VIRUS, MOLONEY
- LYMPHOMA
  - ALLOGENEIC CHEMOIMMUNOTHERAPY, MOUSE, 341*

VIRUS, MOLONEY LEUKEMIA
- H-2 ANTIGENS
  - LYMPHOMA, MOUSE, 377
- CELL-SURFACE ANTIGENS
  - LYMPHOMA, MOUSE, 377
- COMPUTER ANALYSIS
  - INFECTED CELLS, RAT, 229
- POLYADENYLIC ACID
  - SYNTHESIS SUPPRESSION, 334*

VIRUS, MOLONEY SARCOMA
- HISTONE
  - ONCOGENICITY STIMULATION, MOUSE, 306*
- TUMOR REGRESSION
  - MOUSE, 474*

VIRUS, MURINE LEUKEMIA
- ANTIGEN
  - MURINE SARCOMA VIRUS-TRANSFORMED CELLS, 233
- CELL POPULATION CHANGES
  - BONE MARROW, MOUSE, 335*
- DNA POLYMERASE INHIBITION
  - METHYLATED POLYNUCLEOTIDES, MOUSE CELLS, 323*
- RELEASE FROM SYNCHRONIZED CELLS
  - MOUSE, RAT, 296*
- REPLICATION
  - RAT CELL, 246
  - TRANSFORMED MOUSE CELLS, 250
- RIBONUCLEASE ACTIVITY, 331*
- SYNTHESIS
  - CELL CULTURE, 266
- TRANSFORMATION
  - FOCUS ASSAY, MOUSE CELLS, 234

VIRUS, MURINE SARCOMA
- ANTISERUM
  - ANTIGEN, RAT, 364
- DEFECTIVE
  - PROPERTIES, MOUSE, HAMSTER, 345*
- GENOME
  - ULTRASTRUCTURE, RAT CELL LINE, 278
- HOST RANGE SHIFT
  - HUMAN CELLS, 282

- HUMORAL CYTOTOXICITY TECHNIQUE
    - MOUSE, 327*
- LYMPHOCYTE CYTOTOXICITY TECHNIQUE
    - MOUSE, 327*
- MORPHOLOGICAL REVERTANT
    - TRANSFORMED MOUSE CELL, 286
- PSEUDOTYPE, HUMAN CELL-ADAPTED
    - PRODUCTION, CHARACTERIZATION, 255
- RELEASE FROM SYNCHRONIZED CELLS
    - MOUSE, RAT, 296*
- REPLICATION
    - HUMAN CELLS, 282
- TRANSFORMATION
    - LIVER CELLS, RAT, 275
    - SV40 TRANSFORMED MOUSE FIBROBLASTS, 274
- TRANSFORMED CELLS
    - SURFACE ANTIGENS, MOUSE, 407

RUS, NEW CASTLE DISEASE
- IMMUNE RESPONSE
    - TUMOR CELLS, MOUSE, 395

RUS, ONCORNA
- LARYNGEAL CARCINOMA CELLS
    - HUMAN, 241

RUS, PAPOVA
- INDUCED TUMORS
    - FETAL ANTIGENS, MOUSE, 507*
    - TUMOR SPECIFIC CELL SURFACE ANTIGENS, MOUSE, 507*

RUS, PARAMYXO
- INFECTION
    - TRANSPLANTIBILITY, HAMSTER CELLS, 453*

US, PICORNA
- MALIGNANT ADENOPATHY
    - HUMAN, 269

US, POLYOMA
- T ANTIGEN SYNTHESIS
    - EMBRYO CELL CULTURE, MOUSE, 410
- ANTISERUM
    - ANTIGEN, RAT, 364
- BASIC PROTEINS
    - MOUSE EMBRYO CULTURES, 251
- DNA REPLICATION
    - FIBROBLASTS, MOUSE, 271
- DNA REPLICATION, SEMI-CONSERVATIVE
    - FIBROBLASTS, MOUSE, 272
- PROTECTIVE POWER
    - TRANSFORMED ALLOGENIC CELLS, MOUSE, 303*
- SYNTHESIS INHIBITION
    - SENDAI VIRUS, 311*
    - SENDAI VIRUS INTERFERENCE MECHANISM, 312*
- TRANSFORMED STATE MAINTENANCE
    - WORKING HYPOTHESIS, 273
- TRANSPLANTATION ANTIGEN
    - HYBRID MOUSE CELLS, 361

US, PROGRESSIVE PNEUMONIA
- ROUS SARCOMA VIRUS
    - COMPARATIVE STUDY, ULTRASTRUCTURE, 257

VIRUS, RADIATION LEUKEMIA
- LYMPHOMA
    - FREUND'S ADJUVANT, MOUSE, 511*

VIRUS, RAUSCHER LEUKEMIA
- ANTINUCLEAR ANTIBODY INDUCTION
    - MOUSE, 367
- DNA POLYMERASE
    - ACTIVE-SITE-DIRECTED INHIBITORS, 349*
- ERYTHROLEUKEMIA
    - CULTURE LINE ESTABLISHMENT, MOUSE, 348*
- FREUND'S ADJUVANT
    - POTENTIATION, MOUSE, 366
- INFECTION
    - COLONY FORMING UNITS, MOUSE, 288
    - RADIORESISTANCE, MOUSE, 288
    - SPLEEN CELLS, MOUSE, 304*
- LOW, HIGH LEUKEMOGENIC VARIANTS
    - INTERFERENCE, MOUSE, 262
- PHYTOHEMAGGLUTININ
    - MOUSE, 445*
- PRODUCTION
    - MITOCHONDRIA, CHICK CELL, 287
    - MITOCHONDRIA, MOUSE CELL, 287
- REPLICATION
    - GRANULOPOIETIC CELLS, MOUSE, 302*

VIRUS, RNA
- MAMMARY TUMOR
    - MOUSE, HUMAN, REVIEW, 16*

VIRUS, RNA TUMOR
- IMMUNE RESPONSE
    - MOUSE, 463*

VIRUS, ROUS SARCOMA
- 5-AZACYTIDINE
    - TRANSFORMED CELLS, HAMSTER, 225
- BRAIN TUMOR
    - MORPHOLOGY, MOUSE, 237
- CLONAL ANALYSIS
    - RAT, 223
- DNA POLYMERASE
    - RNA TRANSCRIPTION, 263
- DNA POLYMERASE INHIBITION
    - THIOSEMICARBAZONE, 300*
- GENOME
    - ULTRASTRUCTURE, RAT CELL LINE, 278
- IMMUNITY
    - AVIAN LEUKOSIS VIRUSES, CHICKEN, 471*
- PRODUCTION
    - MITOCHONDRIA, CHICK CELL, 287
    - MITOCHONDRIA, MOUSE CELL, 287
- PROGRESSIVE PNEUMONIA VIRUS
    - COMPARATIVE STUDY, ULTRASTRUCTURE, 257
- REPLICATION
    - POLY I:C, CHICKEN CELLS, 220
- RNA REPLICATION
    - CHICK CELLS, 240
- TRANSFORMED RAT CELL LINE
    - CONTACT INHIBITION, 253
    - LOCOMOTION, 253
    - MORPHOLOGY, 253
- TUMOR INDUCTION
    - MOUSE, 270

- VISNA VIRUS
  - COMPARATIVE STUDY, ULTRASTRUCTURE, 257

- VIRUS, SENDAI
  - POLYOMA VIRUS SYNTHESIS INHIBITION, 311*
    - INTERFERENCE MECHANISM, 312*

- VIRUS, SHOPE PAPILLOMA
  - RECOVERABILITY FACTORS
    - RABBIT, 336*

- VIRUS, SV40
  - DNA REPLICATION
    - BIDIRECTIONAL, 249
    - GROWTH CYCLE ANALYSIS, 254
    - INITIATION SITE, 244
  - DNA TRANSCRIPTION
    - TRANSFORMED CELLS, MONKEY, 265
  - INFECTION
    - DNA SYNTHESIS, HAMSTER CELLS, 343*
    - NUCLEAR ACIDIC PROTEIN, MONKEY, HAMSTER, 239
  - PHOSPHOLIPID METABOLISM
    - TRANSFORMED CELL, MOUSE, 231
  - RNA DENSITY
    - MONKEY CELLS, 222
  - SKIN FIBROBLAST INTERACTION
    - XERODERMA PIGMENTOSUM PATIENTS, 332*
  - TEMPERATURE-SENSITIVE MUTANT
    - CONCANAVALIN A BINDING, MOUSE CELL, 284
  - TRANSFORMED CELL
    - CELL GROWTH, MOUSE, 224
    - GLYCOLIPID CONTENT, MOUSE, 232
    - LIPID CONTENT, MOUSE, 224
  - TRANSFORMED CELL LINE
    - ACTINOMYCIN D RESISTANCE, HAMSTER, 261
  - TRANSFORMED CELLS
    - TRANSPLANTATION IMMUNITY, MOUSE, 359, 368
  - TRANSFORMED STATE MAINTENANCE
    - WORKING HYPOTHESIS, 273
  - TUMOR PREVENTION
    - PARITY, HAMSTER FETAL TISSUE, 447*

- VIRUS, TUMOR
  - FIBROBLAST TRANSFORMATION
    - REVIEW, 2

- VIRUS, VISNA
  - ROUS SARCOMA VIRUS
    - COMPARATIVE STUDY, ULTRASTRUCTURE, 257

- VITAMIN A
  - ARYL HYDROCARBON HYDROXYLASE ACTIVITY
    - HAMSTER CELLS, 150*
  - COLON CARCINOMA
    - AFLATOXIN B1, RAT, 66
  - TUMOR PROPHYLAXIS
    - SKIN, MOUSE, 57

- WHEAT GERM AGGLUTININ
  - RECEPTOR SITES
    - ISOLATION, LEUKEMIC MOUSE CELLS, 441*

- XERODERMA PIGMENTOSUM
  - SV40
    - SKIN FIBROBLAST INTERACTION, HUMAN, 332*

- ZINC
  - CADMIUM CARCINOGENESIS INHIBITION
    - RAT, 133*

- ZIRCONIUM
  - CHONDROMA
    - MOUSE, 64

- ZYMBAL'S GLAND
  - 2-ACETYLAMINOFLUORENE
    - CARCINOGENESIS, RAT, 143*

☆U.S. GOVERNMENT PRINTING OFFICE: 1973-544-908/1

LIBRARY U. OF I. URBANA-CHAMPAIGN

LIBRARY U. OF I. URBANA

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list.

DHEW Publications No. (NIH) 72-301

44405

[redacted]

FEBRUARY 1973
Abstracts & Citations Nos. 0601-1200

Vol. 11
No. 2

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health

me XI, No. 2
'ebruary

Abstract and Citation Nos. 0601 - 1200

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

Editor

Robert Love, M.D.

Jefferson Medical College, Philadelphia

THE LIBRARY OF THE
OCT 25 1973
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Associate Editor

George P. Studzinski, M.D.

Jefferson Medical College, Philadelphia

NCI Staff Consultants

Elizabeth Weisburger, Ph.D.

Sidney Siegel, Ph.D.

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Etiology Area
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication
approved by the Director of the Bureau of
the Budget on July 25, 1967.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For rnals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical iodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

TH	adrenocorticotropic hormone
	adenosine diphosphate
	adenosine monophosphate
	adenosine triphosphate
	degrees centigrade
	centimeter(s)
S	central nervous system
m	counts per minute
A	deoxyribonucleic acid
g.	for example
	gram(s)
	microgram(s)
	hour(s)
m.	intramuscular
p.	intraperitoneal
	international unit(s)
v.	intravenous
	kilogram(s)
50	median lethal dose(s)
	meter(s)
	molar
q	milliequivalent(s)
	millimolar
	micromolar
,μC	milli-,microcurie(s)

mg	milligram(s)
min	minute(s)
ml	milliliter(s)
mm	millimeter(s)
MTD	maximum tolerated dose
ng	nanogram (10^{-9})
pg	picogram (10^{-12})
p.o.	orally
ppm	parts per million
r	Roentgen
RBC	red blood cells (erythrocytes), red blood count
resp.	respectively
RNA	ribonucleic acid
s.c.	subcutaneous
sec	second(s)
U	unit(s)
UV	ultraviolet
WBC	white blood cells (leukocytes), white blood count
wk	week(s)
wt	weight(s)
yr	year(s)

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	0601-0653	95
CHEMICAL CARCINOGENESIS	(Chem)	0654-0766	100
PHYSICAL CARCINOGENESIS	(Phys)	0767-0782	118
VIRAL CARCINOGENESIS	(Viral)	0783-0917	120
IMMUNOLOGY	(Immun)	0918-1021	145
PATHOGENESIS	(Path)	1022-1039	162
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	1040-1068	164
MISCELLANEOUS	(Misc)	1069-1200	167
AUTHOR INDEX			
SUBJECT INDEX			

01 IMMUNOSUPPRESSION AND NEOPLASIA. II. IS DEFICIENT IMMUNESURVEILLANCE THE ONLY CHANISM BY WHICH IMMUNOSUPPRESSION PROMOTES OPLASIA? A SPECULATIVE REVIEW. *(E.)* Gleichmann, (Med. School, Hanover, W. Germany) and H. eichmann. *Klin Wochenschr* 51(6):260-265, 1973.

perimental evidence inconsistent with the immune-rveillance theory of neoplasm induction is viewed and discussed in light of two alternative chanisms: 1) altered proliferation of target ssues and 2) increased susceptibility to chemical rcinogens secondary to infection by non-oncogenic ruses. Results from many independently conducted periments have generally indicated that malignant mors can be induced more readily in rapidly oliferating than in resting tissues. Freund's juvant, which enhances the immune response, also hances the formation of malignant lymphomas duced in mice by 3-methylcholanthrene (MCA). is effect has been attributed to the ability of eund's adjuvant to stimulate lymphoreticular oliferation. Also, 6-mercaptopurine, which hibits cell proliferation, decreases the incidence MCA-induced mouse lymphomas. Regenerating mouse ver has been shown to be more susceptible than n-regenerating liver to urethan carcinogenesis. th anti-lymphocyte serum (ALS) and dimethyl-nzanthracene induce reticulum cell sarcomas mice under conditions in which suppression of munosurveillance is unlikely. Although the creased incidence of neoplasms in animals llowing thymectomy or ALS treatment has been tributed to immunosurveillance suppression, no rect evidence has been presented to favor this chanism. The fact that these two procedures wer the resistance to infection by non-oncogenic ruses and the fact that non-oncogenic viruses n markedly increase the tumor rate resulting om application of chemical carcinogens combine suggest the second alternative mechanism for oplasm induction. (161 references)

02 INTERNATIONAL STUDIES ON CANCER IN MIGRANTS. *(E.)* Herman, B. (Netherlands ıst. Preventive Med. TNO, Leiden). *TNO* 27(11): 72-676, 1972.

he results of several studies on the incidence f cancer at various body sites of migrants are eviewed. The fact that some types of cancer (e.g., astric cancer in Japanese) maintain the same inci-ence in migrant populations as that seen in home ountries suggests a genetic basis for the disease. owever, other factors such as dietary and hygienic ustoms and socioeconomic status may contribute ignificantly to the etiology. The latter explain uch findings as the uniformly low incidence of ervical cancer in Jews and the high incidence of hroat cancers in Irish males. A third factor hich must be considered, and which accounts for bserved differences in the incidence of a given ype of cancer between native and migrant populations, s changes in environment. Thus, lung cancer rates re increased in Polish, Yugoslavian and Russian igrants to the U.S.A. Attempts to elucidate the etiologic basis for many cancers in migrants is complicated by the fact that two or more of the above factors may be simultaneously responsible. The net effect seems to be an increased risk of cancer for migrants, since they retain their inherited risks, tend to maintain the risks secondary to the dietary, etc., customs of their native land, and, in addition, increase their risks for cancer of new sites due to the environmental influences of their new country. (20 references)

0603 NEURAMINIDASE, SIALIC ACIDS, AND CELL INTERACTIONS. *(E.)* Weiss, L. (Roswell Park Mem. Inst., Buffalo, N.Y.). *J Natl Cancer Inst* 50(1):3-19, 1973.

Although the sialic acids, N, O-acetylated derivatives of D-neuraminic acid, are involved in the MN antigen system, they have not been specifically associated with the antigenic-determinate sites of tumor-specific antigens or haptens, of the glycolipids associated with the cell surface. At present, there appears to be no consistent relationship between sialoglycoproteins and malignancy; however, such a relationship may exist for membrane sialoglycolipids, which almost disappear on or after malignant conversion and which increase in contact inhibited cells when cell-cell contact is maximum. Neuraminidase-susceptible sialic acids, which significantly contribute to the net cell negative charge, are apparently arranged in high-charge density clusters on the cell membrane. The relationship of these zones to concanavalin A and other receptor sites is speculative. Although evidence does not support the concept that specific adhesions occur between cells when the glycotransferases of one cell recognize their glycosidic substrates at the surface of another, it is possible that release of these enzymes from one cell may change the surface antigenicity and interactions of other malignant or normal cells. Experimental evidence in which inoculation of neuraminidase-treated tumor cells into animals bearing the same type of tumor resulted in specific tumor regression, suggest that cell surface sialic acid may be involved in immunogenicity. The mechanism, however, is probably not one of simple unmasking of previously hidden cell surface antigenic sites by neuraminidase. Neuraminidase has been shown to facilitate adsorption of cytophilic antibodies and/or opsonins to target cells and/or macrophages and, as such, the enzyme may enhance primarily the host-cell mediated part of the immune response. (141 references)

0604 MEMBRANE JUNCTIONS IN GROWTH AND DIFFERENTIATION. *(E.)* Loewenstein, W. R. (U. Miami Sch. Med., Fla.). *Fed Proc* 32(1):60-64, 1973.

Research on cell junctions is reviewed and its pertinence for the problem of the cancer cell is discussed. Cellular junctions are channels formed to connect highly permeable places on the membranes of adjacent cells. It has been shown that molecules in a wide range of sizes can and do pass from cell to cell through these junctions. Junctions, therefore, may be the route by which molecular signals controlling growth and differentiation pass among cells. Cancer cells might be cells in which, possibly as a result of a genetic defect, junctional connections with other cells are faulty. In experiments with hepatoma cells and thyroid carcinoma cells, evidence for closing of normal cell junctions has been found. (45 references)

0605 FACTORS IN THE CAUSATION AND INCIDENCE OF OVARIAN CANCER. *(E.)*

Fathalla, M. F. (Fac. Med., Assiut U., Egypt). *Obstet Gynecol Survey* 27(11):751-768, 1972.

Epidemiologic aspects of ovarian cancer, the spontaneous development of ovarian cancers in animals, and the experimental production of ovarian tumors, are discussed. Among notable features of the epidemiology of ovarian tumors are the low incidence of these tumors in Japan compared with the West, the higher mortality rates in white than in nonwhite women in the U.S., and the positive correlation between mortality and social class reported from Britain. In most countries, the incidence of ovarian cancer is low before age 40 and maximal between 60-70 yr old. A strong positive correlation between ovarian cancer and infertility has also been found. Among domestic animals, dogs and cattle develop more spontaneous ovarian tumors than horses, sheep or cats. Ovarian tumors have been induced by X-irradiation, partial castration, ligature of ovarian blood vessels, 7,12-dimethylbenz(a)anthracene, and progestational steroids. (169 references)

0606 EPIDEMIOLOGY OF PLEURAL MESOTHELIOMA IN NORTH-WESTERN ITALY (PIEDMONT). *(E.)*

Rubino, G. F. (Inst. Occupational Med., U. Turin, Italy), G Scansetti, A. Donna and G. Palestro. *Br J Ind Med* 29(4):436-442, 1972.

Fifty-four cases of mesothelioma of the pleura admitted to the Chest Surgery Centre or to the Department of Medicine of the University of Turin from 1960 to 1970 are reported. Thoracotomy was performed in 22. In the other 32 the diagnosis was based on the clinical, radiographic, and cytological findings and on the results of biopsy. In 50 cases (18 women and 32 men), the majority of whom had always or mostly lived in Piedmont, it was possible to ascertain the family history, previous residence, and occupation, mainly with the aid of information given by the patient's relatives. A similar investigation was made by the same interviewers into 50 other patients of the same sex and age admitted to the same institutions, using an identical technique. In the group with mesothelioma (only two of whom survived more than two years after the diagnosis had been made) occupational exposure to asbestos was demonstrated unequivocally in five men. Three other patients, including one woman, had lived with persons employed in the asbestos industry (16%). Exposure for occupational reasons seemed very likely in another patient, who had been a fireman in the Turin Arsenal for 40 years. One man in the control group had worked for two years in a cement-asbestos manufacturing company (2%). A re-appraisal of the histological sections and examination of new preparations made in the 22 cases operated on was done in the Department of Pathologica Anatomy of the University of Turin, also with the purpose of confirming the diagnosis. This re-appraisal revealed the presence of asbestos bodies in the mesothelioma in one case, a woman who had never been exposed to asbestos for occupational or domestic reasons but who had always lived in one of the two regions of the Province of Turin with the highest number of asbestos industries.

0607 HUMAN LYMPHOBLASTOID CELL LINES AND EPSTEIN-BARR VIRUS: A REVIEW OF THEIR INTERRELATIONSHIPS AND THEN RELEVANCE TO THE ETIOLOGY OF LEUKOPROLIFERATIVE STATES IN MAN. *(E.)*

Miller, G. (Yale U. Sch. Med., New Haven, Conn.). *Yale J Biol Med* 43(6):358-384, 1971.

Epstein-Barr virus (EBV) has been detected by electron microscopy and various immunologic methods in human lymphoblastoid cell lines (HLCL) derived from patients with Burkitt's lymphoma (BL), acute leukemia viral hepatitis, infectious mononucleosis (IM) nasopharyngeal carcinoma, and also from normal donors. The replicative cycle of EBV, a herpes-type virus, is as yet unknown due to the lack of sensitive quantitative assays necessary for its detection. EBV has associated with it an intracellular antigen, a membrane antigen and at least two complement fixing antigens. EBV is able to induce *in vitro* transformation of normal leukocytes into established lines and can superinfect HLCL that are initially devoid of EBV antigens. EBV particles-or antigens are normally detectable in less than 5% of the cells of any HLCL culture; experimental evidence suggests that all cells in an EBV-infected culture contain the EBV genome, but only a small proportion of them spontaneously make EBV particles or antigen. In patients with BL and IM, increased serum anti-EBV antibody levels and a delayed hypersensitivity reaction have been associated with clinical remission. Numerous epidemiologic studies have shown that the age at which antibody to EBV is acquired is influenced by geographic and social factors, with children from underdeveloped, low socio-economic groups developing high serum titers at very young ages. EBV antibodies were, however, found to be no more frequent in residents of the lowlands of Uganda where BL is endemic than in those living in other geographic areas where the tumor is much less frequent. EBV is probably the etiologic agent of IM. College freshmen who do not possess anti-EBV subsequently develop IM, whereas those without antibody invariably do. The route of transmission of EBV infections is unresolved. EBV has never been successfully cultured from the oral cavity or from mosquito pools, nor has evidence of congenital infection been obtained. The only documented evidence of EBV infection is by parenteral transmission. (128 references)

08 CARCINOID TUMORS. REVIEW OF LITERATURE
WITH SPECIAL REFERENCES TO SITES OF ORI-
N AND HUMORAL SUBSTANCES PRODUCED. *(E.)* Hosoda,
(Aichi Cancer Ctr. Res. Inst., Nagoya, Japan).
ta Pathol Jap 22(3):533-540, 1972.

terature on the sites of origin and humoral
bstance production of carcinoid tumors is
viewed and a hypothesis about the genesis of
ese tumors is advanced. Bronchial carcinoid
mors are known to produce ACTH while gastric
rcinoid tumors produce histamine and ileal
d appendiceal carcinoid tumors produce
rotonin. Duodenal carcinoid islet cell tumors
d rectal carcinoid tumors may constitute other
oups of carcinoid tumors, though the humoral
bstances they produce is not known. It is
pothesized that carcinoid tumors arise from
uroendocrine cells which migrate to primitive
cosa at the site of origin and there mature
to endocrine cells. (44 references)

9 THE ROLE OF IMMUNOLOGIC AND ALLERGENIC
FACTORS IN THE TUMOR GROWTH PROCESS. *(Rus.)*
rynin, V. A. (S. V. Kurashov Med. Inst. Kazan,
R) and V. P. Nefedov. *Kazan Med Zh* (5):12-17,
2. (18 references)

0 CANCER EPIDEMIOLOGY AND RELATED PROPHYLAC-
TIC PROBLEMS. *(Rus.)* Chaklin, A. V.
st. Exp. Clin. Oncol., Moscow, USSR), V. B.
levich and M. A. Bul'bulyan. *Gig Sanit* (12):95-
1972. (No references)

1 CARCINOGENIC SUBSTANCES. *(Ger.)* Anonymous.
Naturw Rdsch 25(12):478, 1972.
references)

2 LUNG CANCER - AN ENDOCRINOUS DISEASE.
(Ger.) Anonymous. *Naturw Rdsch* 25(12):
-478, 1972. (5 references)

3 IMMUNOSUPPRESSION, GENESIS AND GROWTH OF
MALIGNANT TUMOURS. *(Ger.)* Reis, H. E.
ncer Res. Dept., Essen Clin. Ruhr U., Essen,
Germany). *Z Krebsforsch* 78(1):42-50, 1972.
references)

4 CARCINOMA OF THE CERVIX AND HERPESVIRUS.
(E.) Anonymous. *Br Med J* (5813):548-549,
2. (9 references)

5 PRIMARY CARCINOMA OF THE FALLOPIAN TUBE.
(E.) Bender, R. I. (Beaver Dam, Wisc.).
bdom Surg 14(9):159-161, 1972. (6 references)

6 CELLS NEED TO STICK TO MOVE. *(E.)*
Garrod, D. (Dept. Biol., U. Southampton,
gland). *New Sci* 57(828):84-85, 1973. (No refer-
ces)

0617 CARCINOMA *IN SITU* AND EVALUATION OF EPITHELIAL CHANGES IN LARYNGOPHARYNGEAL BIOPSIES. *(E.)* Bauer, W. C. (No affiliation) and M. H. McGavran. *JAMA* 221(1):72-75, 1972. (No references)

0618 BENIGN AND MALIGNANT "MIXED TUMORS" OF THE SALIVARY GLANDS. *(E.)* Batsakis, J. G. (Dept. Path., U. Michigan, Ann Arbor). *Univ Mich Med Cent J* 37(4):178-184, 1971. (31 references)

0619 LABORATORY AIDS IN CLINICAL DIAGNOSIS: CARCINOEMBRYONIC ANTIGEN AND ALPHA-FETOGLOBULIN. *(E.)* Cohen, F. B. (No affiliation). *J Newark Beth Israel Med Ctr* 22(3):182-186, 1972. (23 references)

0620 CANCER LITERATURE INFORMATION RETRIEVAL SYSTEM SABIR. *(Ger.)* Wagner, G. (German Cancer Res. Ctr., Heidelberg) and M. Wolff-Terroine. *Z Krebsforsch* 78(1):4-11, 1972. (No references)

0621 THE PATHOLOGIC MANIFESTATIONS OF HODGKIN'S DISEASE. *(E.)* Lukes, R. J. (U. Southern California, Sch. Med., Los Angeles). *Z Krebsforsch* 78:129-136, 1972. (19 references)

0622 PROLIFERATIVE PARAMETERS OF MAMMALIAN CELL SYSTEMS AND THEIR ROLE IN TUMOR GROWTH AND CARCINOGENESIS. *(E.)* Rajewsky, M. F. (Max-Planck-Inst. Virus Res., Tübingen, West Germany). *Z Krebsforsch* 78(1):12-30, 1972. (129 references)

0623 CHLORPROMAZINES AND CANCER. *(E.)* Csatary, L. K. (Jefferson Mem. Hosp., Alexandria, Virginia). *Lancet* 2(7772):338-339, 1972. (9 references)

0624 A REVIEW ON CANCER OF CERVIX. AN ETIOLOGICAL ANALYSIS OF CASES SEEN AT AJMER. *(E.)* Mittal, S. R. (Jawaharlal Nehru Hosp., Ajmer, India). *Indian Pract* 25(5):187-193, 1972. (55 references)

0625 APPROACHES TO CANCER RESEARCH. *(E.)* Anonymous. *WHO Chron* 26(8):364-368, 1972. (No references)

0626 MODERN VIEWS OF THE VASCULAR ANATOMY OF TUMORS. *(Rus.)* Etingen, L. Ye. (Abuali Sino Med. Inst. Tadzhikistan, USSR). *Zdravookhr Tadzhikistana* 109(5):5-11, 1972. (18 references)

0627 CANCER CAUSED BY AN INHERITED SELECTIVE DEFECT IN IMMUNOLOGICAL SURVEILLANCE. *(E.)*

Laroye, G. J. (Dept. Path., Ontario Cancer Inst., Toronto, Canada). *Lancet* (7804):641-643, 1973. (42 references)

0628 SMOKING, CANCER, AND CORONARY HEART-DISEASE. *(E.)* Mehrishi, J. N. (Dept. Radiotherapeutics, U. Cambridge, England). *Lancet* (7804):666-667, 1973. (21 references)

0629 SOMATIC CELL HYBRIDS: IMPACT ON MAMMALIAN GENETICS. *(E.)* Marx, J. L. (No affiliation). *Science* 179(4075):785-787, 1973. (4 references)

0630 AETIOLOGY OF BURKITT'S LYMPHOMA. *(E.)* MacGregor, G. A. (Wood Cottage, Chilworth, Surrey, England). *Lancet* (7796):200-201, 1973. (No references)

0631 FETAL PROTEINS IN CANCER. *(Fr.)* Martin, J.-P. (No affiliation). *Recherche* 4(30): 76-77, 1973. (5 references)

0632 COMMENTS ON "THE CARCINOGENIC EFFECTS OF LOW LEVEL RADIATION. A RE-APPRAISAL OF EPIDEMIOLOGISTS METHODS AND OBSERVATIONS". *(E.)* Jablon, S. (Natl. Acad. Sci., Washington, D.C.). *Health Phys* 24(2):257-258, 1973. (5 references)

0633 LESSONS FROM THE STUDY OF MYELOMA AND ALLIED CONDITIONS. *(E.)* Waldenström, J. G. (No affiliation). *Acta Med Scand* 193(1-2): 3-4, 1973. (No references)

0634 CHILDHOOD CANCER FOLLOWING OBSTETRIC RADIOGRAPHY. *(E.)* Shore, F. J. (Dept. Physics, Queens Coll. City U. New York, N.Y.), J. S. Robertson and J. L. Bateman. *Health Phys* 24(2):258-260, 1973. (7 references)

0635 EFFECTS ON POPULATIONS OF EXPOSURE TO LOW LEVELS OF IONIZING RADIATION. *(E.)* Natl. Acad. Sci. - Natl. Res. Council (Washington, D.C.). *Bull Atomic Sci* 29(3):47-49, 1973. (No references)

0636 THE BIOLOGY OF CELL DEATH IN TUMOURS. *(E.)* Cooper, E. H. (Sch. Med., U. Leeds, England). *Cell Tissue Kinet* 6(1):87-95, 1973. (45 references)

0637 VIRUSES AND HUMAN CANCER. *(E.)* Allen, D. W. (Harvard Med. Sch., Boston, Mass.) and P. Cole. *CA* 23(2):127-136, 1973. (51 references)

0638 CHARACTERISTIC TWO-DIMENSIONAL PATTERNS OF ENZYMATIC DIGESTS OF ONCORNA AND OTHER VIRAL RNAs. *(E.)* Horst, J. (Dept. Molec. Biol. Virus Lab., U. California, Berkeley), J. Keith and H. Fraenkel-Conrat. *Nature [New Biol]* 240(99): 105-109, 1972. (17 references)

0639 RADIATION-INDUCED CANCER—THE PRESENT POSITION. REPORT OF A SYMPOSIUM. *(E.)* Saxby, M. (U. Birmingham Dental Sch., England). *Radiography* 38(451):185-186, 1972. (No references)

0640 THE EPIDEMIOLOGY OF HODGKIN'S DISEASE. *(E.)* Heath, C. W., Jr. (Epid. Program, Ctr. Dis. Control, Atlanta, Ga.). *Ann Intern Med* 77(2):313-314, 1972. (12 references)

0641 SERUM DNA LEVELS IN PATIENTS WITH MALIGNANT DISEASE. *(E.)* Perlin, E. (Natl. Naval Med. Ctr., Bethesda, Md.) and R. B. Moquin. *Am J Clin Path* 58(5):601-602, 1972. (14 references)

0642 CURRENT CONCEPTS CONCERNING THE ROLE OF VIRUSES IN HUMAN CANCER. *(E.)* Rapp, F. (Pennsylvania St. U. Coll. Med., Hershey). *Guthrie Clin Bull* 42(2):9-16, 1972. (No references)

0643 A SUMMARY OF CANCER STATISTICS — ALBERTA, CANADA, 1969 - 1970. *(E.)* Executive Office, Provincial Cancer Hosp. Bd., Alberta, Canada. *Alberta Med Bull* 37(2):37-50, 1972. (No references)

0644 ENDOMETRIAL CARCINOMA: A REVIEW OF 37 CASES IN SINGAPORE. *(E.)* Chew, P. C. T. (Dept. Obstetrics, U. Singapore, China), P. M. Tan, S. C. Chew and S. S. Ratnam. *Singapore Med J* 13(4):195-197, 1972. (6 references)

0645 CARCINOMA OF VULVA IN SINGAPORE: A REVIEW OF 12 CASES. *(E.)* Tan, P. M. (Dept. Obstetrics, U. Singapore, China), P. C. T. Chew and S. S. Ratnam. *Singapore Med J* 13(4):192-194, 1972. (6 references)

0646 EPIDERMAL MOSAIC IN BENIGN AND PRECANCEROUS NEOPLASIA (WITH SPECIAL REFERENCE TO LARGE-CELL ACANTHOMAS). *(E.)* Pinkus, H. (Dept. Dermatol., Wayne St. U., Detroit, Mich.). *Acta Dermatol (Kyoto)* 64,65:53-59, 1969-1970. (16 references)

0647 MALIGNANT TUMORS OF THE NASAL SEPTUM. *(Fr.* Cagnol, C. (Claustre Clin. Otorhinolaryng. Serv., Marseille, France), G. Alessandri and M. Garcin *J Fr Otorhinolaryngol* 21(1):9-17, 1972. (54 references)

0648 SOFT TISSUE TUMORS OF THE HEAD AND NECK: III. NEOPLASTIC AND NON-NEOPLASTIC LESIONS OF SKELETAL MUSCLE. *(E.)* Batsakis, J. G. (Dept. Path., U. Michigan, Ann Arbor) and J. E. Fox. *Univ Mich Med Cent J* 38(2):54-63, 1972. (27 references)

9 RECURRING DIGITAL FIBROUS TUMOURS OF
CHILDHOOD. *(E.)* Allen, P. W. (Armed
ces Inst. Path., Washington, D.C.). *Pathology*
):215-223, 1972. (9 references)

0 ASBESTOS: HEALTH DAMAGE, LIMIT FIGURES,
PREVENTION. *(Ger.)* Hain, E. (No
iliation), H. Bohlig, W. Klosterkoetter, A.
ütz and H.-J. Woitowitz. *Staub* 33(2):51-57,
3. (20 references)

1 PRINCIPLES OF STUDY AND EVALUATION OF
CARCINOGENICITY IN DRUG TESTING. *(Sp.)*
Research Group. *Acta Oncol* 9(1):85-90, 1970.
references)

2 WAYS OF IMPROVING THE DIAGNOSTICS OF
MALIGNANT TUMORS OF THE OVARIES. *(Rus.)*
sov, G. N. (No affiliation). *Vopr Onkol* 19(1):
-118, 1973. (122 references)

3 THE INACTIVATION OF CERTAIN ENZYMES BY
AUTOANTIBODIES IN CANCER PATIENTS. *(Rus.)*
vchenko, V. S. (Leningrad, USSR). *Vopr Onkol*
(2):99-109, 1973. (90 references)

0654 DEMONSTRATION OF DNA REPAIR IN NORMAL AND NEOPLASTIC TISSUE AFTER TREATMENT WITH PROXIMATE CHEMICAL CARCINOGENS AND ULTRAVIOLET RADIATION.

(E.) Lieberman, M. W. (Temple U. Hlth. Sci. Ctr., Philadelphia, Pa.) and P. D. Forbes. *Nature [New Biol]* 241(111):199-201, 1973.

"Unscheduled DNA synthesis" (labeling of non S phase cells) after DNA damage was observed in a variety of tissues. Autoradiographs of untreated epidermis from mouse, rat, and pig showed occasional labeled S phase cells in the basal layer and a background of 0-1 grains/cell scattered randomly over the cytoplasm and nucleus. Similar preparations from epidermis treated with ultraviolet radiation, N-acetoxy-2-acetylaminofluorene (NA-AAF), beta-propiolactone (BPL) or 7-bromoethylbenz(a)anthracene (7 BrMeBA) showed not only labeled S phase basal cells but also "sparse" or "light" labeling (3-5 grains/nucleus) of 50-75% of epidermal nuclei from all layers. Similar grain counts and numbers of labeled cells were used as criteria for unscheduled DNA synthesis in other tissues. Treated skin sections also showed unscheduled DNA synthesis in dermal fibroblast nuclei. Repair of carcinogen- and ultraviolet-induced damage was demonstratable in liver (hepatocyte nuclei), kidney (collecting tubules, distal and proximal convoluted tubules and glomeruli), and cerebellum (vascular endothelial cells). After UV or NA-AAF treatment, unscheduled DNA synthesis was seen in normal mouse epidermis, morphologically normal mouse epidermis from chronically irradiated animals, hyperplastic epidermis and epidermal (squamous cell) carcinomas. These results suggest that DNA repair may be an important or even essential component of neoplastic transformation.

0655 DISTRIBUTION OF BASIC AZO-DYE-BINDING PROTEIN IN NORMAL RAT TISSUES AND CARCINOGEN-INDUCED LIVER TUMORS.

(E.) Bannikov, G. A. (Inst. Exp. Clin. Oncology AMS, USSR), V. I. Guelstein and T. A. Tchipysheva. *Int J Cancer* 11(2):398-411, 1973.

Monospecific antibodies against basic azo-dye-binding protein from the rat liver were obtained. An antigen identical to this protein was shown to be present in the liver, kidney, small intestine, ovary and testicle extracts of rats and also in liver and kidney extracts of mice. The concentration of this antigen was significantly reduced in the liver and kidney of rat embryos and in rats during the early postnatal period. Immunofluorescence technique was employed to study distribution of the azo-dye-binding protein in sections of normal rat liver and liver tumors induced by 3'-methyl-4-dimethylaminoazobenzene. This protein was found in the hepatocytes of the central areas of the acinus but not in the hepatocytes of periportal zones. The azo-dye-binding protein was absent from poorly-differentiated hepatomas, anaplastic carcinomas and from most adenocarcinomas. In all the highly-differentiated hepatomas this protein was found to be uniformly distributed among the tumor cells. Thus the presence of azo-dye-binding protein in tumors seems to depend on the degree of cell differentiation.

0656 RENAL EPITHELIAL DYSPLASIA AND NEOPLASIA IN RATS GIVEN DIMETHYLNITROSAMINE.

(E.) McGiven, A. R. (Dept. Path., Monash U., Melbourne, Australia) and H. J. C. Ireton. *J Pathol* 108(3): 187-190, 1972.

Fourteen of 62 female Wistar rats which received dimethylnitrosamine (0.8 mg/gm body wt) by gastric intubation on six consecutive days developed renal epithelial dysplasia or adenoma between five and thirteen months after the last dose. Dysplastic areas were located in the tubular epithelium. The tubular outline was preserved. Adenomas either were cystic and lined by cuboidal or columnar epithelium forming trabeculae or papillary projections into the lumen, or were solid and composed of sheets of large polymorphic epithelial cells. Mitotic figures were numerous but the adenomas were not encapsulated and did not appear to be invading the kidney. Electron microscopy of two solid adenomas revealed cytoplasmic vacuoles lined by microvilli. It was felt that these structures may represent an abortive attempt at brush-border formation, a feature of the proximal renal tubule. The findings presented no evidence to indicate whether or not the dysplastic areas were precursors of the adenomas.

0657 ULTRASTRUCTURAL CHANGES PRODUCED BY THE CARCINOGEN, AFLATOXIN B_1, IN DIFFERENT TISSUES.

(E.) Bauer, L. (Path. Inst., U. Erlangen-Nuremberg, W. Germany), A. H. Tulusan and E. Müller. *Virchows Arch [Zellpathol]* 10(4):275-285, 1972.

Hepatic parenchyma of a fish (*Brachydanio rerio*) an tentacle epithelial cells of a mollusk (*Planorbariu corneus*) were examined by electron microscopy following 24-72 hr exposure of the animals to 0.3 ppm or 2 ppm aqueous solutions of aflatoxin B_1. The most prominent ultrastructural change--the segregation and zoning of nucleolar components--followed a essentially identical pattern in fish hepatic cells and molluscan epithelial cells and closely resembled the corresponding nucleolar reorganization produced in rat hepatic cells by acute administrati of aflatoxin B_1. The biochemical lesions reflected by these nucleolar changes are apparently inducible in eukaryotic cells in general and are not tissue-specific. No neoplasms were observed in *B. rerio* or *P. corneus* examined nine months after acute exposure to aflatoxin B_1, which has been demonstrated to produce a high incidence of hepatic carcinoma in rats.

0658 ELECTRON-MICROSCOPE OBSERVATIONS ON THE FORMATION OF THE CYTOPLASMIC LAMELLAR INCLUSION BODIES IN MURINE PULMONARY TUMOURS INDUCED *IN VITRO*.

(E.) Flaks, B. (Dept. Exp. Path. Cancer Res., U. Leeds, England) and A. Flaks. *J Pathol* 108(3):211-216, 1972.

An ultrastructural study was conducted on flank tumors arising in Balb/c mice following s.c. implantation of Balb/c lung tissue pretreated *vitro* by 20-methylcholanthrene. The tumors ich arose were classified as adenomas and enocarcinomas. Both cell types appeared to be rived from type-II pulmonary alveolar cells nce, like these, the tumor cells contained dense mellar cytoplasmic inclusion bodies. These clusion bodies were apparently derived either rectly from multivesicular bodies or indirectly om dense non-lamellated cytoplasmic bodies which re also observed in the cytoplasm of tumor cells. e non-lamellated dense bodies were apparently rived from multivesicular bodies and resembled sosomes in morphology. The mature dense lamellar clusion bodies were eventually secreted from the ical surfaces of the tumor cells.

59 EFFECTS OF MARIJUANA AND TOBACCO SMOKE ON HUMAN LUNG PHYSIOLOGY. *(E.)* Leuchtenberger, (Swiss Inst. Exp. Cancer Res., Lausanne), R. euchtenberger and A. Schneider. *Nature* 241(5385): 37-140, 1973.

ng explants prepared from normal tissue of men 45-56 yr) who had undergone surgery for pulmonary umors were exposed to smoke from marijuana or tandard tobacco cigarettes. Cytotoxic effects uch as pycnosis, necrosis and cell death, accompanied by a marked decrease of DNA synthesis and itosis, were observed 1-4 days after exposure. one of the large nuclei in exposed cultures had -4 DNA, but the majority had a significantly igher DNA content than control cultures. This ncrease was observed both during inhibition and ubsequent stimulation of mitosis and DNA synthesis 7-45 days). Mitotic abnormalities were more evere and significantly more frequent in exposed ultures. Fresh smoke from marijuana cigarettes voked essentially the same abnormalities in NA metabolism, mitosis and cell growth as whole resh or gas-vapor phase smoke from standard igarettes.

60 DRUG RESIDUES IN ANIMAL TISSUES. GAS CHROMATOGRAPHIC DETERMINATION OF IETHYLSTILBESTROL RESIDUES IN ANIMAL TISSUES. *(E.)* offin, D. E. (Hlth. Protection Br., Dept. Natl. lth. Welfare, Ottawa, Ontario, Canada) and J. C. ilon. *JAOAC* 56(2):352-357, 1973.

iethylstilbestrol residues were identified by gas hromatography in tissues from beef, chicken and amb. The purification and identification procedure nvolved acetone extraction of the tissue, HCl, ydrolysis, liquid-liquid partition between chloroorm and aqueous acidic and alkaline solutions, forration of diethylstilbestrol trifluoroacetates, lution of the trifluoroacetates from Chromosorb W olumns, and electron capture gas chromatographic etection and measurement of the diethylstilbestrol rifluoroacetates. This method enabled recovery of iethylstilbestrol and its monoglucuronide from issue samples at levels of 2-10 ppb. Diethylstilestrol residues were detected in the liver and idney of a steer fed 10 mg/day for 7-10 days. It is believed that these procedures are adequate for the extraction, hydrolysis, cleanup, and determination of diethylstilbestrol residues in a variety of animal tissues.

0661 STRUCTURE AND ACTIVITY IN CHEMICAL CARCINOGENESIS. COMPARISON OF THE REACTIONS OF 7-BROMOMETHYLBENZ[a]ANTHRACENE AND 7-BROMOMETHYL-12-METHYLBENZ[a]ANTHRACENE WITH MOUSE SKIN DEOXYRIBONUCLEIC ACID *IN VIVO*. *(E.)* Rayman, M. P. (Royal Cancer Hosp., London, England) and A. Dipple. *Biochemistry* 12(8):1538-1542, 1973.

The extents and the products of reaction of 7-bromomethylbenz[a]anthracene (7-BMBA) and 7-bromomethyl-12-methylbenz[a]anthracene (7-BM-12-MBA) with mouse skin DNA were studied *in vivo*. The less carcinogenic compound, 7-BMBA, bound more extensively but less rapidly to skin cell DNA than did the more carcinogenic compound, 7-BM-12-MBA, following painting of the substances on the backs of female Swiss mice. These results indicated that carcinogenicity was not primarily dependent on the overall capacity of the two compounds to react with DNA. Sephadex column chromatographic analysis of hydrolysates of mouse skin DNA previously exposed *in vivo* to either ^{3}H-labeled 7-BMBA or 7-BM-12-MBA showed that both compounds bound to deoxyadenosyl, deoxycytodyl, and deoxyguanosyl residues to the same extent and in the same manner as they did *in vitro*. The maximum amount of any radioactive DNA product formed was approximately the same for both compounds; thus there was no correlation between carcinogenic potential and the extent of attack on any specific nucleoside residue in DNA.

0662 POSSIBLE ROLE OF 6-HYDROXYMETHYLBENZO[a]PYRENE AS A PROXIMATE CARCINOGEN OF BENZO[a]PYRENE AND 6-METHYLBENZO[a]PYRENE. *(E.)* Flesher, J. W. (U. Kentucky Coll. Med., Lexington) and K. L. Sydnor. *Int J Cancer* 11(2):433-437, 1973.

The metabolic fate to benzo[a]pyrene (BP) and 6-methylbenzo[a]pyrene (6-MeBP) was studied in rat liver homogenates prepared from animals injected s.c. 24 hr previously with BP. Double thin-layer chromatography and UV absorption spectroscopy of the *in vitro* reaction products resulting from incubation of the liver homogenates with either BP or 6-MeBP showed that both carcinogens were converted to, among other substances, a compound identical to authentic 6-hydroxymethylbenzo[a]pyrene (6-OHMeBP). 6-OHMeBP showed the same carcinogenic potential as did BP and 6-MeBP following repeated s.c. injections into female rats. These findings were consistent with the hypothesis that methylation or hydroxymethylation is one of the first steps in metabolic activation of carcinogenic polycyclic hydrocarbons.

0663 RENAL MESENCHYMAL TUMOURS INDUCED IN RATS BY DIMETHYLNITROSAMINE: LIGHT- AND

ELECTRON-MICROSCOPE STUDIES. *(E.)* Ireton, H. J. C. (Dept. Path., Monash U., Melbourne, Australia), A. R. McGiven and D. J. Davies. *J Pathol* 108(3): 181-185, 1972.

Mesenchymal tumors, arising in the outer medulla and extending into the cortex, were observed in the kidneys of 10 of 69 female Wistar rats five to thirteen months after a regimen of daily gastric intubation of dimethylnitrosamine (0.8 mg/ 100 gm body wt) for six days. All tumors were unilateral and none invaded the perinephric tissues. The smallest, and hence the youngest, tumors were confined to the inner cortex and outer medulla and were composed of proliferating spindle-shaped cells. The largest and oldest tumors had, however, differentiated into smooth muscle and sarcomatous tissue. The evolution from spindle-shaped to smooth muscle cells with tumor development was confirmed by electron microscopy. The tumor capillary vessels differed in appearance from normal renal peritubular vessels in that they were lined by thick overlapping endothelial cells and contained microvillous projections. No evidence of a neoplastic epithelial element was found in any tumor. These findings support the concept that renal "stromal" tumors arise from a multipotential stem-cell located in the interstitial tissues of the outer renal medulla.

0664 THE MORPHOLOGY OF BETA-NAPHTHYLAMINE-INDUCED BLADDER TUMORS IN DOGS. *(Rus.)*

Romanenko, A. M. (Sci. Res. Inst. Urol. Kiev, USSR) and A. G. Martynenko. *Vopr Onkol* 18(11): 70-75, 1972.

Morphological alterations of the urinary bladder mucosa following p.o. treatment with β-naphthylamine were studied in eight mongrel female dogs. The initial dose of the carcinogen (5 mg/kg once daily) was increased to 30 mg/kg/day within a 7.5 month period. Treatment was performed 4 to 6 times/wk for 16 months and the experiment lasted 4 yr and 7 months. The dogs received 152 to 238 g of carcinogen depending on their weight; 120 biopsies were studied during the experimental period. The first histopathological changes of the bladder mucosa included chronic inflammatory processes of the subepithelial layer and the appearance of polyps 2.5 to 17 months after initiation of the experiment. Hyperplasia and proliferation were observed later in the polypoid formations and other areas of the bladder mucosa. The multicentric tumor formation process could be divided into two stages, an early one of irregular neoplastic growth ending, sometimes, with total tumor regression (1 dog) and a later stage of progressive tumor growth starting 2 yr after the beginning of the experiment, when 7 of 8 dogs presented tumor spread throughout the bladder mucosa. Four types of neoplasia were defined according to the biopsy and autopsy findings. They included transitional cell papilloma, highly and moderately differentiated tumors, and anaplastic (polymorphocellular clear cell) cancer. All neoplasias included portions of glandular or squamous cell metaplasia. Histochemical findings included increased transitional epithelial cell lactate dehydrogenase, alkaline phosphatase, and ATPase and decreased activities of some enzymes involved in the electron transport chain (succinyl dehydrogenase, cytochrome oxidase, NAD - and NADP-diaphoras during the initial period of inflammatory processes. These alterations were enhanced during the irregular tumor growth period while no alterations in cellular glycogen and nucleic acid content were observed. The atypical cells featured severe inhibition of the respiratory and final oxidation processes and a considerable activation of the glycolytic enzymes associated with a drop in cellular glycogen and an increase in nucleic audlevels during the progressive tumor growth stage.

0665 RELATIONSHIPS BETWEEN ARYL HYDROCARBON HYDROXYLASE INDUCIBILITY AND SENSITIVITY TO CHEMICALLY INDUCED SUBCUTANEOUS SARCOMAS IN VARIOUS STRAINS OF MICE. *(E.)*

Kouri, R. E. (Microbiol. Associates, Inc., Bethesda, Md.), R. A. Salerno and C. E, Whitmire. *J Natl Cancer Inst* 50(2):363-368, 1973.

Among 14 strains of mice, a direct correlation was observed between sensitivity to 3-methylcholanthrene (MCA)-induced subcutaneous tumors and the hepatic inducibility of one of the hydrocarbon-metabolizing enzyme systems, aryl hydrocarbon hydroxylase (AHH). Every strain showing a carcinogenic index (a measure of sensitivity to chemical carcinogens) >64 was inducible for AHH and every strain with an index of <31 was noninducible. Inducible strains were C3H/fMai, B10.Br/J, C57Bl/6Cum, C3H/HeN, C58/J, BALB/cCr, and C57BL/10ScSn; noninducible strains were SWR/J, Snell/Mai, 129J, SJL/J, DBA/2J, Akr/J, and DBA/1J. Closer examination involving eight strains indicated that strain sensitivity was consistent over a wide range of MCA concentrations, but was not consistent when benzo[a]pyrene or 7,12-dimethylbenz[a]anthracene were used as carcinogens. Most strains that were noninducible and insensitive to MCA-induced carcinogenicity also exhibited a high incidence of type-C RNA virogene expression, as demonstrated by the group-specific antigen characteristic of these viruses. On the other hand, strains showing a low incidence of virogene expression were usually inducible for AHH and sensitive to MCA-induced carcinogenicity. The results support the idea that certain chemical carcinogens are metabolically activated to a carcinogenic intermediate and indicate that the AHH system plays a role in this activation.

0666 ACUTE RESPONSE OF THE MOUSE BLADDER TO DERIVATIVES OF 4-ETHYLSULPHONYLNAPHTHALENE-1-SULPHONAMIDE AND BLADDER CARCINOGENS MEASURED BY THE UPTAKE OF [^{125}I]5-IODO-2'-DEOXYURIDINE. (E.)

Clayson, D. B. (Sch. Med., Leeds, England), A. J. Bedford and R. Turner. *Chem Biol Interact* 6(2):107-119, 1973.

The structural features of 4-ethylsulphonylnaphthale 1-sulphonamide (ENS) responsible for the induction o

DNA synthesis in the mouse bladder were investigated using a method in which DNA synthesis is measured by the uptake of a thymidine analog, [^{125}I]5-iodo-2'-deoxyuridine (IUdR). ENS and its analogs were administered by gastric intubation, followed one and two days later by i.p. injection of [^{125}I] IUDR. Five days after ENS administration, bladder tissue was excised, fixed, and ^{125}I incorporation into DNA was determined by counting the radioactivity in a spectrometer. The ability to stimulate DNA synthesis was unaffected by the nature of the alkyl group in 4-alkylsulphonylnaphthalene-1-sulphonamide. The sulphonamide group appeared to be essential to the activity of the molecule because naphthalene-1,4-disulphonamide was active whereas 1,4-diethylsulphonylnaphthalene was not. Maximum activity was found when the sulphonamide group was attached to an aromatic system (benzene or naphthalene) containing an alkylsulphonyl or a sulphonamide group. Bladder carcinogens other than ENS failed to stimulate the uptake of [^{125}I] IUdR sufficiently to produce statistically significant results.

0667 POSSIBLE MICROBIAL CONTRIBUTION TO NITROSAMINE FORMATION IN SEWAGE AND SOIL. *(E.)*

Ayanaba, A. (Dept. Agronomy, Cornell U., Ithaca, N.Y.), W. Verstraete and M. Alexander. *J Natl Cancer Inst* 50(3):811-813, 1973.

Sewage sludge was maintained at pH 4.0-7.0 and amended with trimethylamine (TMA, 50 or 500 g nitrogen/mol) and nitrate (10 or 100 g nitrogen/ml). Microorganisms in the sewage converted nitrate to nitrite and TMA to dimethylamine (DMA) at all pH values and produced dimethylnitrosamine (DMNA) at pH 5.0-7.0. DMA formation was solely the result of microbial activity; most of the DMNA generated from added DMA and nitrite at neutral pH was a consequence of microbial metabolism. DMNA also accumulated in acid forest soil receiving DMA and in acid sewage treated with the fungicide thiram (tetramethylthiuram disulfide). Although nitrosamines have not been detected in unamended sewage or soil, these findings indicate a potential for their formation in such environments.

0668 EFFECT OF PR-8 VIRAL RESPIRATORY INFECTION ON BENZO(A)PYRENE HYDROXYLASE ACTIVITY IN BALB/c MICE. *(E.)*

Corbett, T. H. (Biol. Div., Oak Ridge Natl. Lab., Tenn.) and P. Nettesheim. *J Natl Cancer Inst* 50(3):779-782, 1973.

PR-8 influenza virus infection decreased benzo(a)pyrene (BP) hydroxylase activity in lung tissue of BALB/c mice to 10% of control levels without affecting the enzyme activity in the liver. Suppression of lung BP hydroxylase activity was most striking 10 days postinfection, when acute pneumonia reached its peak and involved entire lung lobes. Virus infection also interfered with induction of BP hydroxylase in lung tissue. In infected animals, 20 and 2000 μg 3-methylcholanthrene (MCA) raised lung enzyme activity 12- and 90-fold, resp., above that of infected mice not given MCA. However, the uninduced enzyme level of infected animals was only 4.1% that of uninfected controls.

0669 INHIBITION OF URETHANE-INDUCED PULMONARY ADENOMAS BY INHALED PLUTONIUM-239. *(E.)*

Brightwell, J. (Dept. Path., U. Newcastle upon Tyne, England) and A. G. Heppleston. *Br J Radiol* 46(543): 180-182, 1973.

The number of pulmonary tumors induced in randombred male A2G mice by i.p. injection of urethane (1 mg/g body wt) was reduced by prior inhalation of plutonium dioxide ($^{239}PuO_2$) aerosol. However, tumor size was comparable in plutonium-exposed animals and in those receiving mock exposure, suggesting that plutonium inhalation affects induction rather than tumor growth. Mitotic incidence and labeling incidence measurements indicated that cell proliferation in the adenomas diminished with time, though the decrease was less marked in irradiated than in nonirradiated animals. It is postulated that α radiation by inhaled plutonium may enhance antibody response and thus counteract the immunosuppressive effect of urethane.

0670 STRUCTURE AND ACTIVITY IN CHEMICAL CARCINOGENESIS. COMPARISON OF THE REACTIONS OF 7-BROMOMETHYLBENZ(a)ANTHRACENE AND 7-BROMOMETHYL-12-METHYLBENZ(a)ANTHRACENE WITH DEOXRIBONUCLEIC ACID *IN VITRO*. *(E.)*

Rayman, M. P. (Royal Cancer Hosp., London, England) and A. Dipple. *Biochemistry* 12(6): 1202-1207, 1973.

In reactions with synthetic polyribonucleotides or native or denatured DNA, 7-bromomethyl-12-methylbenz(a)anthracene, a potent carcinogen, reacted less extensively with DNA than did 7-bromomethylbenz(a)-anthracene, a relatively weak carcinogen. Sephadex elution profiles of products of the reactions of the bromo compounds with DNA showed that the compounds reacted to form similar products; this indicated that the two compounds attacked DNA at the same sites, probably the amino groups of the DNA bases. Most radioactive products eluted in fractions 20-35 of the 12-methyl derivative's reaction with DNA had arisen from the interaction of light on major radioactive products.

0671 INTERACTION OF CHOLESTEROL WITH DIBENZANTHRACENES AT AIR-WATER INTERFACE. *(E.)*

Belmonte, A. A. (Sch. Pharm., U. Connecticut, Storrs) and J. Swarbrick. *J Pharm Sci* 62(3):481-483, 1973.

The interaction of cholesterol and dibenzanthracene was studied using a film balance technique under continuous compression. Mixed films spread on water and saline (made of cholesterol dissolved in chloroform and either the carcinogen 1,2:5,6-dibenzanthracene (1,2:5,6-DBA) or the noncarinogen 1,2:3,4-dibenzanthracene (1,2:3,4-DBA) dissolved in benzene) were studied with respect to surface pressure and surface potential. The results showed an association of the carcinogen, 1,2:5,6-DBA, and no association of the noncarcinogen, 1,2:4,4-DBA, with the cholesterol fraction. Surface potential data supported these findings, suggesting a possible, testable difference between carcinogens and non-carcinogens with respect to their interaction with cholesterol at the air-water interface.

0672 MICROSOME-DEPENDENT BINDING OF AFLATOXIN B_1 TO DNA, RNA, POLYRIBONUCLEOTIDES AND PROTEIN *IN VIVO*. Garner, R. C. (Dept. Exp. Path. Cancer Res., U. Leeds, England). *Chem Biol Interact* 6(2):125-129, 1973.

The binding *(in vitro)* of ^{14}C-labeled aflatoxin B_1 to DNA, RNA and synthetic polynucleotides in the presence of a pooled hamster liver microsomal extract and an NADPH-generating system was studied. Aflatoxin-binding capacity, which correlated well with results of a spectrophotometric assay, was totally dependent on the presence of the microsomal extract, indicating that a metabolite of aflatoxin was the actual reactive substance. One aflatoxin B_1 molecule bound per 190 bases of tRNA. DNA-bound aflatoxin B_1 could not be dissociated by passage of the complex through a Sephadex G-10 column.

0673 LOSS OF 7-METHYLGUANINE FROM RAT LIVER DNA AFTER METHYLATION *IN VIVO* WITH METHYLMETHANESULPHONATE OR DIMETHYLNITROSAMINE. *(E.)* Margison, G. P. (Christie Hosp. and Holt Radium Inst., Manchester, England), M. J. Capps, P. J. O'Connor and A. W. Craig. *Chem Biol Interact* 6(2):119-124, 1973.

The rate of loss of ^{14}C-labeled 7-methylguanine (7-MG) from DNA purified from the livers of male Wistar rats was studied following injection of ^{14}C-labeled methylmethanesulphonate (MMS) or dimethylnitrosamine (DMN) in amounts which did not affect hepatocyte rRNA turnover. The rate of hydrolysis of 7-MG, as determined by Dowex column chromatography of liver DNA hydrolysates taken from rats at various times up to 10 days following MMS or DMN injection, approximated first order kinetics with a half life of about 3.0 days. This half life was the same as that determined *in vitro* at pH 7, the presumed normal intracellular value. These results indicated that acid-catalyzed depurination alone could not account for this loss unless the intracellular rat hepatocyte pH at the site of 7-MG hydrolysis were acidic. It was thus impossible to distinguish between noncatalytic and catalytic hydrolysis as the functional mechanism.

0674 THE INTERACTION OF CARBON-14-LABELLED ALKYL CARBAMATES, LABELLED IN THE ALKYL AND CARBONYL POSITIONS, WITH DNA *IN VIVO*. *(E.)* Lawson, T. A. (U. Queensland Sch. Med., Brisbane, Australia) and A. W. Pound. *Chem Biol Interact* 6(2):99-105, 1973.

The binding of ethyl carbamate labeled with carbon-14 in the alkyl or carbonyl group, and of methyl, *n*-butyl and *n*-propyl carbamates labeled in the alkyl group, to the DNA of mouse liver, lung and kidney was studied in male Crackenbush mice. Only ethyl carbamate bound to liver and kidney DNA to any significant extent. The binding of ethyl carbamate labeled with carbon-14 in the C_1, C_2 or the carbonyl position was examined and compared. The levels of binding of [1-^{14}C]- and [2-^{14}C]ethyl carbamate to liver DNA were not significantly different (328 ± 34 and 267 ± 24 dmp/mg DNA, resp.), but there was very little binding of the [*carbonyl*-^{14}C] ethyl carbamate (26 ± 3 dpm mg DNA). Furthermore, only 18% of the radioactivity was removed from the DNA labeled with the alkyl-labeled carbamates, whereas 65% of the radioactivity was removed from the DNA labeled with carbonyl-labeled ethyl carbamate on continuous ether extraction. It was concluded that the bound molecule does not contain the carbonyl carbon and is probably an ethyl group.

0675 DIFFERENTIAL EFFECT OF IMMUNOSUPPRESSION ON THE INDUCTION OF NERVOUS SYSTEM AND BLADDER TUMORS BY N-METHYL N-NITROSOUREA. *(E.)* Denlinger, R. H. (Dept. Vet. Pathobiol., Ohio State U., Columbus), J. A. Swenberg, A. Koestner and W. Wechsler. *J Natl Cancer Inst* 50:87-93, 1973.

A study was undertaken to determine whether suppression of cell-mediated immunity by antilymphocyte serum (ALS) would influence the induction of neural and/or non-neural neoplasms induced by N-methyl N-nitrosourea (MNU) in male Fischer rats. I.v. administration of MNU (10 mg/kg twice weekly for 9 wk) produced a total of 129 neural tumors in 37 rats. The tumors were histologically diagnosed as neurinomas, neurofibrosarcomas and perineurial fibrosarcomas. Although i.p. administration of ALS (twice weekly for 30 wk) along with and following MNU treatment had no effect on the incidence of central nervous system (CNS), meningeal or peripheral nervous system tumors, these rats did develop a significant number of transitional cell carcinomas of the urinary bladder. No such tumors arose in rats treated with either MNU or ALS alone. The incidence of other non-neuronal tumors was unaffected by ALS treatment. These results indicate that the neurotropic activity of MNU in rats is not mediated by immunologic surveillance of neoplastic cells outside the CNS. The failure of immunosuppression to increase the number of neural tumors suggests that cell susceptibility to neoplastic transformation by MNU is more important in determining tissue tropism than the immunologic influence after the transformed cells proliferate.

0676 EFFECT OF CYCLOHEXIMIDE ON DIMETHYLNITROSAMINE-INDUCED POLYRIBOSOME DISAGGREGATION IN RAT LIVER. *(E.)* Stewart, W. W. (Middlesex Hosp. Med. Sch., London, England). *Chem Biol Interact* 6(2):81-90, 1973.

The effect of cycloheximide on dimethylnitrosamine-induced polyribosome disaggregation in the liver of female Wistar rats was studied. The simultaneous i.p. administration of 1.5 mg/kg body wt cycloheximide with 20 mg/kg body wt dimethylnitrosamine to rats did not affect the metabolism of the nitrosamine, as deduced by following its concentration in the blood, or the level of alkylation by the nitrosamine of cytoplasmic RNA in the liver. Incorporation of [^{14}C]leucine into hepatic protein, which was maximally inhibited 60% 3 hr after administration of the same dose of dimethylnitrosamine alone, was reduced by 94% within 1 hr in rats treated with dimethylnitrosamine and cycloheximide. Polyribosome structure was determined by sucrose gradient centrifugation. Disaggregation of hepatic polyribosomes as a result of administration of the nitrosamine alone was most marked at 4 hr, but by 8 hr there was a recovery of polyribosome structure and a relative decrease in the number of monometic ribosomes. Administration of cycloheximide alone did not affect the structure of hepatic polyribosomes. When dimethylnitrosamine and cycloheximide were given simultaneously the immediate breakdown of polyribosomes that normally followed administration of dimethylnitrosamine was prevented for at least 4 hr; however, after 8 hr there was considerable disaggregation of the polyribosomes in the liver.

0677 OESTROGEN RECEPTORS IN HAMSTER TUMOURS. *(E.)* Steggles, A. W. (Imperial Cancer Res. Fund., London, England) and R. J. B. King. *Eur J Cancer* 8:323-334, 1972.

Stilbestrol- or estradiol-induced, estrogen-dependent primary hamster kidney tumors, estrogen-dependent transplant tumors and estrogen-independent transplant tumors were homogenized and centrifuged. Supernatants were sedimented on sucrose gradients after labeling with (6,7-^{3}H)estradiol. Cytoplasmic estrogen receptors with sedimentation values of 8S and 4S, and a nuclear receptor with a value of 5S, were detected. No correlation was apparent between the receptor content of the different tumors and their estrogen responsiveness. The estradiol-receptor complex in tumor tissue was acid labile and estrogen specific. Studies of the 8S and 4S cytoplasmic receptors with protamine, N-ethyl maleimide and KCl suggested that these receptors were related and were similar to those found in rat uterine cytoplasm. In normal immature or ovariectomized hamster uterus, only the 8S estrogen receptor was seen. In intact normal hamster uterus, both the 4S and the 8S receptors were present two days post-estrus discharge.

0678 TOXICITY OF BENZO(a)PYRENE AND AIR POLLUTION COMPOSITE FOR ADULT HUMAN BRONCHIAL MUCOSA IN ORGAN CULTURE. *(E.)* Crocker, T. T. (U. California Coll. Med., Irvine), T. V. O'Donnell and L. L. Nunes. *Cancer Res* 33(1):88-93, 1973.

Bronchial mucosal patches from seven adult human subjects undergoing lobectomy and from five subjects during examination by bronchoscopy were maintained in organ culture for 5-11 days. Benzo(a)-pyrene (BaP), 15 µg/ml, or an air pollution composite (AP), 700 µg/ml, was present in culture media during incubation of treated explants. Some control mucosal samples were fixed at time zero, and others were maintained for 5-11 days on media containing only 0.4% acetone as vehicle for the test materials. All samples were incubated for 30 min with tritiated thymidine before fixation, and each tissue piece was examined by histological and autoradiographic methods. Exposure to BaP and AP produced sufficiently similar changes to permit the results to be pooled for comparison with control cultures. Instances of each state of epithelial differentiation in control and treated cultures were distributed in the following ratios (% in control: % in treated): columnar, 36:14; metaplastic or dysplastic, 8:2; abnormal, unclassified, 31:22; regenerative, 11:13;dead, 14:50. BaP and AP produced toxic destruction of all cell types or, as a less marked effect, suppression of DNA synthesis and distortion of morphological states of columnar but not of regenerative epithelia. Pleomorphic, replicative lesions were not induced in adult human epithelium although such lesions were present elsewhere in untreated mucosal samples. Toxicity due to AP cannot be identified with either a single component of AP nor any particular cell type. Toxicity of BaP for adult human respiratory mucosa is interpreted as evidence that microsomal mixed-function oxidases are present and active in metabolic conversion of BaP. Adult human and suckling rodent respiratory epithelia are susceptible to biological effects of BaP in the organ culture system, but toxic effects were more common in the human mucosal samples examined in this study.

0679 NITROSATION OF SARCOSINE: CHEMICAL KINETICS AND GASTRIC ASSAY. *(E.)* Friedman, M. A. (Hlth. Sci. Div., Virginia Commonwealth Univ., Richmond). *Bull Environ Contam Toxicol* 8(6):375-382, 1972.

The kinetics of the nitrosation of sarcosine (methylglycine) were studied *in vitro* and in the stomachs of Swiss mice. In *in vitro* experiments, sarcosine was incubated with sodium nitrite, and nitroso-sarcosine concentrations measured by absorbency at 250 mµ. The nitrosation reaction rate was first order with respect to both sarcosine concentration and sodium nitrite concentration. *In vivo*, sarcosine nitrosation proceeded rapidly in the stomachs of mice injected intraluminally with 0.1 ml of 1.0 M sodium nitrite or sarcosine alone or in combination. There was 22% conversion of sarcosine and sodium nitrite to nitroso-sarcosine in isolated mouse stomachs within 45 min. A material thought to be nitroso-sarcosine was isolated from stomachs treated with both sodium nitrite and sarcosine.

0680 TALC AND CARCINOMA OF THE OVARY AND CERVIX. *(E.)* Henderson, W. J. (Welsh Natl. Sch. Med., Cardiff), C. A. F. Joslin, A. C. Turnbull and K. Griffiths. *J Obstet Gynecol Br Commonw* 78(3):266-272, 1971.

Ovarian and cervical tumor material from human subjects was examined using an extraction-replication procedure. No asbestos particles were found in any tumor tissue. However, 10 of 13 ovarian tumors contained deeply embedded talc particles. Talc particles were also found in cervical tumors and in primary endometrial carcinomas. In all cases, talc particles were deep within the tumor tissue, rather than uniformly dispersed throughout the tumor mass. Electron microscope microanalysis of the extracted particles has provided preliminary evidence that the crystals contain magnesium and silicon.

0681 LOWER RESPIRATORY TRACT TUMOURS IN SYRIAN GOLDEN HAMSTERS AFTER INTRATRACHEAL INSTILLATIONS OF DIETHYLNITROSAMINE ALONE AND WITH FERRIC OXIDE. *(E.)* Feron, V. J. (Central Inst. Nutrition Food Res. TNO, Zeist, Netherlands), P. Emmelot and T. Vossenaar. *Eur J Cancer* 8(4):445-449, 1972.

Thirty-five male and 35 female hamsters were given 15 weekly intratracheal instillations of diethylnitrosamine (DENA), alone or in combination with ferric oxide. In the lower respiratory tract, bronchial papillomas were seen in four males given DENA and in 11 hamsters given DENA + ferric oxide. Bronchial adenomas were seen in 21 hamsters given DENA alone and in 33 given DENA + ferric oxide. In all, 32 hamsters in the DENA group and 94 in the DENA + ferric oxide group developed bronchiolo-alveolar adenomas. In the upper respiratory tract, nasal cavity tumors, laryngeal tumors and tracheal tumors were seen. The incidence of these tumors was similar in the DENA and DENA + ferric oxide groups.

0682 BENZOYLHYDRAZINE CARCINOGENESIS IN LUNGS AND LYMPHORETICULAR TISSUES OF SWISS MICE. *(E.)* Toth, B. (Eppley Inst., Res. Cancer Omaha, Nebr.). *Eur J Cancer* 8(3):341-345, 1972.

Fifty female and 50 male Swiss albino mice aged 6 wk were given benzoylhydrazine (BH) in drinking water (0.01% solution) for life. Sixty-eight percent of females given BH developed a total of 124 lung tumors after an average latent period of 88 wk. The incidence of lung tumors in untreated female controls was 12%. Treated males developed lung tumors in 63% of cases after a 77 wk latency (10% incidence in male controls). Malignant lymphomas developed in 36% and 10% of BH-treated females and males, resp. (incidences of 14% and 1% in control females and males, resp.). Stomach and breast carcinomas developed in a few BH-treated mice but did not appear related to treatment.

0683 DISTURBANCE BY TUMOR PROMOTORS OF EPIDERMAL GROWTH CONTROL (CHALONE MECHANISM). *(Ger.)* Marks, F. (German Cancer Res. Ctr., Heidelberg, W. German), W. Grimm and L. Krieg. *Hoppe Seylers Z Physiol Chem* 352(12):1970-1972, 1972.

Phorbol ester (TPA) applied topically to the dorsal skin of female NMRI mice completely prevented the B-adrenergic elevation of the level of intraepidermal cyclic AMP caused by a single i.p. injection of isoprenalin (13 nmol/g body wt). This effect depended directly on the amount of TPA applied and was also seen after application of other promotors such as phorbol-dibenzoate and anthraline. Since the basic level of cyclic AMP (without isoprenalin) was not altered, it was concluded that only the B-adrenergic receptor was inactivated by the tumor promotor. The effect of G_1-inhibitor pig-skin extract on epidermal DNA-synthesis was measured by pulse-labeling with tritiated thymidine *in vivo* after a single local application of TPA. The inhibitory effect of the chalone disappeared soon after TPA administration. Thirty-six hours later, when the TPA-stimulated DNA synthesis had passed through its first sharp maximum, the chalone effect re-appeared and within the next seven days increased to its normal degree of inhibition. These results support the idea that the growth-stimulating and tumor-promoting effect of cocarcinogenic phorbol esters is due to a disturbance of the intraepidermal chalone system.

0684 DIMETHYLBENZANTHRACENE-INDUCED MAMMARY TUMORS IN RATS: INHIBITION BY DDT. *(E.)* Okey, A. B. (Dept. Biol., U. Windsor, Ontario, Canada). *Life Sci I* 11(17):833-843, 1972.

Female Sprague-Dawley rats were divided into five groups and fed control chow from age 36-50 days (Group 1), chow containing 10 or 100 ppm 1,1,1-trichloro-2,2, bis (p-chlorophenyl) ethane (p,p'-DDT) from age 36-50 days (Groups 2 and 3), 100 ppm p,p'-DDT only at age 50 days (Group 4), or 250 ppm malathion from age 36-50 days. At 50 days of age, each rat was given 15 mg 7,12-dimethylbenz(a)anthracene (DMBA) by stomach tube. Pretreatment (from age 36-50 days) with p,p'-DDT significantly reduced the incidence of DMBA-induced mammary tumors. Seventy-two percent of controls given DMBA but no p,p'-DDT developed tumors, while tumor incidence in rats given 10 and 100 ppm p,p'-DDT beginning at 36 days was 44 and 36%, resp. Tumor incidence in Group 4 (not given p,p'-DDT until age 50 days) was reduced, but statistically was not significantly different than that in controls. Although the increase was not statistically significant, malathion-pretreated rats had a higher tumor incidence (81%) than controls. It was thought that p,p'-DDT stimulated hepatic methabolism of DMBA, thus reducing the dose of carcinogen reaching mammary tissue.

0685 SOME SYSTEMIC EFFECTS OF MALIGNANT TUMORS. I. CO-ENZYME A LEVELS. *(E.)* Rapp, G. W. (Loyola U. Med. Ctr., Maywood, Ill.). *Cancer* 31(2):357-360, 1973.

Co-enzyme A levels were determined in spontaneous malignant tumors in rats, mice, and humans (230 cases), in neoplasms induced by exposure of animals to carcinogenic conditions (365 cases), d in liver, spleen, adrenal, or mammary tissues f tumor-bearing hosts. The spontaneous animal mors included Walker carcinosarcoma, Jensen arcoma, and Flexner-Joblin carcinoma. The pontaneous human tumors included hepatomas, reast adenocarcinomas, colon adenocarcinomas, nd bronchogenic carcinomas. Treated animals ere exposed to whole body X-irradiation, njected with butter yellow (to induce hepatoma), ainted with 7,12-dimethylbenz(a)anthracene to induce skin cancer), or inoculated i.p. ith Schoolman leukemia ascites tumor. Reardless of source, co-enzyme A levels of alignant tumors were markedly lower than hose obtained from corresponding normal tissues. urthermore, Co-A levels in spleen, liver, and drenal tissue of animals bearing malignant umors or exposed to carcinogenic conditions were uch lower than in their normal counterparts.

686 TUMORS OF THE NERVOUS SYSTEM INDUCED BY ETHYLNITROSOUREA ADMINISTERED EITHER NTRACEREBRALLY OR SUBCUTANEOUSLY TO NEWBORN RATS: ORPHOLOGICAL AND BIOCHEMICAL CHARACTERISTICS.

E.) Grossi-Paoletti, E. (Inst. Pharmacol., . Milan, Italy), P. Paoletti, S. Pezzotta, . Schiffer and A. Fabiani. *J Neurosurg* 37(5): 80-590, 1972.

ewborn Long Evans rats were injected with 50 g/kg preparations of ethylnitrosourea (ENU) ntracerebrally or s.c.; other rats were given NU as fetuses by transplacental administration. ervous system tumors developed in rats in all dministration groups; the incidence of these umors ranged from 81-86%. Multiple tumors ere more frequent in rats given ENU at birth han in rats given ENU transplacentally. Spinal ord tumors were more common in intracerebrally nd transplacentally treated rats than in s.c. reated rats. Gasserian neurinomas, oligodendroglimomas and oligodendroglial foci were the most requent tumors. The DNA content of Gasserian umors was always higher than in the normal asserian ganglion; highest values for DNA ontent were found in gliosarcomas and lioblastomas, the most malignant tumors. RNA/ NA ratios were lower in tumor tissue than in ormal tissue. Desmosterol, a characteristic sterol of brain tumors, was detected in all ENU-induced tumors. Pretumoral areas were seen in brain and spinal cord of ENU-treated rats.

0687 THE SENSITIVITY OF THE SKIN OF HAIRLESS MICE TO THE TUMORIGENIC ACTION OF COAL AR IN RELATION TO THE TETRAZOLIUM TEST.

(E.) Iversen, U. (Oslo U. Inst. Path., Norway) and O. . Iversen. *Acta Pathol Microbiol Scand (A)* 80(5): 612-614, 1972.

airless (hr/hr) mice were painted on dorsal skin twice weekly for 13 months with a 1% solution of coal tar in benzene and the tetrazolium test for carcinogenicity was performed. The coal tar was obtained from the coke production process of a Norwegian coke factory. The tetrazolium test was positive for carcinogenicity. By the 10th month of treatment, 55% of treated mice showed skin carcinomas and 95% showed papillomas and other turmors. At 13 months more than 90% of mice had carcinomas.

0688 MITOCHONDRIAL OXIDATION OF 3,3'-DIAMINOBENZIDINE AND RELATED COMPOUNDS, AND THEIR POSSIBLE RELATION TO CARCINOGENESIS.

(E.) Hirai, K. (Chest Dis. Res. Inst., Kyoto U., Japan) and K. Yasuhira. *Gann* 63(6):665-673, 1972.

Rat liver homogenates were mixed with 3,3'-diaminobenzidine (TABP) and the oxidation of TABP by liver was determined spectrophotometrically. The mitochondrial liver fraction was the most active oxidizer of TABP. Cyanide, azide and heating at 60 C for ten min inhibited TABP oxidation. Cytochrome *c* from horse heart, added to liver mitochondrial fraction material and TABP, enhanced TABP oxidation more than threefold. Although oxidation was inhibited 70% by sodium succinate, activity was restored by sodium malonate. When cytochrome *c* was added to liver and cytochrome *a*, oxidation of TABP increased in proportion to increasing amounts of cytochrome *c*. The most potent carcinogens among various benzidine derivatives (i.e., 3,3'-dichlorobenzidine and *p*-aminobiphenyl) did not reduce cytochrome *c*, while milder carcinogens markedly reduced it.

0689 *IN VITRO* DL-1-^{14}C LEUCINE INCORPORATION INTO PROTEIN OF RAT LIVER SLICES DURING HEPATIC CARCINOGENESIS INDUCED BY DAB IN THE PRESENCE AND IN THE ABSENCE OF ALBUMIN.

(E.) Agostini, C. (Inst. Gen. Path., U. Milan, Italy) and P. Comi. *Pathol Eur* 7(2):151-154, 1972.

Female rats were fed a diet containing 0.06% 4-dimethylaminoazobenzene (DAB) for 150 day. Some rats were given 2% or 5% bovine plasma albumin in diet with or without DAB. At intervals during DAB feeding, rats were killed, their livers were removed, and uptake of DL-1-^{14}C-leucine into liver protein was measured. By 90-150 days of DAB, liver tumors had appeared in 80% of rats. Leucine incorporation in liver proteins was increased after 120-150 days on DAB or the DAB-free diet over its level 30-90 days after start of treatment. Leucine incorporation was higher in rats given the DAB diet than in rats given the DAB-free diet. The presence of 2% or 3% albumin in diet inhibited leucine incorporation into liver protein. However, 0.3% albumin sometimes enhanced leucine incorporation. The effect of albumin was not statistically different in rats fed DAB and not fed DAB.

0690 U.S. LUNG CANCER DEATH RATES BEGIN TO RISE PROPORTIONATELY MORE RAPIDLY FOR FEMALES THAN FOR MALES: A DOSE-RESPONSE EFFECT?

(E.) Burbank, F. (Natl. Cancer Inst., Bethesda, Md.). *J Chronic Dis* 25(8):473-479, 1972.

A dose-response curve for cigarette smoking and lung cancer death was constructed to explain the declining ratio of male to female lung cancer deaths in the United States. This decline, apparent since 1960, results from a rapid increase in lung cancer mortality among females. Consistent with the dose-response hypothesis for cigarette consumption and lung cancer, it was found that after 1960 the annual cigarette consumption rate among women 30 yr old and older rose, and female lung cancer deaths increased in response. The steady rise in the proportion of 30+ yr old female smokers, the lack of discontinuance of smoking among older women and the increase in the cigarette consumption of the average 30+ yr old woman portend a rapid increase in female lung cancer deaths in the near future.

0691 ENHANCED INITIATION FACTOR ACTIVITY IN RATS AFTER ADMINISTRATION OF 3-METHYLCHOLANTHRENE OR β-NAPHTHOFLAVONE.

(E.) Lanclos, K. D. (Med. Coll. Georgia, Augusta) and E. Bresnick. *Res Commun Chem Path Pharmacol* 4(2):421-432, 1972.

Male rats were injected i.p. with 20 mg/kg 3-methylcholanthrene (3MC) or β-naphthoflavone (BNF). Livers were removed and polyribosomes were isolated. A KCl ribosome wash fraction was prepared and the binding of tritiated methionyl transfer RNA (met-tRNA$_f$) to ribosomes in the presence of RNA and adenyl uridyl guanylic acid was investigated. Proteins from 3MC or BNF-treated rat liver, when added to the ^{3}H-met-tRNA$_f$ + washed liver ribosome mixture, increased the formation of binding initiation complexes by 58-100%. Factors present in the KCl wash fraction of liver ribosomes from 3MC-treated rats and control rats were separated on Sephadex G-200, and the stimulation by these factors of poly uridylic acid-directed phenylalanine incorporation was observed. Two stimulating factors, M$_3$ and M$_2$, from the KCl wash fractions from treated rats livers were able jointly to enhance incorporation above that seen with the same fractions from untreated rats. Fraction M$_1$, however, produced the same stimulation whether it was taken from treated or from untreated rats.

0692 INHIBITION BY CHLORAMPHENICOL OF AMINO AZO DYE CARCINOGENESIS IN RAT LIVER: BIOCHEMICAL STUDIES OF PLASMA MEMBRANES.

(E.) Le Page, R. N. (Dept. Path., U. Melbourne, Australia), P. R. Dorling and J. M. Blunck. *Pathology* 4(4):315-320, 1972.

Male rats were fed diets containing 2% chloramphenicol (CAP), 0.06% 3' methyl-4-dimethylaminoazobenzene (3'MeDAB), or 2% CAP and 0.06% 3'MeDAB in combination. CAP inhibited induction of hepatomas by 3'MeDAB. To investigate the protective effect of CAP, three marker enzymes in liver cell plasma membranes were studied in rats in the three treatment groups. 5'-Nucleotidase was decreased in all three diet groups, the greatest decrease occurring in rats fed 3'MeDAB + CAP. Mg^{2+}-ATPase was also decreased in all groups, the decrease being greatest in rats given 3'MeDAB only. (Na$^+$-K$^+$)-ATPase was increased in rats given CAP alone for 13 wk. In rats given 3'MeDAB, (Na$^+$-K$^+$)-ATPase increased for four to eight wk, then decreased at 13 wk of feeding.

0693 VISUALIZATION OF EXPERIMENTALLY INDUCED KIDNEY TUMORS IN RATS THROUGH PYELOGRAPHY AND ARTERIOGRAPHY.

(Ger.) Eckel, J. (Med. U. Hannover, W. Germany), G. Reznik, H. Schueller and J. Hilfrich. *Strahlentherapie* 144(5):618-621, 1972.

Combination treatment including a low-protein diet, a single i.p. injection of dimethylnitrosamine (DMNA) (30mg/kg) and a single injection of actinomycin (6μg/kg) to three-month-old female Sprague-Dawley rats caused kidney tumors in 28 of the 30 experimental animals. Pyelography performed at six week-intervals until the natural death of the animals (contrast media administration into the femoral vein and x-ray examination 3 and 12 min later) allowed the visualization of tumors as small as 5 mm in diameter. Some tumors reached sizes 7-fold that of the kidney causing complete deformation of the renal pelvis and calix system. Arteriography using slow injection of 72% urografin (2 ml) into the *arteria femoralis* and application of histoacryl-N-blue tissue adhesive provided good images 10 and 30 sec post-injection. Mushroom-like venous widening in the marginal tumor regions were visualized along with twice normal width arterial branches and irregular tumor vessels. Pyelo- and arteriography are recommended as valuable tools in the follow-up of tumor development in experimental animals; both provide safe methods for latency period determination, tumor growth and vascular architecture studies.

0694 METABOLIC ACTION OF URETHAN ON PROTEIN SYNTHESIS AND DRUG OXIDATION IN NORMAL AND REGENERATING LIVER.

(E.) Glazer, R. I. (Yale U. Sch. Med., New Haven, Conn.), D. L. Cinti, R. I. Murahata, J. B. Schenkman and A. C. Sartorelli. *Biochem Pharmacol* 21:2867-2875, 1972.

Administration of the carcinogen urethan to sham-operated rats resulted in a dose-dependent increase in the synthesis of protein on membrane-bound polyribosomes and to a lesser degree on free polyribosomes 6 hr after drug. Maximal elevation of the specific radioactivity of nascent peptide synthesized on both classes of polyribosomes of sham-operated animals occurred 6-12 hr after the administration of urethan (1 g/kg). Animals injected i.p. with urethan 12 hr after partial hepatectomy showed no increase in protein synthesis on membrane-bound polyribosomes above that occurring in saline-treated rats 2-24 hr after urethan and only a slight elevation in peptide synthesis on free polyribosomes 12 hr after the carcinogen. Aniline hydroxylation was elevated 2-fold 12-24 hr after the administration of urethan (1 g/kg) to sham-operated animals, while little or no change occurred in: (1) rate of demethyation of aminopyrine, (2) NADPH cytochrome c eductase activity, and (3) levels of cytochrome 5 and cytochrome P-450. Mixed-function oxidase ctivities in partially hepatectomized animals njected with urethan 12 hr after the operation ere either unaffected or increased to a lesser egree than in sham-operated animals.

695 NICKEL SULFIDE CARCINOGENESIS. *(E.)* Mason, M. M. (Mason Res. Inst., Worcester, ass.). *Environ Physiol Biochem* 2:137-141, 1972.

ickel sulfide is a relatively insoluble substance hich gives rise to fibrosarcomas or rhabdomyo-arcomas when injected into susceptible rats. Fischer 44 rats were injected s.c. or intramuscularly with single dose of 3.3 mg or 10.0 mg. A solid tumor ppeared in 90% of the animals within 3 to 6 months. he average weight of tumors from 17 rats selected andomly was 118 grams, a size achieved in most ases in less than one year. The average rat urvival time was from 2 to 5 months. Nickel ulfide as a carcinogen has the following special dvantages: (1) only one dose of low concentration s required to obtain tumors in 85-95% of susceptible ats; (2) the tumor is characteristically a pleo-orphic fibrosarcoma or rhabdomyosarcoma by any route f parenteral injection; (3) nickel sulfide is not azardous to the lives of other animals or to aboratory personnel since it must be given paren-erally; (4) the tumors metastasize regularly to he regional lymph glands and to the lungs; (5) he tumors can be stored successfully in liquid itrogen; (6) it is a readily-transplantable tumor; 7) it grows well in tissue culture; (8) because f differences in the tumor incidence in various at strains, genetic differences in the host can e explored; (9) the subcutaneously-situated tumors an be removed surgically within the first month f their appearance without a recurrence, a situation hich is useful for transplant immunity studies ince the animal can be bled for serum testing nd can then be reinoculated with its own tumor; nd, (10) nickel sulfide can be made into various hapes and particle sizes to see what changes ccur.

696 THE FINE STRUCTURE OF PERIVENTRICULAR PLEOMORPHIC GLIOMAS INDUCED TRANSPLACENTALLY Y N-ETHYL-N-NITROSOUREA IN BD-IX RATS WITH A NOTE ON HEIR ORIGIN. *(E.)* Lantos, P. L. (Middlesex Hosp Med. ch., London, England). *J Neurol Sci* 17(4):443-60, 1972.

he fine structure of periventricular pleomorphic liomas has been studied. The tumors were induced ith a single i.v. dose of 30 mg/kg of N-ethyl- -nitrosourea administered to pregnant BD-IX rats n the 15th day of gestation. The neoplastic trans-ormation observed in the litters was followed from arly neoplastic cell proliferations to tumors of acroscopic size. Foci of proliferating undifferen-iated glial cells (the subependymal plate cells) ere seen subjacent to the ependymal lining of he lateral ventricles. The same cell type was also present in larger tumors. The gliomas contained a mixture of oligodendrocytes, astrocytes and ependymal cells together with anaplastic glial cells. It is suggested that periventricular pleomorphic gliomas might arise from the primitive cells of the subependymal plate.

0697 BIOLOGICAL CHARACTERISTICS OF PERIPHERAL NERVE TUMORS INDUCED WITH ETHYLNITROSOUREA. *(E.)* Cravioto, H. (New York Med. Ctr., N.Y.), J. F. Weiss, E. de C. Weiss, H. H. Goebel and J. Ransohoff. *Acta Neuropathol (Berl)* 23(4):265-280, 1973.

Nervous system tumors were induced in the offspring of BD-IX (inbred) and CFE (non-inbred) rats by transplacental administration of ethylnitrosourea. Tumors of the peripheral nervous system (PNS) were obtained in approximately 80% of all animals, and tumors of the central nervous system (CNS) in 16%. Although several peripheral nerves were sites of tumor formation, the trigeminal nerve was the most frequent nerve affected. In male CFE rats, 70% of all PNS tumors affected the trigeminal nerve. The incidence of trigeminal nerve tumors in female BD-IX rats was only 33% of PNS tumors. Light and electron microscopy showed that the PNS tumors are made up of Schwann cells, and behave like malignant tumors. The tumors began as a proliferation of recognizable Schwann cells, later they lost their basement lamina, destroyed the nerve tissue and invaded neighboring structures, but did not metastasize. Like their human counterparts, they showed "dense" and "loose" areas. In both of these, Schwann cells were recognized as the tumor cells. Trigeminal nerve tumors were transplanted from *in vitro* cultured cells or directly from transplanted fresh tumor fragments to isologous rats (BD-IX). Subcutaneous, intraperitoneal and intracerebral transplants were done. The morphology and behavior of the transplanted tumors were similar to the original malignant neurinomas.

0698 ALTERATIONS IN DMBA-INDUCED RAT MAMMARY TUMORS BY ACTINOMYCIN D. *(E.)* Gardner, H. A. (Banting Inst., U. Toronto, Ontario, Canada), J. A. Kellen and K. M. Anderson. *J Natl Cancer Inst* 50(4):915-920, 1973.

Female rats were injected i.p. with actinomycin D 20 minutes before or 20 minutes after i.v. injection of 7,12-dimethylbenz(a)anthracene (DMBA). Mammary tumorigenesis was observed. All of 36 rats given 2 mg of DMBA but no actinomycin D developed mammary nodules, while 21 of 56 rats given 25 µg actinomycin D after DMBA, and 14 of 46 rats given 25 µg actinomycin D before DMBA, developed nodules. Actinomycin D not only protected against the induction of rat mammary tumors by DMBA, but also influenced the nature of those tumors that developed. Fewer glandular neoplasms, and more secretory adenomas and fibroadenomas were formed. Histologic distinctions among the various mammary nodules are described, and it is stressed that evaluation of experimental data without consideration of the histologic nature of the nodules may be invalid.

0699 THE OXIDATION OF N,N'-BIS(4-AMINOPHENYL)-N,N'-DIMETHYLETHYLENEDIAMINE (BED) BY RAT LIVER.

(E.) Holtzman, J. L. (VA Hosp., Minneapolis, Minn.) and A. M. Seligman. *Arch Biochem Biophys* 155(2):237-246, 1973.

The oxidation of N,N'-bis(4-aminophenyl)-N,N'-dimethylethylenediamine (BED) by homogenates of various tissues and fractions of fresh rat liver was examined. BED was found both to give osmiophilic deposits in tissue blocks and to increase the uptake of oxygen by hepatic homogenates. The highest activity was in the mitochondrial and next, in the microsomal fractions. Kinetics evidence indicates that the former represents two enzymatic activities while the later is a single site. The activity was greatest in the outer membrane of the mitochondria, in agreement with previous electron micrographic studies, and in the rough microsomal fraction. BED was very sensitive to both formaldehyde and detergents. The activity was not well associated with either membrane monamine oxidase or xanthine oxidase activities. The natural function of BED oxidase is still unclear. According to the authors, it is inconceivable that such a widely distributed enzyme with such a high activity does not have some important function. It is suggested that it may be important in the detoxification of aromatic amino alkaloids but some factors make this suggestion unlikely, or it may be associated with some of the other functions found in membrane structures, such as fatty acid desaturase, fatty acid synthetase, steroid synthetase, and so on.

0700 EFFECTS OF THE CARCINOGEN 2-ACETYLAMINOFLUORENE AND ITS DERIVATIVES ON BACTERIA AND BACTERIOPHAGES.

(E.) Lotlikar, P. D. (Temple U. Sch. Med., Philadelphia, Pa.), S. Fukuda and N. Yamamoto. *Mol Pharmacol* 8(6):645-650, 1972.

The interaction of various carcinogenic fluorene derivatives with genomes was studied by examining the viability of bacteria and phage and the induction of prophage. Two strains of *Salmonella typhimurium*, wild type (*hcr+*) and an ultraviolet-sensitive mutant (*hcr-*) lysogenic for phage P221, were used for cell viability and prophage induction studies. Free T5 phage particles were employed for phage viability studies; T5 phage was assayed on *Escherichia coli* K-12. Incubation with 1 mM 2-nitro-, 2-amino-, 2-diacetylamino-, 2-acetylamino-, or N-hydroxy-2-acetylaminofluorene or its glucuronide (sodium salt) for 1 hr caused neither changes in bacterial viability nor prophage induction. N-Hydroxy-2-aminofluorene was very effective in induction of prophage and causing loss of bacterial cell viability. Loss of viability of *hcr-* cells was more rapid than that of wild type (*hcr+*) cells with this compound when tested at 0.1 and 0.2 mM levels. This compound at 0.1 mM also caused rapid prophage induction in *hcr-* cells compared to wild type cells. These results suggest that N-hydroxy-2-aminofluorene damages the bacterial genome, which is restored by the bacterial repair mechanism. Similarly, nitrosofluorene (0.5 mM) and N-acetoxy-2-acetylaminofluorene (1 mM) also caused prophage induction and loss of bacterial cell viability. However, these compounds were not as effective as N-hydroxy-2-aminofluorene. In the presence of either rat liver cytosol fraction or guinea pig live microsomes, N-hydroxy-2-acetylaminofluorene effectiv ly induced the prophage and caused loss of *hcr-* cell viability. Among the compounds tested at 1 mM levels, only N-hydroxy-2-aminofluorene and N-acetoxy-2-acetylaminofluorene caused significant loss of viabi ity of free T5 phage particles, the latter being twi as effective as the former. These data demonstrate a correlation between the carcinogenicity of fluoren compounds and their effects on bacteria and bacterophages. Such systems might serve as useful tools in testing for active and ultimate carcinogens.

0701 PROTEIN MODIFICATIONS BY ACTIVATED CARCINOGENS. I. THE ACETYLATION OF RIBONUCLEASE BY *N*-ACETOXY-2-FLUORENYLACETAMIDE.

(E.) Barry, E. (VA Hosp., Minneapolis, Minn.) and H. R. Gutmann. *J Biol Chem* 248(8):2730-2737, 1973.

The postulated fragmentation of RNase by N-acetoxy-2-fluorenylacetamide (*N*-acetoxy-2-FAA), an activated metabolite of the carcinogen, *N*-hydroxy-2-fluorenylacetamide, has been investigated. The interaction of *N*-acetoxy-2-FAA with RNase resulted i the formation of two new proteins separable from RNas by electrophoresis and ion exchange chromatography. These proteins contained no new NH_2-terminal amino acids, and their amino acid composition was identica with that of native RNase, indicating that the protein had not been modified by cleavage of the peptid chain. The alternative possibility that the formation of the additional proteins was due to a decreas in the positive charge of RNase was examined by the determination of the incorporation of ^3H and ^{14}C fro *N*-[^3H]acetoxy-[9-^{14}C]2-FAA into RNase. The ratio of bound ^3H to bound ^{14}C, which measures the relative extent of acetylation and arylamidation in the modified proteins, suggested that acetylation rather tha arylamidation was the predominant reaction modifying RNase. The conclusion that the acetyl group of *N*-[^3H]-acetoxy-[9-^{14}C]2-FAA had been transferred to RNase was confirmed (*a*) by the recovery of [^3H]aceti acid from the hydrolysates of the modified protein a (*b*) by the isolation of ε-*N*-acetyl-L-lysine from enzymatic hydrolysates of the modified protein. The isolation of the *N*-acetylated amino acid accounted in part for the decrease in the positive charge of RNase after reaction with *N*-acetoxy-2-FAA. The results indicate that *N*-acetoxy-2-FAA is an acetyl donor and modifies proteins primarily by acetylation rather than arylamidation.

0702 HEPATIC TUMORS IN THE GUPPY (*LEBISTES RETICULATUS*) INDUCED BY AFLATOXIN B_1, DIMETHYLNITROSAMINE, AND 2-ACETYLAMINOFLUORENE.

(E.) Sato, S. (U. Tokyo, Japan), T. Matsushima, N. Tanaka, T. Sugimura and F. Takashima. *J Natl Cancer Inst* 50(3):765-778, 1973.

Guppies (*Lebistes reticulatus*) were fed 0.6 mg aflatoxin B_1, 480 mg dimethylnitrosamine, or 30

g 2-acetylaminofluorene/100 g powdered diet for
everal months. These carcinogens induced hepatic
amage, hyperplastic nodules, and tumors of the
iver, together with damage and tumorous lesions
f other organs. Among fish receiving aflatoxin
1, 2 of 5 had hepatic tumors after 9 months and
of 11 after 11 months. Hepatic tumors were
ound after 13 months in 2 of 20 fish given dimethyl-
itrosamine. The latter also had granulomatous
idney lesions and leiomyosarcoma in the mesentery.
ith 2-acetylaminofluorene, 1 of 3 fish developed
epatic tumors after 11 months and 3 of 13 fish
fter 13 months. Carcinogenesis in a small fish
ich as the guppy provides a suitable model for
implifying carcinogenic experiments. The possibility
f use of these fish to screen environmental carcino-
ens is suggested.

'03 ASCORBIC ACID BLOCKING OF AMINOPYRINE NITROSATION IN NZO/Bl MICE. *(E.)* Green-
att, M. (U. Otago Med. Sch., Dunedin, New Zealand).
Natl Cancer Inst 50(4):1055-1056, 1973.

O/Bl mice given a single gavage of 17.3 μmoles
dimethylaminoantipyrine (AP) and 58 μmoles sodium
trite ($NaNO_2$) developed acute centrilobular hepa-
c necrosis in 48 hr as a result of dimethylnitro-
mine (DMN) formation. High concentrations of
corbic acid (twice the molar concentration of
NO_2) blocked the nitrosative dealkylation of AP
d prevented production of DMN in quantities suf-
cient to produce hepatic necrosis. When the con-
ntration of ascorbic acid was equimolar with
NO_2, the protective effect was incomplete.

04 PROLIFERATION OF BILE DUCTULAR CELLS IN THE SPONTANEOUSLY HYPERTENSIVE RAT FED
N'-2,7-FLUORENYLENEBISACETAMIDE. *(E.)* Ito, M.
ichi Cancer Ctr., Res. Inst., Nagoya, Japan), K.
suko, M. Matsuyama, H. Suzuki, T. Nagayo and K.
ki. *J Natl Cancer Inst* 50(3):699-706, 1973.

rked proliferation of the bile ductular cells
companied by ascites was observed in the liver of
ontaneously hypertensive rats (SHR) fed N,N'-2,7-
iorenylenebisacetamide (2,7-FAA) for one month.
milder form of the lesions was also seen in nor-
tensive Wistar, Sprague-Dawley, and Buffalo rats
d 2,7-FAA. The proliferation of the ductular cells
SHR reached a peak at the end of the 4th week of
7-FAA feeding. The degree of proliferation in SHR
s more than seven times higher than in the other
ree strains. The DNA synthesis of the ductular
lls in SHR was markedly increased at the end of the
t week as compared with the normotensive Wistar.
e ductular cell proliferation in SHR was closely
lated to the age at which the administration of
7-FAA was started. When given to rats 10 wk old,
oliferation was prominent and progressed irrever-
ly. However, it was slight when 2,7-FAA was given
7 or 15 wk of age. The administration of 2,7-
evidently suppressed hypertension in SHR but did
affect the blood pressure in Wistar rats. These
ervations suggest that important changes in meta-
lic pathways occur in the liver of SHR at the initial stage of hypertension and amplify the action of 2,7-FAA.

0705 DIETHYLSTILBESTROL IN PREGNANCY. FREQUENCY OF EXPOSURE AND USAGE PATTERNS. *(E.)*
Boston Collaborative Drug Surveillance Program (Waltham, Mass.). *Cancer* 31(3):573-577, 1973.

In view of the reported association between exposure *in utero* to diethylstilbestrol (DES) and clear-cell adenocarcinoma of the vagina, data on the prenatal use of DES in some 51,000 pregnancies in 12 hospitals between the years, 1959 and 1965, were examined. Among these women, in whom prenatal drug exposure was carefully documented, there was considerable variation in DES usage, the frequency being highest in the Boston Lying-In Hospital (exposure in 1.5% of pregnancies) and the Children's Hospital, Buffalo (exposure in 0.8% of pregnancies). Data from two marketing research sources were used to estimate DES usage in the United States. It is calculated that a maximum of 50,000 liveborn females per year, born between 1960 and 1970, were exposed to DES *in utero*, while the most likely estimate is in the range 10,000 to 16,000.

0706 INDUCTION OF CARCINOMAS OF THE COLON AND RECTUM IN RATS BY INTRARECTAL INSTILLATION OF *N*-METHYL-*N*'-NITRO-*N*-NITROSOGUANIDINE. *(E.)*
So, B. T. (American Hlth. Fdn., New York, N.Y.) and E. L. Wynder. *J Natl Cancer Inst* 50(4):927-932, 1973.

Adenocarcinomas of the colon and rectum developed in 30 Sprague-Dawley rats following intrarectal administration of 2 mg *N*-methyl-*N*'-nitro-*N*-nitrosoguanidine (MNNG)/kg body wt twice weekly for 250 days. The tumors were often multiple, and there were 106 nodules in the 30 rats. In one rat, mestastases were found in liver, pancreas and adrenals. In addition to an adenocarcinoma, one animal had an invasive squamous cell carcinoma of the anus. It is believed that intrarectal administration of a local acting carcinogen is an ideal technique to study carcinogenesis in the large bowel because agents like MNNG exert a direct effect without intervening metabolism. Further, the sensitive tissue is exposed directly to agents requiring metabolism by the intestinal epithelium and adequate concentrations at the site can be secured.

0707 FIBROBLAST ANCHORAGE IN CARCINOGENESIS BY FIBRES. *(E.)* Maroudas, N. G. (Imperial Cancer Res. Fund Labs., London, England), C. H. O'Neill and M. F. Stanton. *Lancet* (7807):807-809, 1973.

A correlation is drawn between the lengths of asbestos and other mineral fibers that induce mesothelioma in the rat and the lengths of glass fibers that induce growth of fibroblasts in culture. Particles smaller than 20 μ did not induce growth of mouse or hamster fibroblasts in culture or mesothelioma when implanted into pleural cavities of rats. It is postulated that fibers induce two basic types of hyperplastic reaction, according to size: anchorage-dependent growth (40-320 μ) and phagocytosis (below 20 μ). The resultant hyperplasia, in mesenchymal or reticuloendothelial tissue, leads to mesothelioma or histiocytic lymphoma, resp.

0708 TRACE METALS IN ASBESTOS CARCINOGENESIS.

(E.) Roy-Chowdhury, A. K. (Wayne State U. Sch. Med., Detroit, Mich.), T. F. Mooney, Jr. and A. L. Reeves. *Arch Environ Health* 26(5):253-255, 1973.

Concentrations of trace metals judged potentially carcinogenic were determined in samples of amosite, crocidolite, and chrysotile. The samples included commercial material as shipped, milled and processed material as collected in animal exposure chambers, and International Union Against Cancer (UICC) reference samples. The metals determined were cobalt, nickel, chromium, manganese, and iron. The samples were treated with hydrofluoric acid, the residue taken up with hydrochloric acid, and the determination made by atomic absorption spectrometry. Substantial enrichment of the dusts in nickel and chromium during milling and dissemination was found. Cobalt and nickel content was different from that in UICC samples.

0709 GLANDULAR TUMORS OF THE NASAL CAVITY INDUCED BY DIETHYLNITROSAMINE IN SYRIAN GOLDEN HAMSTERS.

(E.) Stenback, F. (U. Nebraska Med. Ctr., Omaha). *J Natl Cancer Inst* 50(4):895-901, 1973.

Diethylnitrosamine (DEN, 1 mg) administered once weekly for 12 wk induced nasal cavity tumors in 76 of 146 Syrian hamsters. Papillary tumors growing from surface epithelium were polypoid and composed of mucin-containing glandular structures. Squamous metaplasia was a common feature of the papillary tumors, which were preceded by adenomatous hyperplasia of the respiratory epithelium. Acinar tumors of submucous glands in the anterior nasal cavity were weakly mucin-producing, in contrast to those in the posterior part with mucosa covered by olfactory epithelium, which were strongly mucin-producing. A tubular subtype was composed of cylindrical cells with little mucin production. These findings suggest that DEN-produced tumors in hamsters, derived from both the surface epithelium and the submucous glands, are similar to such tumors in man.

0710 INDUCED NUCLEOPHILIC SUBSTITUTION IN BENZO(a)PYRENE.

(E.) Johnson, M. D. (Lab. Chem. Biodynamics., U. California, Berkeley) and M. Calvin. *Nature* 241(5387):271-272, 1973.

It has been suggested that the carcinogenic action of benzo(a)pyrene (BaP) may arise from aryl hydroxylase induced binding to nucleophilic cellular components. It is suggested that attack by electrophilic oxygen produced in the hydroxylase system, occurs at the 6 position of the hydrocarbon to give a carbonium ion, localized primarily at the 1 and 3 positions, which can undergo attack with nucleophilic cellular components. Alternatively, initial attack could occur at 1 or 3, followed by nucleophilic reaction at 6. Some evidence for this theory has been obtained by using iodonium ion as a model for the hydroxylase system. BaP and iodine dypyridine nitrate react to give either the 6-iodo or 6-pyridinium derivative or a mixture depending upon reaction conditions. Equimolar quantities of BaP and iodine dipyridine nitrate react within 10 min in chloroform to give 6-iodobenzo(a)pyrene in nearly quantitative yield. Formation of the 6-pyridinium derivative requires up to 24 hr and a 2:1 molar ratio of iodonium reagent to BaP. The formation of the 6-pyridinium derivative is interpreted as arising *via* the proposed ionic mechanism with initial electrophilic attack occurring at position 1 or 3 rather than at position 6 as occurs in the formation of 6-iodobenzo(a)pyrene. The position of attack might change for steric reasons. Excess pyridine present in the reaction mixture may also provide conditions where the addition of pyridine to the sigma complex formed by electrophilic attack at 1 or 3 is faster than the rate of elimination of a proton to give the 1- or 3-iodo derivative. The dihydro-disubstituted compound thus formed would undergo elimination of HI to give the pyridinium derivative. The nuclear magnetic resonance spectra of 6-pyridinium derivative show tha protons 5 and 7 are shielded, relative to their positions in the parent hydrocarbon, by the pyridine ring current. In contrast, all protons exhibit desheildin in the 6-iodo compound.

0711 CHEMICAL DETERMINATION OF METHYLNITROSOUREA AND ETHYLNITROSOUREA IN STOMACH CONTENTS OF RATS, AFTER INTUBATION OF THE ALKYLUREAS PLUS SODIUM NITRITE.

(E.) Mirvish, S. S. (Eppley Inst. Res. Cancer, U. Nebraska Med. Ctr., Omaha) and C. Chu. *J Natl Cancer Inst* 50(3):745-750, 1973.

A method was described for determining >0.2 mg amounts of methylnitrosourea (MNU) or ethylnitrosourea (ENU) in rat stomach contents or powdered rat food. The method involved hexane extraction, ether extraction from a methanol-water mixture, a second ether extraction after removal of the methanol, thin-layer chromatography, and determination of the nitrosoureas by ultraviolet absorption. Starved rats were given 21.5 mg methylurea or 25.5 mg ethylurea plus 10 mg $NaNO_2$ by stomach tube. After 45-80 minutes the stomach contents showed a mean yield from nitrite of 27% for MNU and 8.7% for ENU (7 rats each). Intubation of sodium bicarbonate before the methylurea and nitrite were given neutralized the stomach contents and prevented MNU formation. MNU was stable when incubated with gastric juice for 1 hour at 37 C. Thirty minutes after intubation of 5 mg MNU or ENU, 27-29% of the nitrosoureas were recovered from the stomach. This is the first demonstration by chemical analysis of nitrosourea formation in the stomach. Estimates

ased on the nitrosation kinetics indicated that
ie nitrosoureas could reasonably be formed by an
cid-catalyzed chemical reaction.

712 CO-CARCINOGENEIC EFFECT OF PROGESTERONE ON 20-METHYLCHOLANTHRENE INDUCED CER-
ICAL CARCINOMA IN MICE. *(E.)* Reboud, S. (U.
esancon Sch. Med., France) and G. Pageaut. *Nature*
41(5389):398-399, 1973.

rogesterone (15 mg/mouse/3 wk) administered to 25
57BL6 mice for nine weeks by s.c. implantation of
ellets induced mucification along the vaginal
ucosa to exocervix and a mucin-producing metaplasia
the endocervical mucosa. Exposure of 20-
ethylcholanthrene for nine weeks in the cervical
anal of 50 mice induced one invasive carcinoma in
ie vagina-exocervix and five in the endocervix.
arenteral administration of progesterone to 50
ce for nine weeks was associated with carcinogenic
tion and significantly increased number of in-
asive squamous carcinoma in vagina-exocervix (15)
d in endocervix (30). Histochemical and histo-
gical examination showed that progesterone selec-
vely influenced the maturation of the induced
docervical invasive carcinoma, gave a muco-
idermoid type and had no effect on the maturation
induced invasive carcinomas from the vagina and
ocervix which remained immature.

13 PRENATAL STILBESTROL EXPERIENCE OF MOTHERS OF YOUNG CANCER PATIENTS. *(E.)* Greenwald,
(New York State Dept. Hlth., Albany), P. C. Nasca,
S. Burnett and A. Polan. *Cancer* 31(3):568-572,
73.

enatal histories, including drug use, were obtained
r the mothers of 48 young females and males reported
the New York State Cancer Registry with tumors of
e breast and urogenital organs (excluding vaginal
enocarcinomas previously reported in the literature).
ilbestrol was taken during pregnancy by the mother
only one patient, an 18-year-old girl, with an
enocarcinoma involving both the cervix and vagina.
ie trends in incidence for the 0 to 24 year age
oup, born at a time when stilbestrol might have
en used in pregnancy, were compared to the unexposed
to 34 year age groups. There were no increases
at could be attributed to stilbestrol use. There
thus far no indication that maternal use of stil-
strol contributes to the development of tumors
ier than those of the lower female genital tract.

4 ONCOGENIC ACTIVITY OF N-NITROSODODECA-METHYLENEIMINE IN LIVER, GLANDULAR STO-
H AND OTHER TISSUES OF NZO/B1 MICE. *(E.)* Goodall,
M. (U. Otago Med. Sch., Dunedin, New Zealand), W.
insky, L. Keefer and E. F. D'Ath. *Int J Cancer*
2):369-376, 1973.

itrosododecamethyleneimine (NDMI), a new member
the alicyclic nitroso imine series, was synthe-
ed and tested in NZO/B1 mice, as part of structure-

activity studies. NDMI was dissolved in olive oil at 2.5 mg/ml. A group of 40 NZO/B1 mice began treatment at eight weeks of age, and were treated for 52 weeks. Twice a week each mouse was given 0.5 ml of the NDMI solution by gavage with a rigid cannula passed into the stomach. Each mouse (average body weight 53 g during treatment) received 2.5 mg NDMI per week and a total of 130 mg NDMI (about 2.5 g/kg) during a year, after which the animals were kept without further treatment until detection of a tumor, when they were killed with ether. A set of 411 untreated NZO/B1 mice served as controls. Treated mice had significant incidences of liver and glandular stomach tumors, thought to represent the characteristic oncogenic activity of NDMI. There were also statistically significant increases in tumors of lung, duodenum and reticular tissues, but these tumors were common in controls. By contrast with the strong carcinogenic activity of lower members of the series towards pharynx and esophagus, NDMI produced no lesions at these sites. A synthesis for NDMI is given.

0715 AFLATOXIN B1 AND ACTINOMYCIN D EFFECTS ON HISTONE ACETYLATION AND DEACETYLATION IN THE LIVER.

(E.) Edwards, G. S. (Rockefeller U., New York, N.Y.) and V. G. Allfrey. *Biochim Biophys Acta* 299(2):354-366, 1973.

The administration of the potent hepatocarcinogen, aflatoxin B1, to rats leads to a sudden increase in the rate at which acetyl groups are released from histones in the liver. The effect on histone deacetylation occurs within 15 min and thus is at least as fast as the inhibitory action of the carcinogen on RNA polymerase activity. The relatively inert structural analog of this carcinogen, aflatoxin B2, does not cause a comparable effect on histone deacetylation *in vivo*. Actinomycin D also fails to elicit any increase in the rate of histone deacetylation. None of these compounds, administered beforehand, affect the rate at which isotopic acetyl groups are incorporated into the histones of the liver. A major effect of aflatoxin B1 is to increase the rate of deacetylation of histone fractions F2A1 and F3. A kinetic study of the release of acetyl groups from F2A1 and F3 subfractions differing in their degree of acetylation reveals a complex pattern of deacetylation and partial recovery. A comparison of the soluble and nuclear histone deacetylase activities of normal and aflatoxin B1-treated livers does not show any significant differences. It is suggested that the effects of aflatoxin B1 on histone deacetylation in the liver reflect a sudden modification of the systems which control the activity of the deacetylating enzymes or the availability of the histone substrates.

0716 DIMETHYLNITROSAMINE IN THE HUMAN VAGINAL VAULT.

(E.) Harington, J. S. (South African Inst. Med. Res., Johannesburg), J. R. Nunn and L. Irwig. *Nature* 241(5384):49-50, 1973.

Dimethylnitrosamine was tentatively identified in dichloromethane extracts from pooled cervical and vaginal discharges taken from 100 African patients. The identification was based on gas chromatography (where a peak coincided with that of dimethylnitrosamine) and on the mass spectrum of the component responsible for this peak. The spectrum displayed distinct peaks at mass 74 (parent ion of dimethylnitrosamine) and at mass 30, could only have been due to NO^{+}, characteristic of nitrosamines.

0717 AN IMPROVED BACTERIAL TEST SYSTEM FOR THE DETECTION AND CLASSIFICATION OF MUTAGENS AND CARCINOGENS. *(E.)* Ames, B. N. (Biochem. Dept., U. of California, Berkeley), F. D. Lee and W. E. Durston. *Proc Natl Acad Sci USA* 70(3):782-786, 1973.

An improved system is described which utilizes four strains of *Salmonella typhimurium*, varying in the composition of their lipopolysaccharide (LPS) coats, in an assay for the detection and classification of various mutagens and carcinogens. Three sets of the four tester strains have been developed: one with a normal LPS, one with a partial LPS, and one (deep rough rfa) lacking LPS down to the ketodeoxyoctanoate-lipid core. Results from testing several known mutagens and carcinogens have established the following points: 1) The newly derived rfa strains are especially advantageous for the detection of mutagens containing large ring systems. 2) If a strain without excision repair is more sensitive to killing by a compound than the corresponding strain with repair, then the compound is assumed to react covalently with DNA. 3) This "repair test", which is ten times more sensitive than the pol A test, does not, however, discriminate simple intercalating mutagens (e.g., 9-aminoacridine, quinacrine, ethidium bromide, ICR compounds), or methylating agents from other classes. 4) Testing the four different strains by assaying the selective ability of a substance to induce reversion of one or more strains to the wild type enables the identification of the following classes of mutagens: acridine half-mustards, ICR compounds, epoxides of polycyclic hydrocarbons and aromatic amine carcinogens.

0718 ULTRASTRUCTURAL, HISTOLOGICAL, AND BIOCHEMICAL ALTERATIONS PRODUCED BY 12-*O*-TETRADECANOYL-PHORBOL-13-ACETATE ON MOUSE EPIDERMIS AND THEIR RELEVANCE TO SKIN TUMOR PROMOTION. *(E.)* Raick, A. N. (Dept. Path., U. Toronto, Ontario, Canada). *Cancer Res* 33(2):269-286, 1973.

A single application of 12-*O*-tetradecanoyl-phorbol-13-acetate to mouse (Swiss-Webster) skin induces an ordered sequence of ultrastructural changes in the cells of all layers of the epidermis, which correlate well with the changes in the rate of precursor incorporation into protein, RNA, and DNA in the whole skin and also with changes in the thickness, number of nucleated cell layers, and mitotic index of the interfollicular epidermis. The incorporation of orotic acid-5-^{3}H into RNA is increased within 1 hr and reaches a peak by 3 hr. An increase in leucine-^{14}C incorporation into protein is observed at 2 hr, but it is marked only after 6 to 7 hr and reaches a high plateau by 12 hr. The incorporation of thymidine-^{3}H into DNA is markedly depressed for up to 9 hr and then sharply increases, to reach a first peak by 18 hr. The mitotic index is depressed at 8 hr, reverting to control levels at 12 to 15 hr followed by an increase in mitotic activity, and reaching a peak by 24 hr. At 16 hr, there is a marked increase in the thickness of the interfollicular epidermis and in the number of its nucleated cell layers. At this time, the mitotic index and DNA synthesis are depressed or are at control level, indicating that the increase in nucleated cell layers is due to hypertrophy, not to proliferation. Two hr after administration of 12-*O*-tetradecanoyl-phorbol-13 acetate, the nucleoli of the basal cells are markedly enlarged, with an increase of the granular and fibrillar components. By 4 hr, the granular component is further increased, polysomes appear, and the intercellular spaces between basal cells are dilated. By 6 hr, the cytoplasm of the basal cells is enlarged and filled with polysomes. The mitochondria are enlarged and have more cristae and a less dense matrix. By 12 hr, the number of polysomes is further increased, and profiles of the granular reticulum and Golgi complexes are more numerous so that the stimulated basal cells resemble those of embryonic epidermis. By 24 hr, markedly hypertrophic cells and granular mitochondrial inclusions are frequently seen. By 48 hr, flattening, involution of the organelles, and accumulations of numerous keratohyalin granules occur in the cells of the upper layers. Granular electron-dense material appears in the dilated intercellular spaces at 6 hr and increases until 36 hr, when it is also in the cisternae of the endoplasmic reticulum. The superficial cells, normally flattened, are swollen by 2 hr, and they progressively regain their nuclear and cytoplasmic organization so that, by 12 hr, they resemble the stimulated basal cells. Dark cells with ultrastructural features distinct from the other epidermal cells are observed after administration of 12-*O*-tetradecanoyl-phorbol-13-acetate, but are not found in normal or acetone-treated controls. The relevance of the cellular events to tumor promotion is analyzed, and it is postulated that tumor promoters act by altering the normal differentiation pathway, thus enabling expression of the neoplastic phenotype.

0719 THE CARCINOGENIC POTENTIAL OF AFLATOXIN OR ITS METABOLITES IN RATS FROM DAMS FED AFLATOXIN PRE- AND POSTPARTUM. *(E.)* Grice, H. C. (Path. Section, Dept. Natl. Hlth. Welfare, Ottawa, Ontario, Canada), C. A. Moodie and D. C. Smith. *Cancer Res* 33(2):262-268, 1973.

Pregnant Wistar rats were fed a diet to which groun nut meal containing aflatoxin B$_1$ and B$_2$ was added at 25% or 50% levels. The rats were fed the diet either prior to or after parturition. The 208 progeny of the treated groups developed inflammatory, hyperplastic, and neoplastic changes in the livers that were not seen in progeny of control animals fed an aflatoxin-free diet. Malignant liver tumors were seen in 3 of 95 female and 1 of 113 male progeny but not in the dams. Two of the tumors were well-differentiated cholangiocarcinomas, one with metatasis and two were hepatocellular carcinomas, both with metastasis. There is no apparent clear-cut relationship between type and incidence of liver lesions and amount of administration of aflatoxin to the dams. No dose relationship was apparent in the large number and variety of benign and malignant tumors of other organs. The treated dams and their offspring had, however, a higher incidence of tumor production generally than did the controls in this experiment.

0720 LOSS OF DENSITY-DEPENDENT REGULATION OF MULTIPLICATION OF BALB/3T3 CELLS CHEMICALLY TRANSFORMED *IN VITRO*. *(E.)* Oshiro, Y. (Natl. Cancer Inst., Bethesda, Md.) and J. A. DiPaolo. *J Cell Physiol* 81(1):133-138, 1973.

The *in vitro* growth characteristics of Balb/3T3 cells chemically transformed by 7,12-dimethylbenz(a)-anthracene (Balb/3T3-D) were studied. The multiplication rate and final cell density of Balb/3T3-D, ike Balb/3T3 cells, was dependent on the serum concentration of the medium. Whereas Balb/3T3 cells showed density-dependent inhibition of cell division and ^{3}H-thymidine incorporation at relatively low cell densities, Balb/3T3-D cells continued to replicate and pile up upon one another and did not decrease their rate of ^{3}H-thymidine uptake until ery high densities were attained. In addition, Balb/3T3-D cells required a lower serum concentration han Balb/3T3 cells to multiply at a comparable rate. Multiplication of Balb/3T3 cells was stimuated by a rat serum fraction and a bovine β-globulin raction; Balb/3T3-D cells were only slightly stimulated. The peculiar growth characteristics of Balb/3T3-D cells, which were very similar to hose of SV-40 transformed 3T3 cells, indicated hat the membrane of these cells was permanently ltered.

721 METABOLISM OF THE CARCINOGENIC HYDROCARBON BENZO(A)PYRENE IN HUMAN FIBROBLAST ND EPITHELIAL CELLS. *(E.)* Huberman, E. (Weizmann nst. Sci., Rehovath, Israel) and L. Sachs. *Int J Cancer* 11(2):412-418, 1973.

etabolism of the carcinogenic hydrocarbon benzo-a)pyrene (BP) to water and alkali-soluble products as measured in cultured fibroblast and epithelial ells from human embryos. The alkali-soluble roducts represented only a small fraction of BP etabolism. Fibroblasts from different organs from he same embryo metabolized similar amounts of BP, hile those derived from different embryos can etabolize different amounts. The fibroblasts ere divided into three groups which metabolized an average of 350, 850 and 3,400 $\mu\mu$moles of water soluble products/10^6 cells/3 days. No significant differences were found in relationship to the age of the embryos from 2.5 to 5 months. Fibroblasts from the endometrium of adult women metabolized less than embryonic fibroblasts. Cultures which contained more than 20% epithelial cells metabolized 3-25 times more BP than fibroblast cultures from the same embryo. Epithelial cells from different embryos also varied in the degree of BP metabolism and low epithelial activity was not necessarily associated with low fibroblast activity in the same embryo. It is suggested that there is a genetic heterogeneity in BP metabolism in fibroblast and epithelial cells, and that the higher activity of epithelial cells might be related to the higher incidence of carcinomas rather than sarcomas in humans.

0722 CHARGE LOCALIZATION IN THE CARBONIUM ION OF THE METHYLBENZANTHRACENES. A CLUE TO THEIR MECHANISM OF CARCINOGENESIS. *(E.)* Cavalieri, E. (Eppley Inst. Res. Cancer, Omaha, Neb.). *Proc Am Assoc Cancer Res* 13(March):125, 1972.

0723 MECHANISM OF RESISTANCE TO THE CYTOLYTIC ACTION OF CORTICOSTEROIDS. *(E.)* Turnell, R. W. (Dept. Biochem., U. British Columbia, Vancouver) and A. F. Burton. *Proc Am Assoc Cancer Res* 13(March):121, 1972.

0724 INDUCIBLE ARYL HYDROCARBON HYDROXYLASE AND 7,12-DIMETHYLBENZ[A]ANTHRACENE-PRODUCED SKIN TUMORIGENESIS IN THE MOUSE. *(E.)* Benedict, W. F. (Natl. Inst. Hlth., Bethesda, Md.), J. E. Gielen and D. W. Nebert. *Proc Am Assoc Cancer Res* 13(March):121, 1972.

0725 INDUCTION OF ADENOCARCINOMAS OF THE COLON IN MICE BY WEEKLY INJECTION OF 1,2 DIMETHYLHYDRAZINE. *(E.)* Thurnherr, N. (Mem. Hosp., Sloan-Kettering Inst., New York, N.Y.), E. Deschner and M. Lipkin. *Proc Am Assoc Cancer Res* 13(March):120, 1972.

0726 THE INTERACTION OF PHORBOL MYRISTATE ACETATE, A TUMOR PROMOTER, WITH 3T3 MOUSE EMBRYO FIBROBLASTS: A FLUORESCENCE PROBE STUDY. *(E.)* Witz, G. (Inst. Environmental Med., New York, N.Y.), A. Sivak and B. L. Van Duuren. *Proc Am Assoc Cancer Res* 13(March):119, 1972.

0727 CARCINOGEN INTERACTIONS WITH TRANSLATION MECHANISMS OF LIVER. *(E.)* Kizer, D. E. (Noble Fdn., Inc., Ardmore, Okla.) and J. A. Clouse. *Proc Am Assoc Cancer Res* 13(March):118, 1972.

0728 SELECTIVE DEPOSITION OF ESTRIOL IN THE DOG PANCREAS. *(E.)* Kirdani, R. Y.

(Roswell Park Mem. Inst., Buffalo, N.Y.), M. J. Varkarakis, D. Sampson, G. P. Murphy and A. A. Sandberg. *Proc Am Assoc Cancer Res* 13(March): 118, 1972.

0729 USE OF SERA FROM PASSIVE TRANSFER OF IMMUNITY TO A METHYLCHOLANTHRENE INDUCED SARCOMA. *(E.)* Albright, N. L. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 13(March):116, 1972.

0730 RAPID STEM CELL DIFFERENTIATION INDUCED BY NANDROLONE DECANOATE. *(E.)* Gorshein, D. (U. Pennsylvania Med. Sch., Philadelphia), W. N. Hait, E. Besa, J. H. Jepson and F. H. Gardner. *Proc Am Assoc Cancer Res* 13(March):114, 1972.

0731 BACTERIOPHAGE T4 AS AN INDICATOR ORGANISM FOR THE HOST-MEDIATED ASSAY SYSTEM. *(E.)* Martone, W. J. (New York U. Med. Ctr., New York) and F. H. Mukai. *Proc Am Assoc Cancer Res* 13(March):113, 1972.

0732 COMPARATIVE CHARACTERIZATION OF THE EMBRYOTOXIC AND TERATOGENIC EFFECTS OF N-NITROSOALKYLUREA DERIVATIVES INDUCED IN RAT FETUSES. *(Rus.)* Aleksandrov, V. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Dokl Akad Nauk SSSR* 208(6):1468-1471, 1973.

0733 FREE RADICALS AND THE LIVER MICROSOMAL DETOXIFICATION SYSTEM DURING CARCINOGENESIS. *(Rus.)* Saprin, A. N. (Inst. Chem. Phys. USSR Acad. Sci., Moscow), S. M. Gurevich and Ye. G. Zvegintseva. *Dokl Acad Nauk SSSR* 208(4):973-976, 1973.

0734 THE EFFECT OF CHLORAMPHENICOL ON THE BINDING OF ^{3}H-DIMETHYLBENZ-(a)-ANTHRACENE BY NUCLEIC ACIDS OF CULTURED MOUSE EMBRYO CELLS. *(Rus.)* Belitskiy, G. A. (M. V. Lomonosov State U. Moscow, USSR), T. A. Bogush and M. A. Panov. *Dokl Akad Nauk SSSR* 208(2):454-455, 1973.

0735 A POSSIBILITY OF SEARCHING POTENTIAL MUTAGENS AND CYTOSTATICS BY COMPUTATION OF INTRAMOLECULAR INTERACTION ENERGIES OCCURRING IN BASE ANALOGUE CONTAINING DNAs. *(Rus.)* Sukhorukoy, B. I. (Inst. Biol. Phys., Pushchino/Oka, USSR), V. I. Poltev, R. V. Polozov and I. A. Il'icheva. *Dokl Akad Nauk SSSR* 208(2):443-446, 1972.

0736 REPAIR SYNTHESIS OF DNA IN HELA AND L-CELLS FOLLOWING TREATMENT WITH METHYLNITROSOUREA OR ULTRAVIOLET LIGHT. *(E.)* Walker, I. G. (Dept. Biochem., U. Western Ontario, London, Canada) and D. F. Ewart. *Can J Biochem* 51(2):148-157, 1973.

0737 NOTE ON LUNG CANCER AMONG CIGAR AND PIPE SMOKERS. *(E.)* Tidings, J. (Roswell Park Mem. Inst., Buffalo, N.Y.) and I. D. J. Bross. *Prev Med* 1(4):543-546, 1972.

0738 THE EFFECT OF HYDRAZINE ON EMBRYONAL RAT CELL TISSUE CULTURE. *(E.)* Gokdogan, G. (Dept. Radiobiol., U. Istanbul, Turkey). *Istanbul Univ Fen Fak Mecm Ser B* 37(3-4):245-247, 1972.

0739 THE OCCURRENCE OF POLYNUCLEAR AROMATIC HYDROCARBONS (PAH) IN OUTDOOR AIR. *(E.)* Blokzijl, P. J. (Res. Inst. Public Hlth. Engineering TNO, Delft, Netherlands) and R. Guicherit. *TNO* 27(11):653-660, 1972.

0740 DETECTION AND MEASUREMENT OF THE CONCENTRATION OF ASBESTOS IN AIR. *(E.)* Meijer, P. B. (Res. Inst. Public Hlth. Engineering TNO, Delft, Netherlands) and W. E. De Koning-Pierens. *TNO* 27(11): 661-666, 1972.

0741 A NOVEL SYNTHESIS AND CARCINOGENICITY OF DIBENZO[*a*,*l*]PYRENE. *(E.)* Masuda, Y. (Daiichi Coll. Pharmaceutical Sci., Fukuoka, Japan) and R. Kagawa. *Chem Pharm Bull (Tokyo)* 20(12): 2736-2737, 1972.

0742 CARBARYL — A NEW CHEMICAL MUTAGEN. *(E.)* Hoque, M. Z. (Dept. Agronomy, U. Philippines, Los Banos). *Current Sci* 41(23):855-856, 1972.

0743 TRANSLOCATION HETEROZYGOTE INDUCED IN *CAPSICUM ANNUUM* L. *(E.)* Bansal, H. C. (Div. Genetics, Indian Agricultural Res. Inst., New Delhi) and D. Singh. *Current Sci* 41(23):853, 1972.

0744 A COMPARATIVE HISTOCHEMICAL STUDY OF RAT LIVERS IN ALPHA-NAPHTHYL-ISO-THIOCYANATE (ANIT) AND DL-ETHIONINE INTOXICATION. *(E.)* Chou, S. T. (Dept. Path., U. Hong Kong, China) and J. B. Gibson. *J Pathol* 108(1):73-83, 1972.

0745 PARTIAL INTESTINAL OBSTRUCTION DUE TO PERITONEAL MESOTHELIOMA IN CHRONIC ASBESTOS EXPOSURE. *(E.)* Adelman, H. (Mary Immaculate Hosp. Div. Catholic Med. Ctr., Brooklyn, N.Y.), P. Berkson and J. P. Sackler. *NY State J Med* 72(18):2332-2334, 1972.

0746 DIAGNOSTIC CYTOLOGY OF URINARY TRACT CARCINOMA. A REVIEW OF EXPERIENCE WITH SPONTANEOUS AND CARCINOGEN INDUCED TUMORS IN MAN. *(E.)* Melamed, M. R. (Mem. Hosp. Cancer Allied Dis., New York, N.Y.). *Eur J Cancer* 8(3):287-292, 1972.

0747 PHORBOL ESTERS AS A TOOL IN CELL RESEARCH? *(E.)* Süss, R. (German Cancer Res. Ctr., Heidelberg), G. Kreibich and V. Kinzel. *Eur J Cancer* 8(3):299-304, 1972.

)748 TERATOGENICITY, MUTAGENICITY, AND CELLULAR TOXICITY OF PHTHALATE ESTERS. *(E.)* Dill-ingham, E. O. (U. Tennessee Med. Units, Memphis) and I. Autian. *Environ Health Perspect* (3):81-89, 1973.

)749 THE EFFECT OF CARBON TETRACHLORIDE ON LYSOSOME FUNCTION IN KIDNEYS AND LIVERS)F MICE. *(E.)* Mego, J. L. (Dept. Biol., U. Alabama, niversity) and J. A. Cain. *Biochim Biophys Acta* :97(2):343-345, 1973.

750 ASBESTOS AND CANCER OF HEAD AND NECK. *(E.)* Stell, P. M. (Ear, Nose Throat Infirm., . Liverpool, England) and T. McGill. *Lancet* (7804): 78, 1973.

751 MORPHOGENETIC AND CYTOGENETIC EFFECTS OF 7,12-DIMETHYLBENZ(a)ANTHRACENE ON *HAWORTHIA* ALLUS *IN VITRO*. *(E.)* Majumdar, S. K. (Dept. Biol., afayette Coll., Easton, Pa.) and R. S. Newton. *xperientia* 28(12):1497-1498, 1972.

'52 INTERPRETATION OF THE SPECTRAL MODIFICATIONS OF AROMATIC HYDROCARBONS OCCURING UPON THEIR 'ACKING WITH NUCLEIC ACIDS. *(Fr.)* Mantione, M.-J. nst. Phys.-Chem. Biol., Paris, France). *Photochem otobiol* 17(3):169-177, 1973.

'53 SELF-FRUSTRATION, NIGHTTIME SMOKING AND LUNG CANCER. *(E.)* Abse, D. W. (Dept. ychiatry, U. Virginia Hosp., Charlottesville), M. Wilkins, G. Kirschner, D. L. Weston, R. S. Brown d W. D. Buxton. *Psychosom Med* 34(5):395-404, 1972.

54 RADIOGRAPHIC METHOD FOR *IN VIVO* ESTIMATION OF BIOLOGICAL ACTIVITY OF INDUCED GASTRO-TESTINAL CANCERS IN RATS. *(E.)* Ben-Menachem, Y. effeerson Med. Coll., Philadelphia, Pa.), S. P. Bra-w and S. Sabesin. *Invest Radiol* 7(6):526-532, 1972.

55 EFFECTS OF URETHANE ON EMBRYONIC DEVELOPMENT IN MOUSE. *(E.)* Diwan, B. A. (Cancer s. Inst., Bombay, India) and B. K. Batra. *Indian Exp Biol* 10(2):81-83, 1972.

56 INTERACTION OF CHROMATID LESIONS INDUCED BY N-NITROSO-N-METHYLUREA AND N-NITROSO-THYLUREA IN *VICIA FABA*. *(E.)* Kucera, J. (Inst. l. Tech. Information, Praha, Czechoslovakia). *romosoma* 39(1):45-51, 1972.

57 UNIMPAIRED DEVELOPMENT OF DMBA-INDUCED MAMMARY TUMORS IN RATS TREATED WITH .Y (I).POLY(C). *(E.)* Anderson, K. M. (Sch. Med., Toronto, Canada). *Res Commun Chem Pathol Pharmacol* l):193-202, 1972.

8 BLADDER CANCER IN RUBBER WORKERS. *(E.)* Noon, C. (No affiliation). *Lancet* (7785): 0-1041, 1972.

0759 COLPOSCOPIC DETECTION OF PRECANCEROUS CONDITIONS OF THE UTERINE CERVIX IN WOMEN WORKING WITH CERTAIN INDUSTRIAL POISONS. *(Rus.)* Vikshraytis, Ch. Yu. (Sci. Res. Inst. Oncol., Vilnyus, USSR) and N. P. Moteyunene. *Vopr Onkol* 18(12):41-45, 1972.

0760 MORPHOLOGICAL ALTERATIONS OF THE LIVER OF *LEBISTES RETICULATUS* FISH FOLLOWING DIETHYL- AND DIMETHYLNITROSAMINE ADMINISTRATION. *(Rus.)* Khudoley, V. V. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Vopr Onkol* 19(1):88-94, 1973.

0761 SOME METABOLISM PARAMETERS OF INDUCED MAMMARY GLAND TUMORS AS AFFECTED BY ESTROGEN THERAPY. *(Ukr.)* Khayets'kyy, I.K. (Inst. Oncol. Probl. Kiev, USSR) and S. P. Osyns'kyy. *Dopov Akad Nauk Ukr RSR Ser B Kh* 85-88, 1973.

0762 LUNG AND MAMMARY GLAND TUMORS INDUCED BY HYDRAZINE SULFATE IN BALB/c/Cb Se FORCE-BRED IN PSEUDOPREGNANT MICE. *(It.)* Biancofiori, C. (Inst. Anat. Histopath. U. Pisa, Italy). *Lav Ist Anat Istol Patol Perugia* 31(3):79-90, 1971.

0763 PRIMARY BRONCHIAL CARCINOMA IN A WINE - GROWER. THE ROLE OF ARSENIC COMPOUNDS. *(Fr.)* Poirier, R. (Regl. Ctr. Cancer Control, Marseille, France), R. Favre, J. P. Kleisbauer, G. Ingenito, J. Paoli, P. Laval and X. Serafino. *Nouv Presse Med* 2(2):91-92, 1973.

0764 CARCINOGENICITY STUDIES OF CLINICALLY USED ANTICANCER AGENTS. *(E.)* Prejean, J. D. (Mem. Inst. Path., U. Alabama, Birmingham), D. P. Griswold, A. E. Casey, J. C. Peckham, J. H. Weisburger, E. K. Weisburger and H. B. Wood, Jr. *Proc Am Assoc Cancer Res* 13(March):112, 1972.

0765 FUNCTIONAL TRANSPLANTABLE OVARIAN TUMORS (OvT) DEVELOPED IN AxC RATS GRAFTED WITH THE GONADOTROPIC PITUITARY TUMOR (GpicPT). *(E.)* Iglesias, R. (Natl. Hlth. Service, Santiago, Chile), S. Salinas, P. Vukusic, S. Girardi and X. Polanco. *Proc Am Assoc Cancer Res* 13(March):110, 1972.

0766 CHEMICAL DETERMINATION OF METHYLNITROSOUREA (MNU) IN THE RAT STOMACH AFTER GIVING METHYLUREA (MU) PLUS SODIUM NITRITE BY STOMACH TUBE. *(E.)* Mirvish, S. S. (U. Nebraska Med. Ctr., Omaha) and C. Chu. *Proc Am Assoc Cancer Res* 13(March):108, 1972.

See also:

* (Rev): 0611, 0628, 0650, 0651
* (Phys): 0770
* (Viral): 0844, 0851, 0874, 0881
* (Immun): 0921, 0926, 0933, 0946, 0953, 0958, 0978, 0979, 0981
* (Path): 1025
* (Epid-Biom): 1054, 1055

0767 VIRUS PARTICLES IN RELATION TO RADIATION-INDUCED CHANGES IN THE THYMUS OF C_3H MICE.

(E.) Järplid, B. (Div. Rad. Biol., Res. Inst. Natl. Defense, Sundbyberg, Sweden). *Acta Radiol [Ther] (Stockh)* 11(5):422-431, 1972.

Electron microscopic studies were conducted to determine the kinetics of appearance of virus particles in the thymus of female C_3H mice following fractionated whole body X-irradiation (550 R total dose). Both A-type and C-type particles were observed in, and budding from, cytoplasmic vacuoles and also within the cytoplasmic matrix and endoplasmic reticulum of thymic medullary epithelial cells 3, 10 and 17 days after the last irradiation. The occurrence of virus particles did not appear to be related to the morphological appearance of the thymus. Intraperitoneal injection of cell-free extracts of virus-containing thymus tissue into newborn mice or into thymic grafts under the kidney capsule of previously thymectomized, X-irradiated adult syngenic mice failed to induce lymphomas within 24 months. A small number of virus particles were also seen in thymic cells of control mice.

0768 RENAL TUMORS IN PARABIOSED IRRADIATED RATS.

(E.) Warren, S. (New England Deaconess Hosp., Boston, Mass.), C. E. Brown and M. W. Porter. *Radiat Res* 53(1):153-163, 1973.

Six hundred forty pairs of female and 604 pairs of male rats were anastomosed two months before the right partner was exposed to 1000 R X-irradiation. Tumor incidence in irradiated and unirradiated parabionts was observed. Ninety-five irradiated partners among 1244 parabiosed pairs developed renal tumors while four nonirradiated partners developed renal tumors. Sixty-two of the rats developing tumors were male; 33 were female. Renal tumors were usually renal cell carcinomas or adenomas. Three of 562 single control rats developed renal tumors. The nonneoplastic renal tubular epithelium of irradiated tumor-bearing kidneys had fewer mitoses than the renal tubular epithelium of kidneys of nonirradiated partners.

0769 AUTOPSY STUDY OF GRANULOCYTIC SARCOMA (CHLOROMA) IN PATIENTS WITH MYELOGENOUS LEUKEMIA, HIROSHIMA-NAGASAKI 1949-1969.

(E.) Liu, P. I. (Atomic Bomb Casualty Commission, Hiroshima, Japan), T. Ishimaru, D. H. McGregor, H. Okada and A. Steer. *Cancer* 31(4):948-955, 1973.

Granulocytic sarcoma (chloroma) was present in 23 of 338 myelogenous leukemia patients autopsied in Hiroshima and Nagasaki during 1949-1969. There was no evidence that granulocytic sarcoma was more frequent among those who received heavy atomic-bomb irradiation, although the incidence of myelogenous leukemia was greatly increased in the heavily irradiated group compared with controls. Granulocytic sarcoma was found in 19 of 237 persons with acute myelogenous leukemia, in 3 of 77 with chronic myelogenous leukemia, and 1 of 24 with chronic myelogenous leukemia with blast crisis. It was distinguished from myelogenous leukemia by: significantly higher frequency in children and young adults; clinical manifestations of pain and tumor formation (often with exophthalmos) followed by paralysis and/or urinary incontinence; slightly shorter survival time; and greater intensity of leukemic infiltration in the pituitary. At autopsy, gross tumors occurred most frequently in bone, ovary, lymph nodes, kidney, dura, lung, arm, and breast. The pathogenesis of tumor formation in leukemia is discussed.

0770 PRIMARY PULMONARY NEOPLASMS IN BEAGLE DOGS EXPOSED TO AEROSOLS OF ^{144}CE IN FUSED CLAY PARTICLES.

(E.) Hahn, F. F. (Lovelace Fdn. Med. Education Res., Albuquerque, New Mexico), S. A. Benjamin, B. B. Boeker, T. L. Chiffelle, C. H. Hobbs, R. K. Jones, R. O. McClellan, J. A. Pickrell and H. C. Redman. *J Natl Cancer Inst* 50(3):675-698, 1973.

To determine the effects of inhaled ^{144}Ce, 126 Beagle dogs (including 15 controls) were exposed to aerosols of either ^{144}Ce in fused clay or stable cerium in fused clay. To date, 15 dogs have died 143-410 days after inhalation exposure from radiation pneumonitis and pulmonary fibrosis. Primary pulmonary neoplasms caused the deaths of 5 dogs 750-1318 days after inhalation exposure. Calculated pulmonary radiation doses until death ranged from 23,000-48,000 rads. Pulmonary hemangiosarcomas were found in all 5 dogs and metastas in 4. In addition, 1 dog had a pulmonary fibrosarcoma and another had a bronchiolo-alveolar carcinoma, both of which metastasized to the tracheobronchial lymph nodes. Only 3 dogs with similar initial lung burdens of ^{144}Ce died without pulmonary neoplasms at 186, 193, and 410 days after inhalation exposure. The 91 dogs exposed to ^{144}Ce-fused clay and all controls have survived 239-1565 days after inhalation exposure including 44 surviving >750 days, with pulmonary radiation doses from 2-36,000 rads.

0771 CHILDHOOD CANCER FOLLOWING OBSTETRIC RADIOGRAPHY.

(E.) Stewart, A. (Dept. Social Med., Oxford U., England) and G. W. Kneale. *Health Phys* 24(3):359, 1973.

0772 DNA REPAIR SYNTHESIS AFTER X-IRRADIATION IN *E. COLI*.

(E.) David, C. B. (Dept. Med., Duke U., Durham, North Carolina), W. B. Kremer, A. T. Huang and D. J. Fluke. *Experientia* 28(12): 1499-1502, 1972.

0773 IS THE ABILITY TO REPAIR DAMAGE TO DNA RELATED TO THE PROLIFERATIVE CAPACITY OF A CELL? THE REJOINING OF X-RAY-PRODUCED STRAND BREAKS.

(E.) Karran, P. (Chester Beatty Res. Inst., Sutton, England) and M. G. Ormerod. *Biochim Biophys Acta* 299(1):54-64, 1973.

0774 CORRELATION BETWEEN DEGRADATION, REPLICATION AND REPAIR OF YEAST DNA IRRADIATED BY ULTRAVIOLET OR γ-RAYS.

(E.) Hatzfeld, J. (Inst. Radium-Biol., Orsay, France). *Biochim Biophys Acta* 299(1):43-53, 1973.

75 EXPERIMENTAL INDUCTION OF BENIGN MESENCHYMAL
JAW TUMOURS. *(E.)* Eulderink, F. (Leiden
Path. Lab., Netherlands) and Th. G. van Rijssel.
hol Eur 7(2):144-150, 1972.

6 MANAGEMENT OF PRECANCER OF THE SKIN. *(E.)*
Smith, J. D. (Baylor Coll. Med., Houston,
.) and J. M. Knox. *Hosp Med* 8(2):40-63, 1972.

77 SINGLE- AND DOUBLE-STRAND BREAKS IN 5-BROMO-
URACIL-SUBSTITUTED DNA OF *B. SUBTILIS*
) PHAGE PBSH AFTER IRRADIATION WITH LONG-WAVE LENGTH
AND THEIR CORRELATION TO INTRAMOLECULAR ENERGY
NSFER. *(E.)* Mönkehaus, F. (Inst, Radiation Biol.,
Münster, W. Germany) and W. Köhnlein. *Biopolymers*
2):329-340, 1973.

8 DNA STRUCTURE DAMAGE IN γ-IRRADIATED ANIMAL
TISSUES. *(Rus.)* Komolova, G. S. (A. N.
h Inst. Biochem., USSR Acad. Sci., Moscow), E. N.
fonov and I. A. Yegorov. *Dokl Akad Nauk SSSR*
(1):248-250; 1973.

9 EQUAL SENSITIVITY OF THE TWO STRANDS OF
ϕX174 REPLICATIVE DNA TO BREAKAGE BY
IZING RADIATION. *(E.)* Achey, P. M. (Dept.
robiol., U. Florida, Gainesville) and D. G.
rtney. *Biophys J* 13(1):72-79, 1973.

0 PREVIOUS RADIATION EXPOSURE IN PATIENTS
WITH LEUKEMIA. *(E.)* Rodriguez, V. (U.
as M. D. Anderson Hosp., Tumor Inst., Houston),
P. Bodey and E. J. Freireich. *Proc Am Assoc*
cer Res 13(March):107, 1972.

1 THE DETERMINATION OF BLOOD NUCLEIC ACIDS
FOR THE DIAGNOSIS OF RADIATION-INDUCED
AGE. *(Rus.)* Kritskiy, G. A. (A. N. Bach Inst.
chem. Moscow, USSR) and S. V. Aleksandrov.
l Akad Nauk SSSR 209(3):728-731, 1973.

2 PHOTOALKYLATION OF PURINES IN DNA. *(E.)*
Ben-Ishai, R. (Dept. Biol., Israel Inst.
h., Haifa), M. Green, E. Graff, D. Elad, H.
inmaus and J. Salomon. *Photochem Photobiol* 17(3):
-167, 1973.

See also:

* (Rev): 0632, 0634, 0635, 0639
* (Chem): 0736, 0743

0783 ESTABLISHMENT OF CONTINUOUS LYMPHOBLAST CULTURES FROM LEUKOCYTES OF GIBBONS (*HYLOBATES LAR*).

(E.) Werner, J. (Children's Hosp. Philadelphia, Pa), G. Henle, C. A. Pinto, R. F. Haff and W. Henle. *Int J Cancer* 10(3):557-567, 1972.

Efforts were made to establish continuous lymphoblast cultures from peripheral leukocytes of gibbons (*Hylobates lar*) by co-cultivation with lethally X-irradiated human lymphoblasts from Epstein-Barr virus (EBV)-producing lines of infectious mononucleosis or Burkitt tumor origins. EBV-positive lymphoblast lines were derived from all of five animals tested, of which four had no antibodies to EBV-related antigens and one a marginal level. Growth became evident after an average of 26 days. Several months after injection of autochthonous cultured cells and seroconversion, co-cultivation again yielded lymphoblast lines, but growth became evident on the average within only 55 days. Separate cultures of the X-irradiated lymphoblasts or the leukocytes, whether obtained before or long after seroconversion, died within 2-3 weeks. Even during the period of seroconversion only one line became established in numerous attempts without the benefit of co-cultivation. Injection of cultured lymphoblasts into the authochthonous animals by different routes failed to induce tumors (or any signs of illness) despite attempted immunosuppressive treatment of three of the animals prior to transplantation of the cells. All animals responded with antibody formation to EB viral capsid antigens within 2 to 5 weeks. The time of seroconversion apparently depended upon the percentages of EBV antigen-containing cells in the cultures. Three animals developed low levels of antibodies to the D component of the EBV-induced early antigen complex.

0784 CYTOTOXIC ANTIBODY RESPONSE TO TUMORS INDUCED IN ADULT AND NEWBORN RABBITS BY FIBROMA VIRUS.

(E.) Tompkins, W. A. F. (Ctr. Zoonoses Comp. Med., Urbana, Ill.) and R. M. Schultz. *Infec Immun* 6(4):591-599, 1972.

Cells cultured from tumors induced in rabbits by inoculation of fibroma virus possessed virus-specific cell surface and cytoplasmic antigens. Tumor cell cultures were capable of a limited number of cell divisions before degenerating. Employing the ^{51}Cr-release test and the microcytotoxicity test, it was demonstrated that sera from rabbits with regressed fibroma tumors contained antibodies cytotoxic for cells infected in culture with fibroma virus. These sera were only weakly cytotoxic for cultured fibroma tumor cells. In addition, newborn rabbits bearing progressively growing tumors had serum antibodies cytotoxic for cells infected with fibroma virus in culture but not for fibroma tumor cells. Immunofluorescence studies also showed that the reaction of immune serum with the surface antigens of fibroma tumor cells was markedly weaker than with the surface antigens of cells infected with virus in culture. Furthermore, membrane cytotoxicity and immunofluorescence reaction were significantly reduced when cells were tested after
prolonged incubation or cell division, or both,
following infection with fibroma virus. The failur
of tumors to regress in newborn rabbits in spite
of the presence of cytotoxic antibodies is believed
to be a result of virus-induced membrane antigens
which are lost or altered during tumor growth *in vivo* or tumor cell proliferation in culture, or both. This is supported by the demonstration of a marked reduction of surface immunofluorescence and cytotoxicity reactions with increasing time after infection of cells with fibroma virus in culture.

0785 INDUCTION OF POLYOMA VIRUS BY FLUORESCENT
(VISIBLE) LIGHT IN POLYOMA-TRANSFORMED CE
PRETREATED WITH 5-BROMODEOXYURIDINE.

(E.) Fogel,
(Dept. Genetics, Weizmann Inst. Sci., Rehovot, Isra
Nature [*New Biol*] 241(110):182-184, 1973.

A polyoma virus-(PV)-transformed cell line was es-
tablished after infection of cultures form rat embr
muscle cells with a large plaque PV strain. Induct
of virus synthesis by 5-bromodeoxyuridine (BUdR) ar
fluorescent light treatment was studied in cells of
first generation clone of the large plaque-transfor
(LPT) cell line. These cells contain polyoma spec
tumor (T) antigen and are resistant to infection w
PV. The frequency of cells induced for synthesis
viral-capsid (V) antigen by fluorescent light incr
by a factor of up to 600 in BUdR-pretreated cells
compared with control cells. Production of infect
virus in the BUdR-treated and irradiated cultures
creased by a factor of up to 100. Induction of vi
synthesis was associated with inhibition of cell g
the number of colonies formed by treated cells dec
in proportion to the increase in frequency of cell
taining V-antigen. 2-Mercaptoethylamine-HCl (cyst
mine), which protected cellular DNA against irradi
damage by fluorescent light, increased cell surviv
(colony formation) three- to twenty- fold while de
creasing cell inducibility for V-antigen synthesis
two- to three-fold. These results support the ide
that induction of virus synthesis in LPT cells is
consequence of damage to cellular DNA.

0786 THE CHROMOSOMES OF THE EB VIRUS-POSITIVE
BURKITT CELL LINE P3J.HR1K STUDIED BY TH
FLUORESCENT STAINING TECHNIQUE.

(E.) Petit, P.
(Dept. Path., U. Brussels, Belgium), A. Verhest, F
Lecluse van der Bilt and A. Jongsma. *Pathol Eur*
7(1):17-21, 1972.

Cytogenetic observations were made in a long-term
established Burkitt cell line (P3J.HR1K) harboring
Epstein-Barr virus. Karyotype analysis of 20 meta-
phases by the acetic saline Giemsa staining techni
showed that the majority of metaphases contained 4
chromosomes. Most of the cells were missing a G
group chromosome but showed one to three additiona
chromosomes. The following stable structural anom
lies were identified in ten well-spread metaphases
studied by the quinacrine mustard staining techniq
a) a monosomy for chromosome G21; b) two markers
the D group, D13q+ and D14q+; c) two secondary con

·ictions in the C group affecting one C7 and one
chromosome. The extra chromosomes could not be
ıracterized since their fluorescent banding pattern
·ied from cell to cell.

7 SV40 VIRUS-SPECIFIC RNA SYNTHESIS IN TRANSFORMED HUMAN CELLS. *(E.)* Young, D.

stern Gen. Hosp., Edinburgh, Scotland), J. Gosden
E. Rogers. *Nature* [*New Biol*] 242(114):16-18, 1973.

SV40 specific RNA formed during lytic infection
monkey kidney cells (BSC-1) was compared with that
nd in SV40-transformed human epithelial cells which
e not producing infectious virus. Experiments in
ch cells were labeled with ^{3}H-uridine for one hour
ore collection, extraction, and polyacrylamide gel
ctionation of RNA showed similar patterns of
ridization with SV40 DNA in both transformed and
ductively infected cells. However, transformed
an cells, in contrast to monkey cells, showed six
tinct peaks of hybridization, representing discrete
e fractions of RNA containing sequences complemen-
y to SV40 DNA. Fraction V was the largest peak
hybridization in monkey cells and was present only
e in infection. The largest size of RNA which
ridized (peak I) was clearly present after one
r of labeling only early in infection and in trans-
med cells. When transformed human cells were labeled
18 hr, the pattern of hybridization was significantly
ferent than that obtained after a short label. The
or peak of hybridization was V as in lytic infection.

8 INDUCTION OF LYMPHOMA IN OWL MONKEYS WITH HEATED NON-CYTOPATHOGENIC *HERPESVIRUS*

MIRI. *(E.)* Ablashi, D. V. (Natl. Cancer Inst.,
hesda, Md.), W. F. Loeb, G. Pearson, M. G. Valerio,
R. Armstrong, H. Rabin, E. W. Kingsbury and U.
ne. *Nature* [*New Biol*] 242(114):28-30, 1973.

owl monkeys were given six weekly i.m. inocula-
ns of 0.7 ml heat-treated *Herpesvirus saimiri*
S) shown to be noncytopathogenic in owl monkey
ney (OMK) and Vero cells. Both monkeys developed
ignant lymphoma with lymphogenous leukemia in a
ner identical to animals inoculated with untreated
. Infectious HVS was rescued from whole blood and
sues collected at necropsy by co-cultivation with
o and OMK cells. Ultrastructural examination of
cultivated cells from lymph nodes of one monkey
ealed herpes particles resembling HVS. Serum
ibody titers to HVS detected by serum neutraliza-
n and immunofluorescence developed in both animals.
vivo and *in vitro* studies to explain the mechanism
erlying the oncogenicity of heat-treated virus are
progress.

SUPERCOILING OF SV40 DNA CAN OCCUR INDEPENDENTLY OF REPLICATION. *(E.)* White, M.

t. Biochem., Glasgow U., Scotland) and R. Eason.
re [*New Biol*] 241(106):46-49, 1973.

virus-infected African green monkey kidney cells
-1) were exposed to tritiated thymidine in the presence of puromycin and protein-DNA complexes were extracted with Triton X-100. Puromycin treatment sharply reduced the rate of incorporation of tritiated thymidine into the complexes, which had a greatly reduced average sedimentation coefficient (25S). Dye-buoyant density gradient centrifugation analysis of SV40 DNA in the puromycin complexes indicated a reduction in the number of superhelical turns. SV40-infected cells were again labeled with tritiated thymidine in the presence of puromycin, washed with Eagle's medium containing excess thymidine, and incubated for two hours. Dye-buoyant analysis of Triton extracts and velocity sedimentation analysis on a neutral sucrose gradient indicated that the puromycin complex had acquired normal sedimentation characteristics (44S) and that its constituent DNA had normal superhelic density. Extensive DNA replication occurred when infected cells were washed free of puromycin and tritiated thymidine and incubated in the presence of 5-bromodeoxyuridine. Addition of arabinofuranosylcytosine (ara-C) to the incubation medium completely inhibited DNA replication; however, dye-buoyant analysis of washed Triton extracts from infected cells to which ara-C was added showed that puromycin DNA had acquired normal superhelix density. The introduction of superhelical turns into puromycin DNA apparently occurred by a nicking-reclosing process, independent of DNA replication.

0790 DEFECTIVE MOUSE SARCOMA VIRUS DEFICIENT IN DNA POLYMERASE ACTIVITY. *(E.)* May, J. T.

(Baylor Coll. Med., Houston, Tex.), K. D. Somers and S. Kit. *J Gen Virol* 16:223-226, 1972.

DNA polymerase and endonuclease activities were determined in purified particles of a mouse sarcoma virus-Moloney leukemia virus complex (MSV-MoLV), MoLV, and two clonally derived MSV variants defective in replication (MSV-1) or in transformation and replication (MSV-6). DNA polymerase activity was assayed by (^{3}H)-TTP incorporation in virus protein; endonuclease activity was measured by the conversion of superhelical Form I SV40 DNA to open-circular Form II SV40 DNA. All preparations contained similar levels of endonuclease activity. However, the DNA polymerase activities of MSV-1 and MSV-6 were 25% and 3%, resp., of the activity in MSV-MoLV or MoLV. These findings suggest that DNA polymerase is essential for cell transformation by MSV and is also required for initiation of infection by leukemia viruses.

0791 INDUCTION OF DNase ACTIVITY IN MONKEY CELL CULTURES INFECTED BY SIMIAN VIRUS 40.

(E.) Medrano, L. (Dept. Biol., Massachusetts Inst. Tech., Cambridge). *Arch Gesamte Virusforsch* 39(4):338-343, 1972.

DNA polymerase and DNase activities were assayed in cell-free extracts prepared from monkey CV1 cell cultures at various times after infection with SV40. DNA polymerase activity, assayed by a radioisotope method using denatured calf thymus DNA as primer, rose sharply within 20 hr after infection and continued to increase gradually over the next 40 hr. DNase activity against host and viral DNA, however, did not begin to increase until 40 hr after infection, and activity doubled over the next 20 hr. Crude cell extracts were partially fractionated on Bio-Gel P-200 columns at various times after SV40 infection and DNase activity of the fractions was assayed to determine whether a single species of DNase or multiple enzyme activities were induced. The pattern of activity before infection and at all times after infection was heterogeneous. There did not, therefore, appear to be a specific induction of any single DNase by SV40 infection.

0792 ISOLATION OF A LEUKOVIRUS FROM A CONTINUOUS HUMAN CELL LINE.

(E.) Zhdanov, V. M. (D. I. Ivanovsky Inst. Virol, Moscow, USSR), V. D. Soloviev, T. A. Bektemirov, F. P. Filatov and A. F. Bykovsky. *Arch Gesamte Virusforsch* 39(4):309-316, 1972.

The properties of a C-type virus particle purified from a continuous cell line (J96) derived from leukocytes of a leukemia patient were studied. Yield of virus particles, which banded in sucrose gradients at a density of 1.16 gm/cm^3 and possessed reverse transcriptase activity, could be increased by inhibiting cellular DNA synthesis with mitomycin C or 5-bromo-deoxyuridine. Extracts from J96 cells did not react in immunodiffusion tests using immune sera against Rous sarcoma virus (RSV) or Rauscher leukemia virus (RLV). The C-type particles from the J96 cells could also be distinguished from RLV and RSV by the fact that J96 C-type ^{3}H-labeled DNA prepared in an endogenous reverse transcriptase reaction did not anneal significantly to RNA from these two latter viruses. It did, however, anneal to RNA extracted from the polysomal fraction of spleen and leukocytes of leukemia patients. These findings suggest that the C-type virus isolated from J96 cells is of human origin.

0793 INTRACYTOPLASMIC A PARTICLES: MOUSE MAMMARY TUMOR VIRUS NUCLEOPROTEIN CORES?

Smith, G. H. (Natl. Cancer Inst., Bethesda, Md.) and N. A. Wivel. *J Virol* 11(4):575-584, 1973.

A rapid method for the isolation of intracytoplasmic A particles, the putative intracellular nucleoprotein cores of mouse mammary tumor virus (MTV), is presented. Spontaneous C_3H/He mouse mammary tumors and transplantable mouse Leydig cell tumor were used as source material. Large aggregations of intracytoplasmic A particles were separated from cellular contaminants on discontinuous sucrose gradients and subsequently further purified by isopycnic banding in linear sucrose gradients. The purified particles were solubilized in sodium dodecyl sulfate, and the structural proteins were analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Purified mouse mammary tumor virions were solubilized, and their proteins were analyzed in the same way. Comparison of co-electrophoretic gels indicated a lack of correlation in the molecular size of the major structural proteins in intracytoplasmic A particles and MTV. The three major proteins of the A particles were calculated to be 80,000; 35,000; and 20,000 daltons. Five major polyacrylamide gel electrophoresis bands were obtained with purified MTV; these were 90,000; 69,000; 55,000; 37,000; and 27,500 daltons. These results suggest the need for the reexamination of the current tenet that intracytoplasmic particles present intracellular MTV nucleoprotein cores.

0794 PRODUCTION OF VIRUS BY EMBRYONIC CULTURES CO-CULTIVATED WITH BREAST TUMOUR CELLS OR INFECTED WITH MILK FROM BREAST CANCER PATIENTS.

(E.) Keydar, J. (Dept. Microbiol., U. Tel Aviv, Israel), Z. Gilead, S. Karby and E. Harel. *Nature* [*New Biol*] 241(106):49-52, 1973.

Fifteen cultures were obtained by infection or co-cultivation of mammary and fibroblast embryonic cells with human breast tumor cells (adenocarcinoma or with milk from breast cancer patients. Nine of the cultures gave reproducible levels of virus production. Production of oncornavirions in all nine cultures was confirmed by the presence of particles with a buoyant density of 1.16-1.19 g/ml, reverse transcriptase activity, the presence of 70S RNA, and the presence of a RNA: (^3H)DNA hybrid. Proliferation and virus production continued for at least 11 months of continuous passage in all lines.

0795 TRANSFORMATION OF CHICK-EMBRYO FIBROBLAST BY WILD-TYPE AND TEMPERATURE-SENSITIVE ROUS SARCOMA VIRUS ALTERS ADENYLATE CYCLASE ACTIVIT

(E.) Anderson, W. B. (Natl. Cancer Inst., Bethesda, Md.), G. S. Johnson and I. Pastan. *Proc Natl Acad Sci USA* 70(4):1055-1059, 1973.

The activity of the enzyme adenylate cyclase, a component of the plasma membrane, has been determin in chick-embryo fibroblasts and in cells transformed by either Bryan high-titer strain of Rous sarcoma virus (RSV-BH) or a temperature-sensitive mutant of this virus (RSV-BH-Ta). Adenylate cyclas activity is reduced in cells transformed by the wild-type virus and also by the temperature-sensitive mutant when the cells are grown at the permissive temperature (37 C). Transformation results in an altered affinity (K_m) for the substrate (MgATP). The apparent K_mATP is 0.23 mM in normal cells and 1.1 mM in cells transformed with wild-type virus. The K_mATP of the cells infected with RSV-BH-Ta is 0.67-1.0 mM at 37 C and 0.28 mM at 42 C. The enzyme from normal cells appears to have two binding sites for Mg^{++}, one at the catalytic site and a second at a regulatory site. Transformation by RSV-BH or RSV-BH-Ta (37 C) apparently alters this second Mg^{++} site. A decrease in adenylate cyclase activity occurs within 10 min after cells infected with RSV-BH-Ta are shifted from 42 C to 37 C; the activity falls to one-half that of normal cells 30 min after the temperature shift. These observations indicate that a viral function lowers cyclic AMP content by lowering the activity of adenylate cyclase, probably through some modification of the plasma membrane.

796 CYCLIC VARIATION IN SUSCEPTIBILITY OF BALB-C 3T3 CELLS TO POLYOMA VIRUS. *(E.)*

horne, H. V. (Dept. Microbiol., U. Sherbrooke, rovince of Quebec, Canada). *J Gen Virol* 18:163-69, 1973.

he susceptibility of synchronized Balb/c 3T3 cells o infection by polyoma virus was studied in elation to the cell cycle. Virus production was etermined by hemagglutination assay or by CsCl quilibrium centrifugation of virus particles adioactively labeled during the course of infection. oth high and low susceptibility clones synchronized y mitotic harvest, double thymidine block, or olcemid showed a cyclic pattern of maximum sus- eptibility to polyoma virus infection which was ocated at, or near, the begining of G1. Cells howed minimal susceptibility to virus infection uring S phase.

797 THE PRODUCTION OF INFECTIVE VIRUS BY SV40-TRANSFORMED HUMAN RENAL CELLS. *(E.)*

oordaa, J. Van Der (U. Amsterdam, The Netherlands) nd W. Pauw. *J Gen Virol* 18:201-202, 1973.

irus production by cells of the post-crisis NB- ine of SV40-transformed human cells was studied y infectious center assay. These NB cells ontinued to produce infectious virus (10-100 .f.u./ml). In addition, all seven primary clones, 3 secondary clones, and seven tertiary clones tudied also continued to produce infectious SV40. t was thus concluded that every cell in the arental NB line was capable of virus production. ne progeny virus of each clone gave rise to nall, medium and large plaques suggesting that ch cell possibly harbored several distinct rus genomes.

798 PAPOVA VIRUS IN URINE AFTER RENAL TRANSPLANTATION. *(E.)*

Lecatsas, G. (Vet. Res. nst., Onderstepoort, Pretoria, South Africa), . W. Prozesky, J. Van Wyk and H. J. Els. *Nature* 41(5388):343-344, 1973.

lectron microscopic studies revealed the presence f papova-type virus particles in the urine of eight f 13 patients who had received renal allografts 3-22 months previously. The particles corresponded ı size and morphology to the polyoma-SV40 subgroup. o particles were observed in urine of eight staff embers or in antilymphocyte serum. The serum of ne patient who excreted the largest number of articles contained antibody against them. The ack of correlation between virus occurrence and ate of transplant suggested that virus excretion ccurred at intervals depending on antibody status.

99 EFFECTS OF ADRENAL GLUCOCORTICOIDS ON POLYOMA VIRUS REPLICATION. *(E.)*

Morhenn, (Dept. Biochem. Biophysics, U. California, San ancisco), Z. Rabinowitz and G. M. Tomkins. *Proc tl Acad Sci USA* 70(4):1088-1089, 1973.

Treatment of polyoma-virus-infected fibroblasts from mouse embryo cells and virus-infected 3T3 cells with physiological concentrations of dexamethasone and cortisol stimulated plaque formation at least ten-fold. Epicortisol, estradiol and progesterone were without effect, the latter hormone actually inhibiting the action of dexamethasone when simultaneously administered. These data suggest that glucosteroids enhance virus multiplication and that the enhancement involves cytoplasmic receptors.

0800 AN INFECTIOUS CENTRES TECHNIQUE FOR THE ASSAY OF INFECTIOUS POLYOMA VIRUS DNA.

(E.) Naude, W. du T. (Med. Sch., Observatory, Cape South Africa) and C. I. Raaff. *J Gen Virol* 18: 405-408, 1973.

A modified infectious centers technique is described for the titration of the LID-I strain of polyoma virus DNA purified from infected mouse kidney cell cultures. The sensitivity of the technique is enhanced by the addition of an appropriate dilution of dimethyl sulfoxide (DMSO). Although DMSO had no effect on infectivity of viral DNA in hypertonic 0.55 M NaCl when assayed on secondary mouse embryo cell cultures, 15% DMSO (V/V) in isotonic phosphate-buffered saline showed an optimal enhancing effect. The number of infectious centers depended on the concentration of cells present in the assay. When more than 10^6 cells/ml were present in the cell-viral DNA mixture, the resultant plaque count increased exponentially. It is thus essential that both an adequate and comparable number of cells be present from one assay to another if different preparations are to be compared. The fact that viral DNA preparations previously exposed to high concentrations of cells still contained infectious DNA when reassayed indicated that this new infectious centers technique requires additional improvements.

0801 ONCOGENICITY OF NON-TRANSFORMING MUTANTS OF AVIAN SARCOMA VIRUSES. *(E.)*

Biggs, P. M. (Houghton Poultry Res. Station, Huntingdon, England), B. S. Milne, T. Graf and H. Bauer. *J Gen Virol* 18:399-403, 1973.

Two non-transforming mutants of Rous sarcoma virus, one (NC-SRV-H) originated by point mutation and the other (NC-SRV-1) by deletion, were studied for their oncogenicity in leukosis-sarcoma virus-free Brown and White Leghorn chicks. One-day-old chicks were inoculated into the wing web i.p. with one of the two virus preparations and were thereafter examined daily for wing-web tumors. No sarcomas were observed in the wing webs of either strains of chick, indicating that both non-transforming mutants had lost their ability to produce tumors characteristic of their parent viruses. Although no wing-web tumors arose in treated Brown Leghorn chicks, the incidence of tumors in White Leghorn chicks was 35.6% following inoculation with NC-SRV-I and 53.5% with NC-SRV-H. The tumors consisted primarily of erythroid leukosis and lymphoid leukosis. Virus isolated from one NC-SRV-I and two

NC-SRV-H-induced lymphoid tumors were non-transforming in cell culture, and, based on interference tests, each belonged to the same subgroup as the mutant which produced the respective tumor. The ability of these non-transforming mutants of Rous sarcoma virus to produce tumors characteristic of leukosis viruses suggests there is more than one gene in the genome of leukosis/sarcoma viruses responsible for neoplasia and also indicates that leukosis viruses can originate from sarcoma viruses through loss of genetic material.

0802 DIBUTYRYL CYCLIC ADENOSINE 3' 5' MONOPHOSPHATE, SUGAR TRANSPORT AND REGULATORY CONTROL OF CELL DIVISION IN NORMAL AND TRANSFORMED CELLS.

(E.) Grimes, W. J. (Coll. Med., U. Arizona, Tuscon) and J. L. Schroeder. *J Cell Biol* 56(2):487-491, 1973.

The effects of dibutryl cyclic AMP (dBcAMP) were studied on 2-deoxyglucose transport and growth of normal and polyoma virus-transformed Swiss 3T3 cells. Addition of 0.2 mM dBcAMP and 2 mM theophylline to the medium of growing transformed 3T3 cells restored density-dependent inhibition of cell division to limits comparable to that of normal 3T3 cells. Accordingly, the ability of transformed 3T3 cells to transport ^{3}H-labeled 2-deoxyglucose was significantly reduced following treatment with dBcAMP. The rate of uptake of 2-deoxyglucose into both normal and transformed cells was high at lower cell densities during logarithmic growth, but was reduced to comparable rates at high cell densities. DBcAMP was lethal to low density transformed cell cultures, but was inhibitory with respect to growth of medium and high density cultures. It appeared that dBcAMP worked by limiting the number of cell divisions following addition to the medium. Removal of dBcAMP in all but low density cultures resulted in recovery of the ability to divide and incorporate 2-deoxyglucose at high rates within 24 hr. DBcAMP could not inhibit the rapid increase in 2-deoxyglucose uptake into fresh serum-stimulated normal 3T3 cells. Fresh serum did not stimulate 2-deoxyglucose transport in dBcAMP-treated transformed cells.

0803 CONTACT-INHIBITED REVERTANT CELL LINES ISOLATED FROM SV40-TRANSFORMED CELLS. IV. MICROFILAMENT DISTRIBUTION AND CELL SHAPE IN UNTRANSFORMED, TRANSFORMED, AND REVERTANT BALB/C 3T3 CELLS.

(E.) McNutt, N. S. (Massachusetts Gen. Hosp., Boston), L. A. Culp and P. H. Black. *J Cell Biol* 56(2):412-428, 1973.

A comparison was made of the ultrastructure of the cell periphery in three cloned cell lines: untransformed Balb/c 3T3 cells, SV40-transformed Balb/c 3T3 cells, and revertant cells obtained from the transformed cell line by a selection technique utilizing concanavalin A. Both thin-section and surface replication techniques were used for *in situ* examination of the cell lines. Microfilaments, 70 Å in diameter (called alpha filaments), were abundant in untransformed and revertant cell lines, particularly in the anterior expansions of the cells, which tended to have many microvilli and small pseudopodia. Alpha filaments were diminished in the anterior expansions of transformed cells, which contained large blunt pseudopodia and relatively few microvilli. Surface replicas confirmed the impression gained from thin sections that transformed cells have a greater proportion of their cell surface involved in bulging pseudopodia than either untransformed or revertant cells. Since alpha filaments bind heavy meromyosin and are similar to F-actin, these filaments are thought to be important in cell motility. These observations suggest that a close relationship exists between decreased alpha filaments, bulging pseudopodia, and loss of contact inhibition of movement in transformed cells.

0804 HEMOGLOBIN SYNTHESIS IN MURINE VIRUS-INDUCED LEUKEMIC CELLS *IN VITRO*. III. EFFECTS OF 5-BROMO-2'-DEOXYURIDINE, DIMETHYLFORMAMIDE AND DIMETHYLSULFOXIDE.

(E.) Scher, W. (Mt. Sinai Sch. Med., City U., New York, N.Y.), H. D. Preisler and C. Friend. *J Cell Physiol* 81(9):63-70, 1973.

The effect of 5-bromo-2-deoxyuridine (BUdR) on the enhancement of hemoglobin synthesis in Friend leukemia cells by dimethylsulfoxide (DMSO) and dimethylformamide (DMF) was studied *in vitro*. DMSO, which produced a 100-fold increase in the incorporation of transferrin-bound ^{59}Fe over that in controls, was more effective in stimulating heme synthesis than DMF which induced a 32-fold increase. BUdR at a concentration of 3 µg/ml inhibited DMSO-or DMF-stimulated heme synthesis in Friend leukemic cells by 70% after four days of growth. DMSO plus BUdR produced only a 40% inhibition of cell growth during this period. The rates of RNA, DNA and protein synthesis in the presence of BUdR and DMSO or DMF followed the pattern of cell multiplication. The inhibitory effect of BUdR was reduced when it was added after the first two days of growth in DMSO or DMF. No effect was seen when BUdR was added on day three when heme synthesis was maximal. The inhibitory effect of BUdR could be prevented by simultaneous addition of an excess of thymidine but not by uridine. This effect correlated with the ability of thymidine, but not uridine, to prevent incorporation of ^{3}H-BUdR into DNA.

0805 THE RESPONSE OF NORMAL AND POLYOMA VIRUS-TRANSFORMED BHK/21 CELLS TO EXOGENOUS PURINES.

(E.) Clarke, G. D. (Imperial Cancer Res. Fund Lab., London, England) and C. Smith. *J Cell Physiol* 81(1):125-132, 1973.

The differential effects of exogenously supplied purines on normal and polyoma virus-transformed BHK21 cells were studied. Adenosine (10 µM) appreciably stimulated the initial growth rate of normal BHK21 cells seeded at low, but not at high, densities. The final cell density of the treated cultures was not altered compared with that of untreated cultures, and adenosine did not permit growth of normal cells in agar. Adenosine also consistently more than doubled the response of quiescent cells to stimulation by medium containing 2% serum, as determined by the extent of ^{3}H-thymidine incorporation. This effect was only slightly decreased when serum was used which was dialyzed to remove oxypurines, indicating that BHK21 cells can endogenously synthesize purines. Adenosine was also able to prevent the inhibitory effects of dextran sulfate and heparin on the response of quiescent cells to medium with 5% serum. ATP, adenine, hypoxanthine and 1-methyladenine and its riboside were also active in increasing the response of quiescent cells to 2% serum stimulation. Xanthine, which cannot be converted to ATP, was not active, and it was concluded that nucleotide synthesis was necessary for the above observed effects. In contrast to normal cells, the growth of polyoma-transformed BHK21 cells was not stimulated by adenosine; however, their ability to form colonies in agar was increased five-fold.

0806 EVIDENCE FOR NATURALLY OCCURRING MURINE SARCOMA VIRUS. *(E.)* Ball, J. K. (Cancer Res. Lab., U. Western Ontario, London, Canada), D. Harvey and J. A. McCarter. *Nature* 241(5387): 272-275, 1973.

A murine leukemia virus (MuLV) was purified by multiple cloning from productive JLS-V9 cells. The virus (JLS-V9MuLV) contained no detectable transforming murine sarcoma virus (MuSV) yet was able to induce typical MuSV-like multiple sarcomas following i.p. injection into newborn or X-irradiated adult mice. The mice inoculated with JLS-V9 MuLV, however, showed no spleen enlargement. Plasma from tumor-bearing mice and extracts of JLS-V9MuLV-induced tumors produced sarcomas in NIH Swiss, Balb/c and CFW/D mice and showed both MuSV and MuLV activity *in vitro*. CFW/D mice inoculated with plasma from mice bearing JLS-V9MuLV-induced tumors developed two populations of sarcomas, one with a hort and another with a long latent period. It was concluded that the early arising tumors were derived from a focus-forming virus and that the ate appearing tumors arose from a sarcoma-inducing ctivity activated by the original focus-forming irus.

807 INDUCTION OF VIRUS SYNTHESIS IN POLYOMA-TRANSFORMED BHK-21 CELLS. *(E.)* Folk, W. R. Imperial Cancer Res. Fund, London, England). *J irol* 11(13):424-431, 1973.

HK-21 cells were transformed with polyoma virus utants Ts-a and Ts-25 by using a temperature hift from 31 to 39 C at 5 days after infection so hat rescuable transformants could be isolated. lones which yielded virus after fusion with mouse ells were scored and maintained at 39 C in the resence of antipolyoma virus antiserum. Generally, o infectious viral deoxyribonucleic acid (DNA) ould be found in Hirt supernatant fractions of these lines when maintained at 39 C, but DNA-DNA reannealing measurements detected two to six viral genomes per diploid cell genome in the nuclear DNA. Fusion with permissive cells was not necessary to induce the synthesis of infectious virus; cell lines shifted to 31 C produce the equivalent of 100 viral genomes per cell after 5 days. In some cell lines up to 1% of the cells formed infectious centers upon a shift to 31 C, and 100% of the subclones of a line were inducible. Growth at 31 C selected for a noninducible population which was still transformed.

0808 PROTEINS AND GLYCOPROTEINS IN PLASMA MEMBRANE FRACTIONS OF AVIAN LEUKOSIS-SARCOMA VIRUS SUSCEPTIBLE AND RESISTANT CHICKEN EMBRYO FIBROBLASTS. *(E.)* Smith, E. J. (USDA, Animal Physiology Genetics Inst., Beltsville, Md.) and L. B. Crittenden. *Biochim Biophys Acta* 298(3):608-619, 1973.

^{3}H- or ^{14}C-labeled proteins and glycoproteins from plasma membrane-enriched fractions of avian leukosis virus susceptible and resistant chick embryo fibroblasts were solubilized with sodium dodecyl sulfate and compared after polyacrylamide gel electrophoresis at pH 9.0. No apparent differences in electrophoretic patterns were observed when both phenotypes were cultured in the presence of radioactive leucine or glucosamine. A major leucine-rich polypeptide of apparent molecular wt 42000 was common to both cell types whereas most of the glycoproteins components were found in the molecular wt range 82000-110000. Leucine, glucosamine and fucose were present in a major low molecular weight component which migrated near bromophenol blue. Limited studies comparing fucose-labeled proteins suggested that phenotypic differences may occur in plasma membrane fucoproteins.

0809 ULTRASTRUCTURAL DISTRIBUTION OF CONCANAVALIN-A RECEPTORS ON HAMSTER EMBRYO AND ADENOVIRUS TUMOUR CELL CULTURES. *(E.)* Rowlatt, C. (Inst. Sci. Res. Cancer, Villejuif, France), R. Wicker and W. Bernhard. *Int J Cancer* 11(2):314-326, 1973.

An electron microscope cytochemical reaction, based on the specificity of concanavalin A for certain sugar residues, was used to study the surface coat of cells in cultures of an Adenovirus 12 hamster tumor cell line (TAd$_{12}$ XIV), of cell strains from other hamster tumors induced by simian Adenovirus 7 (SA$_7$) and of secondary cultures of normal hamster embryo cells. When the reaction was performed on living cells, *in situ* in the bottles in which they had been grown, a uniform surface coat of positive material was found on the normal embryo cells. The tumor cells were also positive, but the reaction product had a tendency to be more patchily distributed over the surface. This discontinuity of the labelling was particularly pronounced in the strain TAd$_{12}$ XIV, but was less visible in the highly malignant strains SA$_7$. When the reaction was performed on cultures previously fixed with formaldehyde, no difference was found between the three types of cell cultures. Simple stereological analysis confirmed these findings. It was concluded that loss of material occurred differentially between the different types of culture during the stages of the reaction before fixation. The possibilities of this loss being due to physicochemical differences in the cell surface material or to underlying metabolic changes are discussed.

0810 HOMOLOGY BETWEEN EPSTEIN-BARR VIRUS DNA AND VIRAL DNA FROM BURKITT'S LYMPHOMA AND NASOPHARYNGEAL CARCINOMA DETERMINED BY DNA-DNA REASSOCIATION KINETICS. *(E.)* Nonoyama, M. (U. North Carolina Med. Sch., Chapel Hill) and J. S. Pagano. *Nature* 242(5392):44-47, 1973.

DNA-DNA reassociation kinetics studies were conducted to estimate the number of Epstein-Barr Virus (EBV) genome equivalents in the nonproducer Raji cell line, and to determine whether viral DNA purified from test cells or Burkitt's lymphoma and nasopharyngeal sarcomoma biopsies was identical to EBV DNA isolated from producer HR1K cells. Hybridization of Raji cell DNA to high specific activity ^{3}H-labeled EBV DNA indicated that each cell contained approximately 53 to 55 EBF genome equivalents. Reassociation kinetics studies also showed that one Burkitt's lymphoma biopsy contained 49.4 genome equivalents/cell and that one nasopharyngeal carcinoma biopsy contained 19.2 EBV genomes/cell. The reassociation curves for the DNA from Raji cells and the two biopsies were identical to the reassociation curve for purified EBV DNA, indicating that all three cell types contained complete or nearly complete viral genomes.

0811 AN INVESTIGATION OF FACTORS INFLUENCING THE SUSCEPTIBILITY OF BALB-c 3T3 CELLS TO POLYOMA VIRUS. *(E.)* Thorne, H. V. (Dept. Microbiol., U. Sherbrooke, Quebec, Canada). *J Gen Virol* 18:153-162, 1973.

The replication of polyoma virus was studied in Balb-c 3T3 monolayer cultures. Virus yield/cell from infected Balb-c 3T3 cultures was found to decrease with increasing cell density. Close cell contact *per se* did not inhibit yield nor did moderate depletion of medium constituents by cell growth. Absorption of input virus/cell decreased as the cultures aged. Addition of fresh medium to resting cultures followed by replating of the cells at lower densities provoked a delayed increase in susceptibility. Treatment of resting cultures with trypsin initially enhanced their susceptibility, but a gradual decline to the original level followed; absorption of input virus followed a similar course. Total yield of virus, as estimated by hemagglutination assay, was not increased, however, when infected resting monolayers were trypsinized and replated at the same density. These results are consistent with the interpretation that only cells in a state of active division are susceptible and that resting cells acquire a trypsin-sensitive surface component which blocks infection. Yields of virus from sparse cultures of several clones tested varied. Study of the clone of highest susceptibility revealed that cell DNA synthesis was not induced by virus infection and that the total amount of virus DNA made was small and closely equivalent to the amount found encapsidated.

0812 PURIFICATION BY OLIGO(dT)-CELLULOSE OF VIRAL-SPECIFIC RNA FROM SARCOMA VIRUS-TRANSFORMED MAMMALIAN NONPRODUCER CELLS. *(E.)* Scolnick, E. M. (Natl. Cancer Inst., Bethesda, Md.), R. Benveniste and W. P. Parks. *J Virol* 11(4):600-602, 1973.

Virus-specific RNA was purified from Kirsten sarcoma virus-transformed nonproducer rat kidney cells by oligo(dT) cellulose chromatography. Eight to 15 μg of the purified RNA saturated a ^{3}H-DNA product made from an endogenous reverse transcriptase reaction as compared to 400 to 500 μg for the unpurified starting material, representing a 30- to 60-fold purification. Comparison of the hybridization characteristics of column purified RNA with those of 70S viral RNA suggested that approximately 3% of the (dT) cellulose purified RNA from nonproducer cells represented virus-specific RNA.

0813 STUDIES ON INVASIVENESS OF POLYOMA VIRUS TRANSFORMED BHK CELLS ON CHORIO-ALLANTOIC MEMBRANE OF CHICK. *(E.)* Palayoor, S. T. (Cancer Res. Inst., Tata Mem. Ctr., Bombay, India) and B. K. Batra. *Indian J Exp Biol* 10(27):87-90, 1972.

The penetration pattern and invasive character of polyoma virus-transformed BHK cells (PY cells) was studied following explantation onto the chorioallantoic membrane (CAM) of chicken eggs. Two days after explantation, microscopic observation of fixed and stained specimens revealed the presence of tumor cells in aggregates on the surface of the CAM. Central necrosis of the PY cell aggregation was evident, and a few cells could be seen to penetrate the CAM. PY cells completely penetrated the CAM by four days after explantation. Invading cells either formed small groups with a few giant cells or were present as large colonies. Mesenchymal cells were found in large numbers near the tumor cells. By day six, the CAM showed severe inflammatory response with a hyperplastic mesenchyme and infiltrating leucocytes. Isolated giant PY cells were found in areas distant from established tumor colonies, reflecting their invasive nature. Once in the CAM, PY cells showed no definite nodular or colony patterns as described previously for other tumors.

0814 STRUCTURAL CHARACTERISTICS OF SOME MURINE RNA TUMOR VIRUSES STUDIED BY LACTOPEROXIDASE IODINATION. *(E.)* Witte, O. N. (Stanford U. Sch. Med., Calif.), I. L. Weissman and H. S. Kaplan. *Proc Nat Acad Sci USA* 70(1):36-40, 1973.

Radiation leukemia virus (RadLV), Moloney leukemia

s (MLV) and mouse mammary tumor virus (MTV) were
ified on discontinuous sucrose gradients and
jected to lactoperoxidase iodination. Protein
stituents of the viruses were examined in
assie blue-stained 7% acrylamide gel electro-
esis studies. RadLV and MLV showed distinctive
similar protein spectra; each had six different
r protein bands in which the Coomassie blue-
tive material was found mostly in the four
r molecular wt proteins (10,000-30,000). MTV
ed five protein bands. Lactoperoxidase iodi-
on labeled one major protein in each virus:
0,000 dalton protein in RadLV and MLV, and
00 dalton protein in MTV. These were thought
e enzyme-accessible surface proteins, while the
r molecular wt protein bands were thought to
nzyme-inaccessible proteins.

ISOLATION OF RNA VIRUS FROM PAPILLARY TUMORS OF THE HUMAN RENAL PELVIS. *(E.)*

ott, A. Y. (U. Minnesota Hlth. Sci. Ctr.,
eapolis), E. E. Fraley, P. Cleveland, A. E.
ro and N. Stein. *Science* 179(4071):393-395,
.

astructural studies of primary transitional
carcinomas of the renal pelvis removed from
e adult males revealed the presence of cyto-
mic C-type and A-type virus particles. A-type
icles were also detected in normal kidney
ue. Primary tumor cell cultures, but not
al kidney cell cultures from the patients,
inued to produce infective C-type virus over a
month period. Although the C-type virus
icles banded in sucrose-TRIS gradients at 1.14
.17 gm/ml, incorporated ^{3}H-orotic acid, and
essed RNA dependent DNA polymerase activity,
differed from known C-type viruses in that
were cytopathic to human and chick embryo
s and they did not appear to reproduce by
ing from the cell membrane. The C-type in-
ivity was ether sensitive and the virus did not
utinate or adsorb human erythrocytes. Virus
ralization studies indicated that the agents
all three tumors were the same virus. Serum
all three tumor patients neutralized virus
n directly from two of the three tumors as
as virus passaged once in chick embryo fibro-
t cultures. These studies suggest that this
t is not a known human RNA virus.

RAPID VIRAL INDUCTION OF MURINE LYMPHOMAS IN THE GRAFT-*VERSUS*-HOST REACTION. *(E.)*

lius, E. A. (Yale U. Sch. Med., New Haven,
). *J Exp Med* 136(6):1533-1544, 1972.

J x C57BL/1)F$_1$ mice were given five weekly
injections of spleen cells from SJL/J donors.
days after treatment graft-*versus*-host reactions
) were noted and by 40 days, all treated mice
oped reticulum cell sarcomas. Tumors from
(SJL x C57BL/1)F$_1$ mice treated with viable
n cells were transplanted into syngeneic mice.
ransplants were accepted. However, when these transplanted tumors were in turn transplanted to syngeneic, SJL/J, C57BL/1, (NZB x SJL/J)F$_1$ or NZB mice, only syngeneic mice and C57BL/1 mice accepted transplants. Similar results were seen with transplants from tumors originally induced by frozen- and thawed spleen cells. Cell-free tumor preparations from (SJL/J x C57BL/1)F$_1$ given mice viable spleen cells produced tumors in newborn C57BL/1 or (C57BL/1 x A)F$_1$ mice. These results provide conclusive evidence for a viral etiology of GVHR-induced tumors.

0817 MINOR BASE COMPOSITION OF "70s-ASSOCIATED" 4S RNA FROM AVIAN MYELOBLASTOSIS VIRUS.

(E.) Rosenthal, L. J. (Massachusetts Gen. Hosp., Boston) and P. C. Zamecnik. *Proc Natl Acad Sci USA* 70(3):865-869, 1973.

The minor base composition of "70S-associated" 4S RNA of avian myeloblastosis virus (AMV) purified from the plasma of infected chickens was determined following treatment of the 70S fraction with dimethylsulfoxide. Base composition was analyzed by an isotope derivative method involving the stoichiometric incorporation of ^{3}H-label into periodate-oxidized enzymatic digests of "70S-associated" 4S RNA, chromatographic separation of the labeled digests into individual nucleoside derivatives, and evaluation by liquid scintillation counting. The "70S-associated" 4S RNA contained all the minor bases previously reported for the "free" 4S RNA from AMV and for myeloblast 4S RNA. As observed with "free" 4S RNA from AMV, the concentrations of all methylated guanines were elevated and the concentrations of all modified pyrimidines (with the exception of pseudouridine) were depressed compared with normal or neoplastic host-cell 4S RNA. No differences in methylated adenine composition were observed. This minor base content, plus the capacity to esterify amino acids, established that the "70S-associated" 4S RNA contains transfer RNA. The finding that this fraction was undermethylated suggests that the "70S-associated" 4S RNA also contains nontransfer RNA. This undermethylated RNA might represent either degradation products of high molecular wt RNA or indigenous 4S RNA of undetermined function.

0818 GROWTH REGULATION AND TUMOUR FORMATION OF NORMAL AND NEOPLASTIC RAT CELLS.

(E.) Weiss, R. A. (Dept. Anat. Embryol., U. Coll. London, England), P. Vesely and J. Sindelarova. *Int J Cancer* 11(1):77-89, 1973.

The oncogenicity of cells in syngeneic hosts was compared with the regulation of cell growth *in vitro*. Normal embryonic fibroblasts (LWF) derived from inbred Lewis rats were not tumorigenic when inoculated s.c. or i.v. into syngeneic females. Spontaneously transformed fibroblasts (LW13) and Rous sarcoma virus transformed epithelioid cells (RsK4) both yielded sarcomas following s.c. or i.v. inoculation. RsK4 cells gave rise to tumors after a short (<15 wk) latent period after which no further tumors developed, whereas rats inoculated with LW13 cells continued to develop tumors after this time. RsK4 and LW13 derived tumor cells were histologically similar, being primarily spindle-shaped. RsK4 derived tumors, however, tended to be more pleomorphic. Tumorigenic potential correlated well with a high plating efficiency on plastic or in soft gel suspension and a decreased sensitivity to topoinhibition *in vitro*. RsK4 cells initiated DNA synthesis in low serum medium whereas LWF and LW13 cells did not. Neither LW13 nor RsK4 cell proliferation was inhibited by contact with confluent, static 3T3 or normal LWF cells. The degree to which altered cells responded to the regulatory contact signals of normal LWF cells appeared to be the best *in vitro* indicator of oncogenicity.

0819 "SPONTANEOUS" RELEASE OF TYPE C VIRUSES FROM CLONAL LINES OF "SPONTANEOUSLY" TRANSFORMED BALB/3T3 CELLS.

(E.) Todaro, G. J. (Natl. Cancer Inst., Bethesda, Md.). *Nature [New Biol]* 240(100):157-160, 1972.

Transformed and untransformed BALB/c 3T3 and random-bred Swiss mice cells were studied for the induction of endogenous type C viruses. A radiation-transformed and two spontaneously transformed BALB/c 3T3 subclones grew to high saturation densities, were capable of growing in serum factor-free medium, and were tumorigenic in newborn BALB/c mice in conditions where untransformed cells did not produce tumors. One of the spontaneous transformants (an epithelioid variant) and the radiation transformant produced high titers of type C virus for many months after single exposure to BrdU or IdU. On two occasions the epithelioid variant spontaneously released virus without added inducers, as did 6 of 11 subclones and a subclone of a transformed fibroblastic clone. Virus-free spontaneously transformed cells gave rise to virus-producing cells from 40-200 generations after cloning. An SV40-transformed clone and spontaneously transformed clones of ramdom-bred Swiss mice showed the same pattern of exogenous virus susceptibility as BALB/c cells. No virus production was detected in untransformed BALB/c or random-bred 3T3 cells.

0820 RNA-LINKED SHORT DNA FRAGMENTS DURING POLYOMA REPLICATION.

(E.) Magnusson, G. (Karolinska Inst., Stockholm, Sweden), V. Pigiet, E. L. Winnacker, R. Abrams and P. Reichard. *Proc Natl Acad Sci USA* 70(2):412-415, 1973.

Nuclei from polyoma virus-infected mouse cells were studied to investigate the mechanism of elongation of daughter DNA strands of the polyoma replicative intermediate in this system. Radioactive polyoma DNA showed a sharp peak at 4-5S, suggesting that this 4-5S DNA is synthesized as an intermediate in the synthesis of longer chains. ATP, CTP, GTP and UTP all stimulated the formation of the 4-5S DNA. Polyoma replicative intermediates were subjected to equilibrium centrifugation in neutral Cs_2SO_4. Results indicated a linkage of polyoma DNA to RNA at the 5'-end of the short DNA chains. This finding was confirmed by the recovery of isotope from α-^{32}P-labeled deoxynucleoside triphosphates in 2'(3')-ribonucleotides after alkaline hydrolysis. Treatment of polyoma cells *in vitro* with hydroxyurea resulted in formation of newly synthesized, single stranded, RNA-deficient DNA fragments which were polyoma-specific and which sedimented for the most part at 4S.

0821 MAMMARY TUMOUR VIRUS SPECIFIC NUCLEOTIDE SEQUENCES IN MOUSE DNA.

(E.) Varmus, H. E. (San Francisco Med. Ctr., Calif.), J. M. Bishop, R. C. Nowinski and N. H. Sarkar. *Nature [New Biol]* 238(34):189-191, 1972.

Mammary tumor virus (MTV) DNA was extracted from virus isolated from RIII mouse milk and single and double stranded DNAs were separated by batch elutions from hydroxyapatite powder. Sixty-five percent of the DNA eluted as double stranded material, but when the DNA was denatured and reassociated, only 50% reformed as duplexes. About half the reannealed DNA hybridized with MTV 70S RNA. The remaining DNA showed reassociation kinetics like those of double stranded Rous sarcoma virus and murine leukemia virus DNA. Double stranded MTV DNA was used as a probe for virus-specific nucleotide sequences in DNA from GR and C57BL/6 mouse liver cells and from virus-producing nontumorous mammary tissue of GR mice. DNA from liver cells accelerated the annealing rate of MTV double stranded DNA, indicating that about 90 copies of the MTV sequence were present per diploid liver cell.

0822 HOMOLOGY BETWEEN RNA FROM HUMAN MALIGNANT BREAST TISSUE AND DNA SYNTHESIZED BY MILK PARTICLES.

(E.) Das, M. R. (Tata Inst., Bombay, India), E. Sadasivan, R. Koshy, A. B. Vaidya and S. M. Sirsat. *Nature [New Biol]* 239:92-95, 1972.

RNA-dependent DNA polymerase was isolated from milk particles obtained from Hindu women. The DNA produc of the transcriptases were hybridized with polysomal RNAs from human mammary carcinoma, human normal breast, human placenta and mouse embryo. DNA from milk transcriptase hybridized with polysomal RNAs from an undifferentiated human breast carcinoma but not with RNAs from normal breast, placenta or mouse embryo. Polysomal RNA from the breast carcinoma showed no homology with DNA from avian myeloblastos virus reverse transcriptase.

0823 ONCOGENICITY OF PLAQUE VARIANTS OF CELO VIRUS IN NEWBORN HAMSTERS.

(E.) Pendola, R. G. (Animal Path. Dept., U. Rhode Island, Kingsto V. J. Yates, R. E. Wolke and V. Jastry. *J Natl Cancer Inst* 50(3):791-795, 1973.

Small-plaque (SP) and large-plaque (LP) variants of chicken-embryo-lethal-orphan (CELO) virus, an avian adenovirus, were tumorigenic in 26 newborn

msters who survived s.c. or i.p. inoculation with
^{8} plaque-forming units of virus. There was no
gnificant difference in tumor incidence between
e two virus variants or in latent period of in-
ction. Tumors appeared at the site of s.c. in-
ulation. With the i.p. route, tumors were induced
the peritoneal cavity and liver. The s.c. and
ritoneal tumors induced by the SP and LP variants
re classified histologically as anaplastic tumors,
e liver tumors as bile-duct carcinomas. Sera from
mor-bearing hamsters contained CELO-specific T
tibodies.

24 STUDIES ON CELL FUSION AND THE RESCUE OF SV40 FROM CELL CULTURES OF SUCCESSIVE
NERATIONS OF Cl_2TSV_5-AND RHaT-PRODUCED HAMSTER
MORS.

(E.) Menezes, J. (Fac. Med., U. Ottawa,
tario, Canada) and E. Perry. *Can J Microbiol*
(1):119-127, 1973.

msters were inoculated s.c. with cells of either
two SV40-transformed lines: Cl_2TSV_5, from
hamster tumor; and RHat, from SV40-transformed
mster kidney cells in culture. Five successive
nerations of tumors were produced by inoculation
th these cells. Tumor cells were fused with monkey
lls by mediation of UV-inactivated Sendai virus.
40 could be rescued from all cultures of Cl_2TSV_5 or
aT primary tumors at all five *in vivo* passage
vels by cell fusion. The fusion capacity of
40-transformed cells increased after *in vivo*
ssage in hamsters. Fluorescent antibody staining
tumor cells detected SV40 capsid antigen as well
T antigen; the latter was detected in all cultures
hamster tumor cells.

25 HUMAN LEUKAEMIC CELLS CONTAIN TRANSCRIPTASE ASSOCIATED WITH A HIGH MOLECULAR WEIGHT
RUS-RELATED RNA.

(E.) Baxt, W. (Coll. Phys. Surg.,
lumbia U., New York, N.Y.), R. Hehlmann and S.
iegelman. *Nature New Biol* 240:72-75, 1972.

clei from peripheral white blood cells of leukemia
tients were centrifuged and the supernatants were
centrifuged to isolate membranes and viral par-
cles (if present). This material was used in a
verse transcriptase reaction. The products of
e reaction often formed 70S complexes involving
A from leukemic white blood cell material and a
S RNA molecule. These complexes were formed
white blood cell material from 95% of 23 leu-
mia patients sampled. DNA from this 70S-DNA
mplex did not anneal to 70S RNA from murine
mmary tumor virus or avian myeloblastosis virus.
e DNA did hybridize with Rauscher leukemia virus
A; 75% of leukemic DNA complexed to RLV RNA.

0:6 REVERSE TRANSCRIPTASE ACTIVITY OF HUMAN ACUTE LEUKAEMIC CELLS: PURIFICATION OF
ENZYME, RESPONSE TO AMV 70S RNA, AND CHARACTERI-
ION OF THE DNA PRODUCT.

(E.) Sarngadharan, M. G.
onetics Res. Labs., Bethesda, Md.), P. S. Sarin,
M. S. Reitz and R. C. Gallo. *Nature New Biol* 240(98): 67-72, 1972.

Human acute leukemic lymphoblasts were collected, disrupted and centrifuged to obtain a cytoplasmic supernatant which was itself centrifuged (60,000g) and disrupted by sonic oscillation. This high-speed pellet fraction of leukemic cells contained a reverse transcriptase DNA polymerase which catalyzed endogenous DNA synthesis. The DNA polymerase in the leukemic cells was completely RNAase-sensitive. The DNA polymerase could transcribe heteropolymeric portions of 70S avian myeloblastosis virus RNA; this reaction, however, was entirely abolished when the RNA was incubated with RNAase. The DNA polymerase had greater affinity for dT_{12-18}.poly rA than for dT_{12-18}.poly dA. Altogether, the DNA polymerase from the leukemic cells resembled that seen in RNA tumor viruses and appeared to be distinct from other human cellular DNA polymerases.

0827 BIOCHEMICAL CHARACTERISTICS OF NORMAL AND VIRALLY TRANSFORMED MOUSE CELL LINES.

(E.) Yoshikawa-Fukada, M. (Inst. Virus Res., Kyoto U., Japan) and T. Nojima. *J Cell Physiol* 80(3): 421-430, 1972.

Normal and SV40-transformed 3T3 mouse cells were grown in media having high K+ concentrations. In 100 mM K+, 3T3 cells did not multiply while SV40-3T3 cells did. Consistent with this finding, it was discovered that membranous fractions of 3T3 cells had 1.5-1.7 times higher specific activity of Na-K dependent ATPase than SV40-3T3 cells. Incubation with trypsinized concanavalin A caused SV40-3T3 to show contact inhibition of growth accompanied by higher Na-K dependent ATPase and adenyl cyclase activities than were seen in untreated cells. ATP contents in 3T3 and SV40-3T3 cells were similar. However, the content of cyclic 3',5'-AMP in normal 3T3 cells was almost double that in SV40-3T3 cells.

0828 REMARKS ON VIRUS-LIKE PARTICLES IN HUMAN BREAST CANCER.

(E.) Calafat, J. (Netherlands Cancer Inst., Amsterdam) and P. C. Hageman. *Nature* 242(5395):260-262, 1973.

Milk from Dutch women was examined by electron microscopy for the presence of virus-like particles similar to the B-type particles observed by Moore in milk of American and Paris women. Forty-two of the Dutch women had no cancer history and one had a positive family history of breast cancer. Electron microscopic observation of negatively stained preparations of each milk sample revealed the presence of membranous structures resembling those described by Moore and also resembling C-type particles. However, a correlated study of the same material viewed in thin sections failed to reveal the presence of either B-type or C-type virus-like particles. The fact that the particles observed in negatively stained material resembled those found by others in normal bovine and human milk made it likely that they represented cytoplasmic debris or secreted mammary gland products. In addition, B particles from mouse milk were not morpholigically altered following mixture with human milk as previously reported. It was thus concluded that one must exercise caution in associating the presence of unidentified virus-like particles in human milk with breast cancer.

0829 A TYPE-C VIRUS IN HUMAN RHABDOMYOSARCOMA CELLS AFTER INOCULATION INTO NIH SWISS MICE TREATED WITH ANTITHYMOCYTE SERUM. *(E.)* Todaro, G. J. (Natl. Cancer Inst., Bethesda, Md.), P. Arnstein, W. P. Parks, E. H. Lennette and R. J. Huebner. *Proc Natl Acad Sci USA* 70(3):859-862, 1973.

A C-type RNA virus was isolated from one line of human rhabdomyosarcoma (RD) cells following s.c. serial transplantation in antithymocyte serum-treated NIH Swiss mice, a strain from which C-type viruses have never been isolated. The RNA-dependent DNA polymerase activity from the newly isolated C-type virus was neutralized by specific antiserum against mouse, but not cat, primate or RD-114, viral polymerase. The cell from which the virus was isolated contained only mouse gs antigen as determined by complement fixation and radioimmunoassay. The virus replicated readily in human, nonhuman primate, and feline cells, less readily in rat cells, and not at all in mouse cells, a host range very similar to that of the RD-114 c-type virus. The newly isolated C-type virus showed no evidence of transforming ability in any cell system tested and was therefore assumed to be a helper (i.e., "leukemia") virus rather than a transforming (i.e., "sarcoma") virus. Virus expression was not due to antithymocyte serum since various other human tumor cells similarily transplanted remained free of overt C-type virus expression. The new isolate is presumed to be either a previously undescribed, endogenous C-type virus from NIH Swiss mice or a recombinant with both mouse and human C-type genetic information.

0830 SUGAR TRANSPORT IN NORMAL AND ROUS SARCOMA VIRUS-TRANSFORMED CHICK-EMBRYO FIBROBLASTS. *(E.)* Venuta, S. (Dept. Molecular Biol., U. California, Berkeley) and H. Rubin. *Proc Natl Acad Sci USA* 70(3):653-657, 1973.

The transport of isotopically labeled 3-*O*-methylglucose (3-OMeG), and nonmetabolizable glucose analog, was studied in normal and Rous sarcoma virus (RSV) transformed chick embryo fibroblasts. Both 3-OMeG and glucose are transported via the same carrier system. Competition experiments indicated that the carrier has a greater affinity for glucose than for 3-OMeG. Both the rate influx and of efflux of 3-OMeG was about three times faster in RSV-transformed cells than in normal chick fibroblasts. Experiments in which cells were grown for long periods of time in medium containing 3-MeG indicated that, at equilibrium, the intracellular 3-OMeG concentration in both RSV-transformed and normal cells was the same as that in the medium, It was thus concluded that 3-OMeG and glucose are transported in both RSV-transformed and normal chick fibroblasts by a facilitated diffusion process. Kinetic analysis showed that the three-fold faster incorporation rate for RSV-transformed cells could be accounted for by an increase in the Vmax. Small differences in the K_m were not reproducible.

0831 ISOLATION OF DNA AND DNA POLYMERASE FROM MC_{29} TUMOR VIRUS. *(E.)* Weber, G. H (Dept. Agricultural Chem., Oregon State U., Corvall A. O'C. Deeney and G. S. Beaudreau. *Biochim Biophys Acta* 299(1):8-16, 1973.

DNA polymerase was separated from the nucleic acids of purified avian myeloblastosis virus (AMV) and MC virus, another leukosis virus, by centrifuging Nonidet NP40-treated virus preparations on CsCl bouyant density gradients. Maximum polymerase activity of both viruses, which banded at a density 1.25-1.27 g/cm, required the addition of nucleic acid primer recovered from the viral nucleic acid pellet. The MC_{29} polymerase had a slightly greater sedimentation rate (7.7S) than did AMV polymerase (7.4S) in sucrose density gradients. Ribonuclease treatment of the MC_{29} polymerase had no effect on its sedimentation rate. MC_{29} viral primer nucleic acid recovered from the pellet fraction of the polymerase purification procedure banded in CsCl gradients at a density of 1.73g/cm^3 which shifted only slightly to 1.70 g/cm^3 if the nucleic acid was first treated with RNase. The conclusion that this primer was DNA was further supported by its susceptibility to DNase. The shift in density following RNase treatment, however, suggested that fragments of RNA were attached to the DNA of MC_{29} virus.

0832 SURFACE ALTERATIONS OF CELLS CARRYING RNA TUMOUR VIRUS GENETIC INFORMATION. *(E.* Salzberg, S. (St. Louis U. Sch. Med., Mo.) and M. Green. *Nature New Biol* 240:116-118, 1972.

The agglutinability of RNA tumor virus-transformed cells was studied. Transformed rat, mouse and cat cells, all of which replicated the sarcoma-leukemia virus complex of murine (MSV) and feline (FeSV) origin, were strongly agglutinated by concanavalin A (Con A), 80-90% of cells forming aggregates. Mouse and human cells replicating murine or feline viral components alone were not agglutinated by Con A. Normal cells developed the capacity to agglutinate when infected with MSV at a rate which paralleled virus replication. Non-viru producing transformed hamster and mouse cells which synthesized virus-specific RNA were either not agglutinated by Con A or were agglutinated to a lesser degree than were virus-producing transformed cells. These results suggest that surface changes associated with cell agglutination are a function

the expression of information present in the
nome of sarcoma but not of leukemia viruses.

833 STUDIES OF THE ROUS SARCOMA VIRUS RNA: CHARACTERIZATION OF THE 5' TERMINUS. *(E.)*

ilber, R. (Albert Einstein Coll. Med., Bronx, N.Y.),
. G. Malathi, L. H. Schulman and J. Hurwitz.
iochem Biophys Res Commun 50(2):467-472, 1973.

he 5'-terminal nucleoside of Prague strain Rous
arcoma virus (RSV) 30-40S RNA was identified.
iral RNA was treated with polynucleotide kinase
nd (γ-^{32}P) ATP, and the 5'-(^{32}P) RNA was degraded
ith alkali. This degradation yielded pAp while
egradation with venom phosphodiesterase yielded
abeled 5'-AMP. The results indicated that the
'-terminus of this viral RNA is almost exclusively
denosine.

834 RIBONUCLEASE H ACTIVITY PRESENT IN PURIFIED DNA POLYMERASE FROM AVIAN
YELOBLASTOSIS VIRUS. *(E.)*

Watson, K. F. (Inst.
trol, Berlin, W. Germany), K. Molling and H. Bauer.
ochem Biophys Res Commun 51(1):232-240, 1973.

A polymerase was purified from avian myeloblastosis
irus (AMV); ribonuclease H activity during purifi-
ation of DNA polymerase was monitored by DEAE-
llulose and Sephadex chromatography. Both DNA
lymerase and ribonuclease H co-chromatographed.
rthermore, ribonuclease H activity was found in
e purified two-polypeptide complex of DNA poly-
rase. These findings suggested that ribonuclease
activity resided in the DNA polymerase complex.
n requirements for optimal ribonuclease H activity
re identical to requirements for the purified
V DNA polymerase. Additional evidence for the
esence of ribonuclease H activity in the DNA
lymerase complex was provided by the neutralization
ribonuclease H activity with monospecific anti-
rum against the purified DNA polymerase.

35 BURKITT'S TUMORS CONTAIN PARTICLES ENCAPSULATING RNA-INSTRUCTED DNA POLY-
RASE AND HIGH MOLECULAR WEIGHT VIRUS. *(E.)*

fe, D. (Coll. Phys. Surg., Columbia U., New
rk, N.Y.), J. T. Magrath, J. L. Ziegler and
Spiegelman. *Proc Natl Acad Sci USA* 70(3):
7-741, 1973.

nor cells from 15 patients with Burkitt's
mphoma were examined for the presence of RNA-
structed DNA polymerase activity. Glycerol
imentation velocity gradient centrifugation
polymerase assay products from partially
rified cell lysates revealed the presence
70S RNA-DNA complexes in 13 of the 15 patients.
or treatment of these complexes eliminated
peak. Non-Burkitt, non-neoplastic Epstein-
r virus-containing control lymphoblastoid
l lines, as well as peripheral leukocytes
om a patient with infectious mononucleosis,
contained no such polymerase activity. The ^{3}H-labeled DNA product of the RNA-instructed DNA polymerase assay annealed to Rauscher leukemia virus RNA, but not to avain myeloblastosis virus RNA. Equilibium centrifugation of a pellet fraction prepared from a Burkitt's tumor showed banding of the RNA-instructed DNA polymerase activity at a density of 1.16-1.19, the range characteristic of the oncogenic RNA viruses. The possible etiologic role of an RNA virus in the pathogenesis of Burkitt's lymphoma, which has been presumed to be caused by Epstein-Barr virus, remains to be elucidated.

0836 EFFECT OF TREATMENT OF C57BL/6 MICE WITH THE METHANOL EXTRACTION RESIDUE FRACTION OF BCG ON LEUKEMOGENESIS INDUCED BY THE RADIATION LEUKEMIA VIRUS. *(E.)*

Haran-Ghera, N. (Weizmann Inst., Rehovot, Israel) and D. W. Weiss. *J Natl Cancer Inst* 50:229-234, 1973.

The effect of repeated i.p. injections of a methanol extraction residue (MER) fraction of BCG on leukemia incidence of radiation leukemia virus (RLV)-inoculated male C57BL/6 inbred mice was studied. Treatment with MER prior to RLV inoculation slightly enhanced the incidence of leukemia when compared with that of control mice pretreated with saline solution. Injection of MER on the 30th and 60th day after RLV inoculation significantly lowered leukemia incidence. Experiments with a second RLV line which required whole body X-radiation for maximal leukemogenesis indicated that MER could protect against leukemia development when given two or three times, but not when injected only once. The MER-induced protection lasted for the life of many animals.

0837 MORPHOGENESIS OF C-TYPE AND A-TYPE PARTICLES IN CELLS INFECTED BY AN ARBOVIRUS. *(E.)*

Grimley, P. M. (Natl. Cancer Inst., Bethesda, Md.), I. K. Berezesky and J. G. Levin. *J Natl Cancer Inst* 50:275-279, 1973.

The morphogenesis of C-type and A-type virus particles was studied by electron microscopy in several established murine cell lines during simultaneous infection with Semliki Forest virus (SFV), a group A arbovirus. In spite of exponential replication of SFV, each of the cell lines continued to produce both C-type and A-type particles which were seen budding from the same cell membranes in close proximity to each other. In addition, both immature and mature C-type particles were observed to accumulate within membranous cytoplasmic cytopathic vacuoles characteristic of group A arbovirus-infected cells. Budding of intracisternal A-type particles was also found in the same sections with the arbovirus-specific membranous structures. This persistent formation of C-type and A-type virus particles in established murine cell lines simultaneously infected with SFV presumably reflected either a persistent expression of tumor virus genomes or the existence of a pool of virus precursors which could continue to assemble as particles for many hours, even though cellular metabolic conditions were altered.

0838 REPLICATION OF THE MOLONEY MURINE SARCOMA-LEUKEMIA VIRUS IN XC CELLS.

(E.) Trowbridge, S. T. (Baylor College Med., Houston, Texas), M. Benyesh-Melnick and N. Biswal. *J Virol* 11(1):146-149, 1973.

XC cells have been found to support the replication of the Moloney strain of murine sarcoma-leukemia virus (MSV-MLV) derived from virus-releasing transformed Wistar rat cells. XC monolayers infected with a high multiplicity of MSV-MLV showed cell fusion by 6 hr after infection. By 73 hr, fusion was complete and vacuolization was extensive. Infected cells began to produce progeny virus after an eclipse of 12 to 18 hr. The progeny virus particles contained RNA characteristic of the input MSV-MLV, were neutralized by anti-MLV rabbit serum, and contained MLV-specific antigens detectable by indirect immunofluorescence.

0839 ACTION OF 1-β-D-ARABINOFURANOSYLCYTOSINE ON MAMMALIAN TUMOR CELLS. 2. INHIBITION OF MAMMALIAN AND ONCOGENIC VIRAL POLYMERASES.

(E.) Müller, W. E. G. (Inst. Physiol. Chem., Guttenberg U., Mainz, W. Germany), Z.-I. Yamazaki, H. H. Sögtrop and R. K. Zahn. *Eur J Cancer* 8:421-428, 1972.

Inhibition of DNA polymerase in normal mouse liver cells, mouse lymphoma cells and Rauscher murine leukemia virus (RMLV) by 1-β-D-arabinofuranosylcytosinetriphosphate (ara-CTP) was tested. DNA-dependent DNA polymerases from the three sources were competitively inhibited by ara-CTP; inhibition was most nearly complete for the RMLV enzyme. RNA-dependent DNA polymerases were also competitively inhibited by ara-CTP. The RNA-dependent enzymes were 200 times more sensitive to ara-CTP than the DNA-dependent enzymes.

0840 VSV PSEUDOTYPE PARTICLES WITH THE COAT OF AVIAN MYELOBLASTOSIS VIRUS.

(E.) Zavada, J. (Slovak Acad. Sci., Bratislava, Czechoslovakia). *Nature* [*New Biol*] 240(99):122-124, 1972.

A temperature-sensitive mutant, *ts*-45, of vesicular stomatitis virus (VSV) was incubated at low temperatures with avian myeloblastosis virus (AMV) to produce a pseudotype virus VSV(AMV). The pseudotype virus produced with *ts*-45 was contaminated with 1% wild-type VSV revertants. When another thermolabile VSV mutant, *tl*-17, which had been produced by growing wild-type VSV with anti-VSV serum at 31 C, was used to produce pseudotypes with AMV, the pseudotypes were contaminated with less than 0.01% VSV material. The *tl*-17(AMV) pseudotype had neutralization, host-range and interference specificities corresponding to those of AMV.

0841 VIRUS-LIKE PARTICLES IN LEUKAEMIC MITOCHONDRIA.

(E.) Schumacher, H. R. (Harrisburg Hosp., Pa.), I. E. Szekely and S. A. Park. *Lancet* 1(7803):601-602, 1973.

Virus-like particles were observed in the mitochondria in three of six patients with acute myeloblastic leukemia but not in 28 normal bone marrows. These mitochondria had damaged cristae with clear areas which sometimes contained the virus-like particles. Monoblasts and lymphoblasts in clear areas of rare mitochondria contained similar particles. Subsequent electron microscope analysis revealed virus-like particles in damaged mitochondria of a rat-embryo fibroblast culture infected with Moloney sarcoma virus and Moloney leukemic virus. The particles contained a dense nucleoid with an outer membrane. They measured 919-1040 A and in some areas exhibited triple membranes. One particle was observed budding into the mitochondrial membrane and another budding from the cristae.

0842 LEVELS OF MEMBRANE MARKER ENZYME ACTIVITY IN NORMAL AND RNA AND DNA VIRUS-TRANSFORMED FIBROBLASTS.

(E.) Bosmann, H. B. (U. Rochester Sch. Med. Dent., N.Y.). *Biochim Biophys Acta* 291(2):582-586, 1973.

Activities of three enzyme systems were observed in four mouse cell lines: 3T3, a line of normal mouse embryo fibroblasts; MSV-3T3, 3T3 cells transformed by murine sarcoma virus; RSV-3T3, 3T3 cells transformed by Rous sarcoma virus; and PY-3T3, 3T3 cells transformed by polyoma virus. The levels of the following enzymes were determined in these cells: 5'-nucleotidase, a plasma membrane marker enzyme; UDPase, a smooth endoplasmic reticulum marker enzyme; and esterase, a rough endoplasmic reticulum marker enzyme. 5'-Nucleotidase and esterase activities were significantly elevated in virus-transformed cells compared with untransformed 3T3 cells. Even in plasma membranes prepared with high concentrations of Zn^{2+}, 5'-nucleotidase showed high levels. UDPase was slightly elevated in the virus-transformed cells.

0843 QUANTITATION OF RNA TUMOR VIRUSES AND VIRU LIKE PARTICLES IN HUMAN MILK BY HYBRIDIZATION TO POLYADENYLIC ACID SEQUENCES.

(E.) Schlom, J. (Coll. Physicians Surgeons, Columbia U., N.Y.), D. Colcher, S. Spiegelman, S. Gillespie and D. Gillespie. *Science* 179(4074):696-698, 1973.

RNA tumor viruses and virus-like particles from human milk were quantitated by hybridization of e polyadenylic acid [poly(A)] regions in their -70S RNA to radioactive polyribouridylic acid known specific activity. The poly(A) stretches the 60-70S RNA from human milk particles were e same length, approximately 200 nucleotides ng, as those found in the mouse mammary tumor rus and in other RNA tumor viruses. Five (14%) 37 samples of human milk were positive for ly(A) regions, ranging in particle counts om 1.0 X 10^6 to 4.0 X 10^7 particles/ml. That % of the milks tested were negative indicated at the 14% positives were not due to normal A species of human lactating mammary tissue.

44 EFFECT OF ETHIDIUM BROMIDE ON CYTOCHROME SYNTHESIS OF SV-40 TRANSFORMED CELLS. .) Sato, N. (U. Pennsylvania Sch. Med., iladelphia), B. Chance, K. Kato and W. Klietmann. *3S Letters* 29(3):222-226, 1973.

-1 cells, a line of SV40-transformed hamster cells, re treated with ethidium bromide (EB, 2 µg/ml) d cytochrome synthesis was observed by spectro- otometry. EB decreased absorbance of cytochromes $_3$, b and c_1, but did not affect that of cytochrome until 11 days after EB. For four days after EB, e contents in cells of cytochromes aa_3, $b_{(K)}$ d c_1 declined at equal rates. After six days, tochrome aa_3 decreased significantly more than tochrome $b_{(K)}$ and c_1, amounting to only 10% of e control level at eight days. Cytochrome c ntent however, remained unaltered. Cytochrome $_{57}$ increased with EB as cytochrome aa_3 decreased. e effects of EB were reversible; cytochrome ntents recovered by four to six days after with- awal of EB.

45 DEFECTIVITY OF RAUSCHER MURINE ERYTHROBLASTOSIS VIRUS. *(E.)* Bentvelzen, P. adiobiol. Inst., TNO, Rijswijk, Netherlands), M. Aarssen and J. Brinkhof. *Nature* [*New Biol*] 9(91):122-123, 1972.

uscher leukemia virus (RLV), which produced ythroblastosis in BALB/c mice, was found to stand a linear dose-response relationship with the rmation of splenic erythroblast foci in inoculated LB/c mice. Though most RLV showed a single-hit ttern of dose and formation of foci, one RLV rain showed a two-hit response. When this wo-hit" RLV was mixed with Tennant leukemia rus before inoculation into mice, one-hit kinetics re restored. Preinfection of mice with nnant virus before inoculation with two-hit RLV so changed the response to a single-hit pattern. ce given urethane before two-hit RLV gave a ngle-hit response after a week of urethane eatment. It was concluded that the two-hit V contained a defective focus-forming principle d needed coinfection with another leukemia virus.

6 HIGH INTERFERON PRODUCING LINE OF TRANSFORMED MURINE CELLS. *(E.)* Oie, H. K. (Natl. Inst. Allergy Infectious Dis., Bethesda, Md.), A. F. Gazdar, C. E. Buckler and S. Baron. *J Gen Virol* 17:107-109, 1972.

Production of interferon in C-243-3 cells, a cloned line of Moloney sarcoma virus (MSV)-transformed murine cells, was induced by Newcastle disease virus (NDV), Chikungunya virus (CV), or polyinosinic·polycytidylic acid (poly I·poly C) plus diethylaminoethyl-dextran (DEASE-D). NDV was consistently better than the other two stimulators in inducing interferon production. The lowest titer obtained with NDV was $10^{4.2}$ units/ml, while the highest was $10^{5.6}$ units/ml. The interferon produced by C-243-3 cells was stable at pH 2 for least five days, sensitive to trypsin, not removed by frequent washings of treated cells, had antivirus activity against vesicular stomatitis virus, GD-7 virus and Sindbis virus, and was active in mouse L cells and secondary mouse embryo fibroblasts but not in chicken embryo cells. Pretreatment of C-243-3 cells with interferon did not alter interferon yields of NDV or poly I·poly C plus DEAE-D.

0847 SIZE DISTRIBUTION OF RNA SPECIES HYBRIDIZABLE TO CELLULAR OR VIRAL DNA IN CELLS TRANSFORMED BY ADENOVIRUS TYPE-2. *(E.)* Shimada, K. (Aichi Cancer Ctr. Res. Inst., Nagoya, Japan), K. Sekikawa, K. Fujinaga and Y. Ito. *Gann* 63:801-803, 1972.

RNA was purified from rat embryo cells transformed by adenovirus type 2. The RNA samples were fractionated by polyacrylamide gel electrophoresis, and the fractions used to detect RNA hybridizable with viral or cellular DNA. A single sharp electrophorectic peak of 19-20S was seen for viral RNA. However, RNA's hybridizable with cellular DNA showed an entirely different distribution. Previous work has indicated that virus-specific RNA in cytoplasm is heterogeneous in molecular wt (10-30S) with the same cell line. The present results show the lack of an RNA component over 25S.

0848 CHEMICAL AND PHYSICAL RELATIONSHIPS OF ONCOGENIC AND NON-ONCOGENIC SIMIAN ADENOVIRUS DNA'S. *(E.)* Burnett, J. P. (Lilly Res. Labs., Indianapolis, Ind.), N. Mayne, L. K. Butler and J. A. Harrington. *J Gen Virol* 17(3):245-253, 1972.

Simian adenoviruses of a number of different species were purified and their DNA's were extracted and characterized by various means. In base composition, five oncogenic viruses had more guanine + cytosine (G + C) in their DNA than nononcogenic viruses; however, other oncogenic viruses had the same G + C contents as non-oncogenic viruses. The molecular wt of oncogenic as well as nononcogenic viruses was ramdomly distributed between 17 and 23 million. DNA hybridization was used to estimate the genetic relatedness of the simian adenoviruses. Three groups of viruses were distinguished; in each group, member viruses were more closely related to other members than they were to nonmembers. Two such homologous groups contained oncogenic viruses, one contained nononcogenic viruses.

0849 ANTIGEN BINDING ROSETTE FORMING CELLS IN A FRIEND VIRUS-INDUCED LEUKEMIA.

(E.) Cerny, J. (Harvard Sch. Pub. Hlth., Boston, Mass.) and J. Halasa. *Experientia* 29(1):101-102, 1973.

BALB/c mice were inoculated with Friend leukemia virus (FLV); one or five days later, some mice were immunized with sheep red blood cells (SRBC). Four days after SRBC, the anti-SRBC plaque-forming cells (PFC) and rosette-forming cells (RFC) in mouse spleens were counted. Normal numbers of RFC were found in FLV-infected spleens from mice not given SRBC immunizations. However, virus infection five days before SRBC inhibited the immune response to SRBC; PFC and RFC were decreased in number in mice given FLV before SRBC.

0850 SIMIAN TUMOUR VIRUS PROLIFERATION IN INOCULATED *MACACA MULATTA*.

(E.) Fine, D. L. (Bionetics Res. Labs., Kensington, Md.), E. W. Kingsbury, M. G. Valerio, M. T. Kubicek, J. C. Landon and H. C. Chopra. *Nature [New Biol]* 238(84):119-192, 1972.

Newborn female *Macaca mulatta* monkeys were injected i.p. or s.c. with M-PMV, an oncorna-type virus detected in a spontaneous rhesus monkey mammary tumor. Lymph nodes were biopsied at intervals after inoculation and biopsies were examined under the electron microscope. Lymphadenopathy followed M-PMV injection. M-PMV particles were seen in lymph nodes from seven of nine inoculated monkeys. They appeared as intracytoplasmic A particles. Infectious M-PMV was also found on four of six nodes assayed by cocultivation in monkey cells.

0851 BLOCKS IN NUCLEOTIDE PHOSPHODIESTERASE AND ALKALINE PHOSPHATASE ACTIVITY IN TRANSFORMED MAMMALIAN CELLS.

(E.) Sela, B.-A. (Weizmann Inst., Rehovot, Israel) and L. Sachs. *FEBS Letters* 30(1):100-104, 1973.

The ability of transformed cells to hydrolyze sugar-nucleotides was studied. The transformed cells were hamster cells transformed *in vitro* by polyoma virus or dimethylnitrosamine, and hamster tumor cells induced by Rous sarcoma virus or SV40. All transformed cells showed blocked hydrolysis of GDP-mannose, GDP-fucose and UDP-glucose; while normal hamster cells could convert 88-95% of test material in the enzyme studies, transformed cells could convert 3% at most. The block in overall hydrolysis was not due to increased levels of enzyme inhibitors in transformed cells, since hydrolysis was unimpaired when normal and transformed cells were mixed. The RSV- and SV40-transformed lines were presumably blocked in the nucleotide phosphodiesterase activity, as cell extracts hydrolyzed sugar-1-phosphate into free sugar, 30 and 50%, resp., of the hydrolysis found with normal cell extracts. The phosphohydrolase that was blocked in transformed cells was an alkaline phosphatase.

0852 INHIBITION OF DNA POLYMERASES FROM RNA TUMOR VIRUSES BY NOVEL TEMPLATE ANALOGUES: PARTIALLY THIOLATED POLYCYTIDYLIC ACID.

(E.) Chandra, P. (Inst. Therap. Biochem., U. Frankfort, W. Germany) and T. J. Bardos. *Res Commun Chem Path Pharmacol* 4(3):615-622, 1972.

Polycytidylic acid was partially "thiolated" to convert 9.5% of the cytidylate units to 5-mercaptocytidylate. The resulting product, mercapto-(5-SH, 9.5%)-polycytidylic acid (MPC), was incubated with purified Friend leukemia virus (FLV) or Moloney sarcoma virus (MSV-M) material; poly (da-dT) or poly $rA.(dT)_{14}$ was included in the reaction mixtures. The incorporation of ^{3}H-thymidine into DNA was observed as a measure of DNA polymerase. MPC inhibited DNA polymerase in FLV and in MSV-M; inhibition was similar in the two viruses when poly (dA-dT) was used as template. With template poly $rA.(dT)_{14}$, MPC inhibited MSV-M DNA polymerase much more than it inhibited the FLV polymerase. Time studies suggested that MPC blocked template binding to the polymerase.

0853 ASPECTS OF THE ENCAPSIDATION OF SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID.

(E.) Girard, M. (Inst. Sci. Res. Cancer, Villejuif, France), D. Stehelin, S. Manteuil and J. Pages. *J Virol* 11(1):107-115, 1973.

The time course of virus formation was determined in growing cultures of SV40-infected subclones of DV1-cells. When CV-1 cells, labeled with ^{3}H-thymidine at various times after infection, were fractionated and analyzed by sucrose gradient centrifugation, most of the progeny SV40 particles were recovered in the cytoplasmic extract and not in the nuclei. This result was independent of both the fractionation method and of the time after infection at which the extracts were prepared. These findings, along with observations made by the electron microscope on virus-infected cells, suggested that virions located in the nucleus leak into the cytoplasm during the fractionation procedure, possibly due to increased fragility of the nuclear membrane. Results from short-term and pulse-chase labeling experiments indicated that newly synthesized viral DNA is rapidly encapsulated and that progeny viral DNA is withdrawn at random from the pool of replicated DNA molecules. Virions were found to accumulate in a nonlinear fashion, at the time when the number of viral DNA molecules increased linearly with time after infection, suggesting that the size of the intracellular capsid protein pool increases constantly during the late phase of virus replication.

0854 RNA RELATED TO THAT OF A MURINE LEUKEMIA VIRUS IN BURKITT'S TUMORS AND NASOPHARYNGEAL CARCINOMAS. *(E.)* Kufe, D. (Coll. Phys. Surg., Columbia U., New York, N.Y.), R. Hehlmann and S. Spiegelman. *Proc Natl Acad Sci USA* 70(1): 5-9, 1973.

Annealing reactions between ^{3}H-DNA of Rauscher leukemia virus and RNA from polysomes of Burkitt lymphoma and nasopharyngeal carcinoma were analyzed in Cs_2SO_4 gradients. Results indicated that Burkitt lymphoma and nasopharyngeal carcinoma contained RNA homologous with that of Rauscher virus. Of 21 Burkitt tumors examined, 76% yielded a positive response in annealing reactions with Rauscher virus DNA; of 20 nasopharyngeal carcinomas, 50% reacted positively. In contrast, none of the polysomal RNAs prepared from 52 normal adult and fetal tissues showed a clear positive response to Rauscher DNA.

0855 EFFECT OF ENVIRONMENTAL pH ON RESCUE OF SIMIAN VIRUS 40. *(E.)* Calothy, G. (Wistar Inst., Philadelphia, Pa.), C. M. Croce, V. Defendi, H. Koprowski and H. Eagle. *Proc Natl Acad Sci USA* 70(2):366-368, 1973.

SV40-transformed mouse cells (line mKs Bu 100) or hamster cells from an SV40-induced tumor (line BTH) were fused by Sendai virus mediation with SV40-permissive African green monkey cells (line CV-1), and SV40 rescue from fused cells was observed at pH values from 6.4-8.8. Virus rescue from both mouse and hamster cells was strikingly pH-dependent. SV40 yield was minimal (titers = 10^2/ml) at pH 6.4 and pH 8.8. Between pH 6.8-8.4, however, the yield increased progressively to reach a maximum titer of $10^{7.8}$ at pH 8.4. Environmental pH did not affect SV40 replication in CV-1 cells over the range pH 7.2-8.4. Nor did pH influence the number of heterokaryocytes formed in cell fusion, heterokaryocyte formation remaining constant between pH 7.6-8.4.

0856 SURFACE CHARACTERISTICS OF SV40 TRANSFORMED TRANSPLANTABLE AND NON-TRANSPLANTABLE MOUSE CELLS. *(E.)* Kurimura, T. (Osaka Prefectural Inst. Public Hlth., Japan) and A. Hirano. *Biken J* 15(3): 193-197, 1972.

The surface characteristics of a nontransplantable SV40-transformed mouse kidney cell line (mKS-A) and a transplantable line derived from it (mKS-A TU-7) were compared. Transplantable mKS-A TU-7 cells had the greater capacity to absorb A/Okuda/57 (H2N2) influenza virus and were more agglutinable by concanavalin A. From the capacity to absorb influenza virus, the surface of mKS-A TU-7 cells may have more sialic acid residues than that of mKS-A, or the distribution of sialic acid residues on the two strains may be very different. If so, it is possible that the acid residues reduce cell immunogenicity or cover transplantation antigens on the cell surface. Differences in the agglutinabilities of mKS-A and mKS-A TU-7 may in part be due to differences in the grade of expression of the SV40 virus gene function(s).

0857 VIRUS-INDUCED GENITOURINARY MALIGNANCY. MACROMOLECULAR IDENTITY. *(E.)* Paulson, D. F. (Natl. Cancer Inst., Bethesda, Md.), E. E. Fraley, J. B. de Kernion and A. S. Rabson. *Invest Urol* 10(4):298-306, 1973.

Malignant tumors of the hamster kidney and prostate have been produced with the deoxyribonucleic acid (DNA) oncogenic viruses SV40 and E46, and by the continuous administration of diethylstilbestrol. In addition, two human renal malignancies have been maintained in tissue culture. The agents responsible for the initiation of these two human renal malignancies have, as yet, not been identified. Infection and transformation of hamster genitourinary tissue with the DNA oncogenic viruses SV40 and E46 induce characteristic changes in average size distribution of the host cell polyribosomes. However, this characteristic polyribosomal distribution is not found in the hamster kidney tumors induced by continuous estrogen administration. Within hamster genitourinary tissues, the presence of a cytoplasmic polyribosome distribution characterized by a lack of heavy rapidly sedimenting polyribosomes and an oligosomal segment dominated by monomer and dimer peaks appears directly correlated with the presence of an oncogenic DNA viral genome in the transformed cells. One of two human renal malignancies presents a polyribosome distribution similar to that seen in the tumors produced by the oncogenic DNA viruses SV40 and E46. The relationship between the presence of this peculiar polyribosome profile in a human genitourinary tumor and the precipitating influence of an oncogenic virus in the generation of that specific human tumor has yet to be established.

0858 MALIGNANT LYMPHOMA INDUCED BY PARTIALLY PURIFIED *HERPESVIRUS SAIMIRI* AND RECOVERY OF INFECTIOUS VIRUS FROM TUMOROUS LYMPH NODES. *(E.)* Laufs, R. (U. Hyg.-Inst., Gottingen, Germany) and B. Fleckenstein. *Med Microbiol Immunol (Berl)* 158(2):135-146, 1972.

Supernatant of OMK cells infected with *Herpesvirus saimiri* (HVS) and partially purified HVS were shown to induce malignant lymphoma in New World monkeys *(Saguinus oedipus)*. Radioactively labeled HSV was partially purified by velocity sedimentation in sucrose and buoyant density centrifugation in potassium tartrate and inoculated in a monkey. Infectious HVS was isolated from a tumorous lymph node of this animal 2 hr after its cultivation *in vitro*. In contrast to the lymphocytes from fresh peripheral blood cocultivation of the cells obtained from the lymph node was not necessary for the isolation of the virus. The development of comparatively high amounts of neutralizing antibodies against HVS in the sera of the fatally diseased monkeys in an indication for a replication of HVS in the tumor bearing animals.

0859 SPECIFIC NEUTRALIZING ANTIBODIES IN EPSTEIN-BARR VIRUS ASSOCIATED DISEASES.

(E.) Rocchi, G. (Children's Hosp., Philadelphia, Pa.), J. Hewetson and W. Henle. *Int J Cancer* 11(3):637-647, 1973.

The results of Epstein-Barr virus (EBV) neutralization tests on 650 sera from patients with EBV-associated diseases and controls were compared with the titers of antibodies to EB viral capsid antigens (anti-VCA) and to EBV-induced early antigens (anti-EA). All anti-VCA negative sera failed to neutralize EBV. Among 546 anti-VCA positive sera, only eight yielded negative results in neutralization tests. Four of these came from patients in the early acute phase of infectious mononucleosis who subsequently developed neutralizing antibodies. The ratios between neutralizing and anti-VCA titers varied over a wide range indicating that different antibodies are involved in the two tests. In line with the regular presence of mostly elevated anti-VCA titers, the geometric mean neutralizing titers in Burkitt's lymphoma and nasopharyngeal carcinoma were also higher (5- to 7-fold) than in appropriate controls. In Hodgkin's disease, an over-representation of high neutralizing as well as anti-VCA titers was noted, although some patients had neither of these antibodies. All patients with infectious mononucleosis developed neutralizing antibodies with a geometric mean ultimately matching that of controls whereas in the early acute phase it was lower than in the controls. In contrast, the geometric mean anti-VCA titer in the early acute phase was higher than in controls and then declined in convalescence toward the control value. There was no evident relationship between neutralizing and anti-EA activities whether directed against the D or the R component of the EA complex.

0860 FACTORS INFLUENCING THE CELL TYPE OF BRAIN TUMORS INDUCED IN DOGS BY SCHMIDT-RUPPIN ROUS SARCOMA VIRUS.

(E.) Bigner, D. D. (Dept. Pathol., Duke U., Durham, N.C.), J. P. Kvedar, B. A. Thomas, C. Shaffer, N. A. Vick, W. K. Engel and E. D. Day. *J Neuropathol Exp Neurol* 31(4):583-595, 1972.

Bacteria was eliminated from chicken sarcoma-extracted Schmidt-Ruppin Rous sarcoma virus (SR-RSV) and the virus was aseptically concentrated up to 80-fold with no loss of viral infectivity by differential centrifugation, sonication, and filtration. Suspension of the highly concentrated SR-RSV in 0.05 M sodium citrate buffer, pH 6.7, and storage at -70 C maintained brain tumor-inducing potential of the virus suspension. After intracerebral inoculation of 0.1 ml of the highly concentrated 80 gm equivalents/ml SR-RSV into neonatal beagles, a 96% survival incidence and 100% brain tumor incidenc (both sarcomas and gliomas) within 6 wk was observed In dose-response studies a graded host response with decreasing number of brain tumors per animal as well as decreasing numbers of inoculated animals with tumors was observed with decreasing SR-RSV doses. With a smaller (0.01 ml) inoculum volume of the most highly concentrated SR-RSV, brain inoculation site was the determinant of type of tumor induced; 7/10 dogs inoculated in the subependymal cell plate developed only gliomas, and all dogs in which tumors were induced after inoculation in the parietal subdural space (5/5) or the cerebellar vermis (4/5) developed sarcomas.

0861 EFFECTS OF RAUSCHER LEUKAEMIA VIRUS ON THE IMMUNE SYSTEM IN SUSCEPTIBLE AND RESISTANT MOUSE STRAINS.

(E.) Toth, F. D. (U. Med. Sch., Debrecen, Hungary), T. Karsai and L. Vaczi. *Acta Microbiol Acad Sci Hung* 18:271-278, 1971.

BALB/c, DBA/1 and C57BL/10 Sn mice inoculated i.p. with Rauscher leukemia virus (RLV) showed increased α_1, α_2 and β_2 globulin levels. The increase was in direct relation to the susceptibility of the mouse strain. Gamma globulin level decreased in the BALB/c and DBA/1 strains parallel with progress of leukemia, while in C57BL/10 Sn mice it increased and remained at an elevated level. These changes were attributed partly to the effects of RLV on the immune system and partly to production of new proteins by tumor cells. Multiplication of virus in lymph nodes of BALB/c and DBA/1 mice was parallel by a decrease in the production of immuno-specific globulins, presumably IgG-type globulins.

0862 DNA SYNTHESIS IN ISOLATED NUCLEI AND ISOLATED MITOCHONDRIA OF HERPES SIMPLEX VIRUS INFECTED HeLa CELLS.

(E.) Radsak, K. (Hyg. Inst., U. Marburg, W. Germany). *FEBS Letters* 31(1):42-46, 1973.

DNA synthesis was observed in (^{3}H)thymidine triphosphate-labeled nuclei and (^{3}H)thymidine-labeled mitochondria from Herpes simplex virus (HSV)-infected HeLa cells isolated 2-4 hr post-infection. Infected nuclei exhibited a clearly lower incorporating activity than those from mock-infected cells. In contrast, infected mitochondria showed a DNA synthesizing activity comparable with that of mitochondria from mock-infected cells. The radioactivity profile resulting when DNA from mock-infected nuclei was analyzed by density centrifugation on cesium chloride gradients exhibited a single band at a density of approximately 1.7 g/cm^3. Infected nuclei showed several bands, the lightest of which corresponded in density to cellular DNA of nuclear origin. The heavier band (1.710-1.720 g/cm^3) presumably represented DNA relat to viral DNA. Specific radioactivity of cellular DN of nuclear origin was significantly lower in nuclei

om infected cells. Radioactivity profiles of
tochondria from infected and mock-infected cells
re similar. These results suggest that stimulation
mitochondrial DNA synthesis observed in intact
lls early after infection is an indirect consequence
viral activity.

63 STRAND SWITCH DURING *IN VIVO* POLYOMA TRANSCRIPTION. *(E.)* Mueller, N. (Inst.
g., Freiburg, W. Germany), J. Zemla and G. Brandner.
BS Letters 31(2):222-224, 1973.

rand orientation during transcription of polyoma
y) virus was investigated. Hybridization of *in*
tro (^{3}H)Py cRNA with increasing amounts of
lter-bound Py DNA resulted in up to 60% hybridization of the transcript. Hybridization of filter-
und Py DNA with increasing amounts of (^{3}H)Py
NA resulted in up to 20% hybridization of the DNA.
cubation of *in vitro* (^{3}H)Py cRNA with unlabeled
A from uninfected mouse cells increased symmetri-
lly transcribed RNA. Annealing of *in vitro* (^{3}H)
cRNA with unlabeled late *in vivo* RNA from Py-
fected mouse embryo cells increased RNAase
sistant radioactivity by an additional 30%. The
crease represented the portion of late *in vivo*
cRNA transcribed from the strand complementary
that transcribed *in vitro*. No further increase
RNAase resistant radioactivity occurred during
nealing of *in vitro* (^{3}H)Py cRNA with early *in*
vo Py cRNA, indicating that early *in vivo* and
vitro Py cRNA were transcribed from the same
A strand. These results indicate that trans-
iption of late Py genes, like that of SV40
nes, occurs after a strand switch.

64 MAMMARY TUMOR VIRUS (MTV) INFECTION OF CBA/BlN MICE INVOLVING PRODUCTION OF
TIBODIES TO MTV. *(E.)* Müller, M. (Carl Gustav
rus Med. Acad., Dresden, Germany) and S. Zotter.
Natl Cancer Inst 50(3):713-717, 1973.

th acetone-fixed, frozen-cut slices of syngeneically
afted CBA/Bln murine mammary tumors extremely rich
mammary tumor virus (MTV) A and B particles,
ontaneously occurring antibodies could be detected
43 of 46 MTV-infected mice by means of the
direct immunofluorescent technique. These anti-
dies were found in male and female MTV-infected
A/Bln and XVII/Bln mice but not in MTV-free
ce of both strains. The antibodies could be
sorbed totally by MTV-containing milks of the
o strains, whereas MTV-free milks of both strains
iled to do so. In correspondence to the specificity
precipitating antibodies detectable in a limited
mber of infected mice, which are directed against
e B particles or an antigen of the outer viral
at, the resulting fluorescence was restricted to
e intercellular spaces and glandular lumina of
e tumor tissue. The results show that no trace
any true immunologic tolerance to this particular
V antigen is established in CBA/Bln mice. Further-
re, this type of antibody response seems to be
e most sensitive indicator for an MTV infection.

0865 COMPARISON OF SOME OF THE BIOLOGICAL PROPERTIES OF FRIEND VIRUS AND ITS PSEUDOTYPES. *(E.)* Dawson, P. J. (U. Oregon Med. Sch., Portland) and A. H. Fieldsteel. *Int J Cancer* 11(2):484-493, 1973.

The properties of the Moloney and Graffi pseudotypes of Friend virus have been compared with the authors original strain of Friend virus. Qualitatively the pathologic effects of the three were indistinguishable. The spleens of mice inoculated with each of the three viruses appeared to have a similarly low potential for the initiation of transplantable tumors in BALB/c mice. Both pseudotypes were highly age dependent when first isolated, although the Moloney pseudotype lost this property after the sixth passage in newborn BALB/c mice. Compared with the original Friend virus, there were marked differences between the host ranges of both pseudotypes. None of these viruses induced Friend disease in C57BL/6 mice. However, preparations of both pseudotypes and of the lymphatic leukemia viruses used as helpers readily induced lymphatic leukemia in this strain, indicating that susceptibility to the helper virus is not the only factor governing host range.

0866 TEMPERATURE-DEPENDENT ALTERATIONS IN 2-DEOXYGLUCOSE UPTAKE IN RAT CELLS TRANSFORMED BY A COLD-SENSITIVE MURINE SARCOMA VIRUS MUTANT. *(E.)* May, J. T. (Baylor Coll. Med., Houston, Texas), K. D. Somers and S. Kit. *Int J Cancer* 11(2):377-384, 1973.

Cells transformed by Moloney sarcoma virus (MSV) take up 2-deoxyglucose at a faster rate at 39 C than uninfected or Moloney leukemia virus (MoLV)-infected normal rat kidney (NRK) cells. In a sarcoma-virus-transformed cell line, NRK(MSV-1b), whose transformed phenotype is expressed at 39 C (permissive) but not at 33 C (non-permissive), the stimulation of 2-deoxyglucose uptake at 39 C is temperature dependent; the increase is observed when cells are grown at 39 C but not at 33 C. When these cells were shifted from the non-permissive to the permissive temperature, the uptake increased from a rate near that of uninfected cells to a rate half that of NRK cells infected with Moloney sarcoma-leukemia complex (MSV-MoLV). The reverse change occurred when the cells were shifted from the permissive to the non-permissive temperature. Thus in a sarcoma virus-infected rat cell line where the maintenance of the transformed state is dependent on a cold-sensitive viral function, the uptake of 2-deoxyglucose is reduced in the cold-sensitive cells and correlated with the expression of the transformed phenotype.

0867 INCREASED ONOGENICITY AND SYNTHESIS OF HERPESVIRUS ANTIGENS IN HAMSTER CELLS EXPOSED TO HERPES SIMPLEX TYPE-2 VIRUS. *(E.)* Kutinova, L. (Inst. Sera Vaccines, Prague, Czechoslavakia), V. Voska and J. Broucek. *J Natl Cancer Inst* 50(3):759-766, 1973.

When cells of a continuous line derived from hamster embryo fibroblasts and oncogenic for hamsters were exposed to ultraviolet-irradiated herpes simplex virus type 2 (HSV-2), infectious virus and the development of persistently infected cultures resulted. The culture was freed of infectious HSV by addition of HSV antibody or by incubation of the cells at higher temperature. Either treatment permitted the isolation of cell lines (denoted TR cells) that carried HSV-associated antigens and possessed an increased oncogenic potential for hamsters. HSV-associated antigens in TR cells were detected by demonstration of HSV-neutralizing antibody in hamsters bearing tumors induced by TR cells. The neutralizing antibody was specific for type 2. All attempts to induce infectious HSV formation failed. The mechanisms restricting HSV infection in the persistently infected culture may have caused the selection of transformed cell clones thus paralleling the situation *in vivo*, where the host defense reactions limit virus spread and thus decrease the possibility of reinfection of cells already infected with defective virions.

0868 MEMBRANE COMPONENTS AND ENZYMES IN VIRALLY TRANSFORMED CELLS.

(E.) Brady, R. O. (Natl. Cancer Inst., Bethesda, Md.), P. H. Fishman and P. T. Mora. *Fed Proc* 32(1):102-108, 1973.

There is a reduction of higher ganglioside homologs and in the activity of the enzyme uridine diphosphate *N*-acetylgalactosamine:hematoside *N*-acetylgalactosaminyltransferase in mouse cell lines after transformation with SV40 or polyoma virus. These changes are reversibly coupled to the changes in growth properties in tissue culture. In the "flat revertant" derivatives of the virus-transformed cell lines, there is a trend to restoration of the ganglioside pattern and in enzymatic activity to that of the parent cell line, in spite of the continued presence of the viral DNA. Cells productively infected with virus do not show the decrease in aminosugartransferase activity. Cocultivation of transformed cells with enzymatically active cells did not result in reduced enzyme activity in the control cells, or in the restoration of enzyme activity in the virally transformed cells. Similarly, no change in enzyme activity occurred when admixing homogenates of the reciprocal cells. These findings are consistent with a similar repressor mechanism in both SV40 and polyoma virus-induced cell transformation. Recent studies indicate similar alterations of ganglioside pattern and synthesis in RNA virus-transformed cells.

0869 INDUCTION OF TUMORS IN THE LIVER OF HAMSTERS BY AN AVIAN ADENOVIRUS (CELO).

(E.) Stenback, W. A. (Baylor Coll. Med., Houston, Texas), J. P. Anderson, J. McCormick and J. J. Trentin. *J Natl Cancer Inst* 50(4):963-970, 1973.

The oncogenic activity of CELO virus, an avian adenovirus, was tested in randombred or LSH/LAK inbred hamsters inoculated as newborns. Approximately 50% of the animals developed tumors, usually at the site of injection. The randombred hamsters appeared to be slightly more susceptible to tumor formation. Injection by the subcutaneous (s.c.) route elicited more tumors than did injection by the intraperitoneal (i.p or intracerebral (i.c.) routes. In approximately 4% of the animals, remote tumors of the liver were found These liver tumors were classified as hepatocellular carcinomas or hepatomas (in 4 inbred hamsters; in 1 randombred), adenocarcinomas (in 5 randombred), and sarcomas (in 2 inbred). They developed in animals wh ther inoculated i.p., s.c., or i.c. No local tumors were found in these animals. Electron microscopic examination revealed a preponderance of cytoplasmic type A virus particles, especially in the hepatocellular carcinomas of inbred animals, and sporadic occurrence of budding type C and "spoked" hamster virus particles. All liver tumors tested (except the 2 sarcomas) induced antibodies to CELO T antigen.

0870 ADENOVIRUS-LIKE TRANSFORMATION OF HAMSTER EMBRYO CELLS MEDIATED BY SIMIAN VIRUS 40.

(E.) Diamandopoulos, G. T. (Harvard Med. Sch., Boston, Mass.) and S. Sanborn-Redmond. *Am J Pathol* 71(1):81-92, 1973.

Primary hamster cells, derived from embryos of 10 days gestation, were exposed in culture to the oncogenic effect of the DNA virus SV40. While the fibroblastoid cells transformed soon after virus inoculation, the small, round or oval cells also present preserved their characteristic morphologic features for a long time. When these cells finally transformed under the influence of SV40, they developed the capacity to induce, in the homologous host, small-, round-cell sarcomas, that were morphologically indistinguishable from neoplasms usually produced by adenoviruses. These findings indicate that different cells differ in their susceptibility to virus-mediated neoplastic transformation. They demonstrate also that the morphology of virally induced tumors is not always pathognomonic of their specific etiology.

0871 HISTOPROLIFERATIVE EFFECT OF RAUSCHER LEUKE VIRUS ON LYMPHATIC TISSUE. IV. LACTIC DEHYD GENASE VIRUS AND ERYTHROPOIETIN POTENTIATION OF THE ERYTHROID RESPONSE.

(E.) Snodgrass, M. J. (Oak Ridg Natl. Lab., Tennessee), J. M. Yuhas and M. G. Hanna, Jr. *J Natl Cancer Inst* 50(3):735-743, 1973.

Lactic dehydrogenase virus (LDV) and erythropoietin potentiate erythroid hyperplasia stimulated by Rausch leukemia virus (RLV) in the red pulp of the spleens o BALB/c mice. Studies of the incorporation of ^{59}Fe in the peripheral blood erythrocytes indicated that this stimulation was probably chronic with LDV and an earl acute response with erythropoietin. Both stimulants combination with RLV induced about a 60% greater sple megaly at 25 days than RLV alone when they were injec one hour before the leukemia virus. Histologically, splenomegaly appeared to be a consequence of enhanced erythroid hyperplasia associated with the RLV infecti C-type virus particles were found, ultrastructurally, budding from erythroblasts at various stages of their elopment in the splenic red pulp and from reticulo- es in the peripheral blood. Erythroblasts were ected by the leukemia virus at both early and late ges of development, but apparently some cells con- ued to differentiate at least to the enucleated l stage. Thus the stimulation of erythropoiesis the splenic red pulp by LDV provides another basis the synergistic interaction of LDV and RLV, and tinued differentiation of the RNA tumor virus- ected cells of the erythrocytic series supports contention that all cells of this compartment do undergo a true malignant transformation during the -induced splenomegaly.

2 C-TYPE PARTICLES IN NORMAL HUMAN PLACENTAS. *(E.)* Kalter, S. S. (Microbiol. Infectious ., Southwest Fdn. Res. Education, San Antonio, .), R. J. Helmke, R. L. Heberling, M. Panigel, K. Fowler, J. E. Strickland and A. Hellman. *J l Cancer Inst* 50(4):1081-1084, 1973.

ype particles were detected in four of six mal full-term human placenta after extensive nning with an electron microscope. The particles e morphologically identical to those observed baboon placentas, though fewer in number. Average ticle diameter was 100 nm, with an inner electron- ent core of approximately 50 nm. All particles e at the junction of the synctiotrophoblast the basement membrane or stroma. A physiologic ction for these particles is suggested.

3 STRUCTURAL STUDIES ON AVIAN MYELOBLASTOSIS VIRUS: CONDITIONS FOR ISOLATION AND BIO- MICAL CHARACTERISTICS OF THE CORE COMPONENT. *(E.)* omberg, K. (Natl. Cancer Inst., Bethesda, Md.), Gantt and S. H. Wilson. *Biochim Biophys Acta* (1):1-11, 1973.

core component of avian myeloblastosis virus V) was isolated in amounts sufficient for bio- mical analysis. Core material which approached ogeneity by electron microscopy had a composition weight of 86% protein, 8% RNA, and 6% phospholipid. s amount of phospholipid may indicate either con- ination by non-core structures, or more probably presence of phospholipid as an intrinsic consti- nt of AMV cores. The influence of various condi- ns of the isolation procedure on the yield of AMV es from intact virus was also examined. The hest yield of core material was obtained by use of odium phosphate buffer with EDTA, a 10-min exposure unfrozen Ficoll-purified AMV (protein concentration 1 mg/ml) to Sterox SL (final concentration 1.5%,), and velocity sedimentation through a layer of (w/w) sucrose into a pellet.

4 STRAIN DEPENDENCY OF HORMONALLY ACTIVATED C-TYPE RNA TUMOR VIRUS MARKERS IN MICE.) Fowler, A. K. (Natl. Cancer Inst., Bethesda,), P. J. McConahey and A. Hellman. *J Natl cer Inst* 50(4):1057-1059, 1973.

rine tissue of ovariectomized mice given two intramuscular injections of 1.0 μg estradiol-17β was assayed for group-specific antigen of C-type RNA tumor viruses and RNA-directed DNA polymerase. With or without estrogen treatment, AKR mice were consistently positive for group-specific antigen in complement fixation and radioimmune assays, while C57L/CR mice were uniformly negative. Detectable group-specific antigen in BALB/c and NIH Swiss mice was dependent on estrogen treatment. RNA-directed DNA polymerase activity, determined by incorporation of ^{3}H-thymidine triphosphate, closely paralleled group-specific antigen expression. Enzyme activity was high in AKR mice and low in C57L/CR mice, with neither strain showing estrogen dependency. In BALB/c and NIH Swiss mice, enzyme activity was markedly increased by estrogen treatment. The expression of the virogene markers in the four compared strains was in close agreement with their expected incidence of tumorigenesis.

0875 SV40 PROTECTION IN MYELOMA ONCOGENESIS. *(E.)* Reinisch, C. L. (Natl. Inst. Med. Res., London, England). *Nature* 242(5398):470-471, 1973.

Female C3H mice were protected against challenge with syngeneic myeloma tumor cells (1.5 x 10^6) by active or inactive SV40 (0.2 ml) given i.p. simultaneously with tumor cells or up to 13 days before and after challenge. Protection was most significant when mice were pretreated 13 days before tumor inoculation with ultraviolet-inactivated virus. No animals so treated died up to 75 days after inoculation. It is postulated that *in vivo* repair of ultraviolet-damaged virus might have occurred and resulted in new and varied antigen. Protection occurring when SV40 was given simultaneously with tumor may have been due to altered cell antigenicity resulting from absorption of virus to the tumor cell. Since the virus did not lyse tumor cells *in vitro,* it is not understood why SV40 can protect when given 13 days before or after tumor challenge.

0876 FURTHER STUDIES ON DNA-POLYMERASE ACTIVITY IN RAT EMBRYO CELLS INFECTED WITH PICODNAVIRUSES X 14 AND RV. *(E.)* Ricceri, G. (U. Catania, Italy), D. Duscio, M. Todaro and G. Russo. *G Batt Virol Immun* 64(1-4):7-13, 1971.

DNA polymerase in cell extracts from RV or X 14 virus-infected stationary rat embryo (RE) cells was studied to determine if the increase in activity following infection was similar to that of infected growing RE cells and whether or not enzymes present in infected and non-infected cells were antigenically related. Enzyme activity was determined by the rate of incorporation of ^{3}H TTP into acid-insoluble material in an *in vitro* assay. In the growing cultures, infection with either virus induces an increase of enzymatic activity with respect to controls from 12-72 hr postinfection (p.i.). In cultures infected in the stationary phase, there is a decrease of DNA-polymerase activity beginning at 12 hr p.i. In every case the changes in the levels of this enzymatic activity, both in resting or growing cells, following the infection, do not go beyond 50% of the control levels. No difference was observed in kinetics of heat-inactivation between polymerase extracted from infected and non-infected cells. DNA polymerase levels of infected cells were not affected by preincubation of cell extracts with either immune or preimmune sera. DNase activity, assayed by the lanthanum nitrate method, was unchanged in infected cells.

0877 EXPRESSION OF AVIAN SARCOMA VIRUS (ASV) GENOME B77 IN RAT TUMOUR CELLS: STUDY OF VIRUS PRODUCTION, GROUP-SPECIFIC ANTIGEN CONTENT AND ASSOCIATION WITH PERMISSIVE CELLS. *(E.)*

Popovic, M. (Slovak Acad. Sci., Bratislava, Czechoslovakia), M. Grofova, A. Lizonova and V. Thurzo. *Neoplasma* 19(5):371-384, 1972.

The production of infectious B77 avian sarcoma virus (ASV) in cell lines, clones and subclones derived from the rat RBI ascites tumor was investigated. In clones producing relatively few viruses (low producer clones) infectious B77 virus was seen only in 100-fold concentrated culture fluids and then only 5-20 ED_{50}/ml of infectious B77 were produced. Some high producer clones produced as many 8 x 10^3 ED_{50} of infectious B77. The degree of change from high to low producer clones was gradual. In studies of production by RBI cells of ASV group-specific (gs) antigen, it was found that low producer RBI clones made substantially less gs antigen than high producer clones. The lowest number of RBI cells required to produce tumors on inoculation into chickens was found with high B77-producing, high ASV gs antigen-producing, clones. Focus formation on chick cells by high-producer, high tumorigenicity, high gs antigen RBI cells was also more efficient than focus formation by low producer RBI cells. In rat cells transformed by B77 *in vitro*, a small amount of infectious virus could be detected.

0878 INHIBITION OF ERYTHROLEUKEMIA IN MICE BY INDUCTION OF HEMOLYTIC ANEMIA PRIOR TO INFECTION WITH RAUSCHER LEUKEMIA VIRUS. *(E.)*

Weitz-Hamburger, A. (Biol. Dept., New York U., N.Y.), T. N. Fredrickson, J. LoBue, W. D. Hardy, Jr., P. Ferdinand and A. S. Gordon. *Cancer Res* 33(1):104-111, 1973.

The hematological changes induced by Rauscher leukemia virus (RLV) under normal, erythropoietically stimulated, and erythropoietically depressed conditions were studied quantitatively. Polycythemia was produced in one group of 7-11 wk old BALB/C mice prior to i.p. injection of 0.1 ml of RLV by exposure of the mice to 0.4 atm of air, 19 hr/day, for a total of 219 hr. The animals were kept plethoric by weekly i.p. hypertransfusions of unwashed red cells obtained from donors of the same sex. Mice in a second group were rendered anemic by pretreatment with phenylhydrazine (PHZ) prior to inoculation with RLV. The mice received 0.1 ml of PHZ s.c. daily for five days. Suppression of erythropoiesis induced by posthypoxic polycythemia, and maintained by transfusion-induced plethora, retarded the disease process over a 4-month period, whereas stimulation of erythropoiesis by PHZ-induced hemolytic anemia resulted in a greatly decreased incidence of RLV disease. The measurement of *gs3* antigen by fluorescent antibody definite established that, while most erythroblasts in PHZ-treated mice were infected, normal maturation to erythrocytes occurred. In mice not pretreated with the drug prior to virus infection, reduced numbers of infected cells were seen, although maturation inhibition and consequent terminal anemia and hepatosplenomegaly were prominent.

0879 SYNCYTIAL ASSAY FOR THE PUTATIVE HUMAN C-TYPE VIRUS, RD-114, UTILIZING HUMAN CELLS TRANSFORMED BY ROUS SARCOMA VIRUS. *(E.)*

Rand, K. H. (Natl. Cancer Inst., Bethesda, Md.) and C. Long. *Nature* [*New Biol*] 240(101):187-190, 1972.

RD-114 cells, a line derived from human rhabdomyosarcoma which shed a nonfocus-forming type-C RNA virus, were grown to confluence and overlayed by cells of 13 lines producing mammalian type-C viru to test for syncytia induction among RD-114 cells. Of the tested lines, the only one to form syncytia was a Rous sarcoma virus-transformed human glioma cell line (118-MG-EH). Cells of this line yielded abundant syncytia on RD-114 cells. When 118 MG-EH cells were overlayed on cells other than RD-114, no syncytia were induced. Other viruses were studied for their ability to induce syncytia on 118 MG-EH cells; β-propiolactone-inactivated Sendai virus induced syncytia in a reaction seen only with 118 MG-EH cells. These syncytia were identical in form to those induced by RD-114 cells.

0880 *IN VITRO* NEOPLASTIC TRANSFORMATION OF HAMSTER EMBRYONIC SKIN AND SUBCUTANEOUS TISSUE CELLS WITH SMALL-PLAQUE AND LARGE-PLAQUE VARIANTS OF AN AVIAN ADENOVIRUS (CHICKEN EMBRYO LETHAL ORPHAN). *(E.)*

Pendola, R. G. (Animal Path. Dept., U. Rhode Island, Kingston) and V. J. Yates. *Cancer Res* 33(2):351-355, 1973.

Neoplastic cell lines were established when Syrian hamster embryonic skin and s.c. tissue cells were infected *in vitro* with small-plaque (SP) and large-plaque (LP) variants of chicken embryo lethal orphan virus. The SP cell line was made up of pleomorphic cells, whereas the LP cell line consisted of cells that were spindled to oval. The SP but not the LP cell line revealed virus-specific, intranuclear T antigen by the indirect immunofluores cent technique. Virus isolations were possible during the early passage levels in both of the cell lines. The SP- and LP-transformed cell lines induced tumors in weanling hamsters; however, the SP cells induced tumors at an earlier time in passage. Histologically, the SP tumors resembled anaplastic sarcomas, whereas the LP tumors were spindle-celled sarcomas. The SP and LP malignant ce lines as well as the tumors induced by them did not

ıal type C particles on electron microscope examina-
.on. These observations suggest that there was no
pe C particle transformation involved. However, since
ımster embryo cells may undergo spontaneous neoplastic
ansformations the possibility that this may occur in
ıe SP- and LP- infected cells cannot be completely
.iminated.

381 EFFECT OF 3-METHYLCHOLANTHRENE AND
DIMETHYLNITROSAMINE ON ANCHORAGE DEPENDENCE
RAT FIBROBLASTS CHRONICALLY INFECTED WITH RAU-
HER LEUKEMIA VIRUS. *(E.)* Mishra, N. K. (U.
braska Coll. Med., Omaha) and W. L. Ryan. *Int
Cancer* 11(1):123-130, 1973.

ıis report describes the loss of anchorage de-
ndence (AD-) after treatment with 3-methylcho-
nthrene or dimethylnitrosamine in rat embryo
broblasts which normally exhibit anchorage de-
ndence (AD+). AD+ fibroblasts which were
ronically infected with Rauscher leukemia virus (RLV)
ıowed the highest frequency of transformation to
- cells with chemical carcinogen treatment. A
ıall percentage of AD- cells also appeared in
infected control cells treated with dimethyl-
trosamine or at much higher frequency with 3-
thylcholanthrene. The virally infected cells
so showed a detectable level of transformation
AD- cells without added chemicals. However,
ıe combined effects of RLV and chemicals on the
equency of appearance of AD- cells were much
eater than the additive effects of the low fre-
ıency AD- cells produced by RLV alone or by a
ıemical carcinogen alone. Six different sets of
ft agar (AD-) clones were isolated from RLV+ cells
RLV- cells with and without treatment with chemical
.rcinogen. Each clone was then tested for its
ility to grow in media containing agamma calf
rum depleted of 3T3 growth factors, density
pendence of growth and colony-forming efficiency.
clear correlation between AD- transformation and
ıe ability to grow in factor-free medium was found.
ıe present study indicates that in the rat fibro-
.ast system loss of anchorage dependence is
obably not synonymous with morphologic trans-
rmation.

82 A FACTOR FROM A TRANSFORMED CELL LINE
THAT AFFECTS CELL MIGRATION. *(E.)*
.rk, R. R. (Imperial Cancer Res. Fund Labs.,
ndon, England). *Proc Natl Acad Sci USA* 70(2):
9-372, 1973.

rmal BALB/c3T3 mouse cells and SV40-transformed
LB/c3T3 cells (SV BALB/c373) were grown as
nolayers. A gap was cut in the monolayer and
e migration of cells into the cleared space
s observed. Untransformed cells did not enter
e gap unless there was serum in the culture
dium; in contrast, transformed cells migrated
the absence of serum. When normal BALB/c3T3
nolayers in serum-free medium were treated
th a factor extracted from the culture medium
highly malignant SV40-transformed hamster
lls (SV28), cell migration occurred. The SV28 migration factor had 1500 times the specific migration-promoting activity of whole calf serum, promoted overgrowth of BALB/c3T3 cells to high density, and prolonged cell survival in the absence of serum. The migration factor was not extractable from the medium of untransformed hamster cells.

0883 CARBOHYDRATE METABOLISM AND THE ENHANCEMENT OF UPTAKE OF GALACTOSE IN HAMSTER CELLS TRANSFORMED BY POLYOMA VIRUS. *(E.)* Kalckar, H. M. (Massachusetts Gen. Hosp., Boston), D. Ullrey, S. Kijomoto and S. Hakomori. *Proc Natl Acad Sci USA* 70(3):839-843, 1973.

The transport and metabolism of galactose (gal) were studied in untransformed (BHK, NIL) and polyoma virus-transformed (BHKpy, HILpy) hamster cell cultures. BHKby and NILpy, if preincubated in glucose (glu) or gal, incorporated isotopically labeled gal as intracellular gal and gal-1-P 2.5 to 3.0 times faster over a one hour label period than did untransformed BHK and NIL cells. The increase rate of gal incorporation was not accompanied by an increase in epimerization of UDP to UDP glu. Based on the extent of lactic acid and CO$_2$ production, it was concluded that gal was catabolized more slowly than glu in both untransformed and transformed cells. No xylose was produced from gal. Growth in 0.08% gal was just as rapid, however, as growth in glu. BHKpy and HILpy cells grown for 10 days in gal tended to become well-oriented in a multilayered pattern as opposed to the disoriented pattern observed in similar cells grown for 10 days in glu. They did not, however, exhibit contact inhibition. This orientation rapidly disappeared when the cells were subcultured into glu medium. After 24 hr growth in gal medium, both transformed and untransformed cells accumulated product which was identified by paper chromatography as almost exclusively galactitol.

0884 EVIDENCE FOR 30-40S RNA AS PRECURSOR OF THE 60-70S OF ROUS SARCOMA VIRUS. *(E.)* Canaani, E. (Virus Lab., U. California, Berkeley), K. V. D. Helm and P. Duesberg. *Proc Natl Acad Sci USA* 72(2):401-405, 1973.

Prague Rous sarcoma virus (RSV) was harvested from chick embryo cells at intervals of 3 min (3 min RSV) or of 5 hr. Viruses harvested at 3 min intervals had the same sedimentation coefficients ($\sim$600S), distributions and template characteristics for DNA polymerase, and buoyant densities as viruses harvested at 5 hr intervals. The sedimentation distribution of RNA from 3 min RSV showed a major class at 30-40S; 25% of RNA sedimentation at 60-70S, and there was a third class at 4-12S. Schmidt-Ruppin RSV showed more 60-70S RNA than 30-40S RNA. When 3 min RSV was incubated at 40C in cell growth medium or Tris-saline, most of the 30-40S changed to 60-70S RNA. The electrophoretic mobility of the 30-40S RNA from the 3 min RSV was lower than that of the 30-40S RNA obtained by complete melting of the 3 min RSV

60-70S RNA; after heating, the mobilities of the RNA fractions became similar. Heating also released some small RNAs from 30-40S RNA of 3 min RSV. The template activity of the 3 min RSV 30-40S RNA was five times lower than that of the 60-70S RSV RNA.

0885 PERSISTENCE OF ROUS SARCOMA VIRUS IN TRANSFORMED NON-PERMISSIVE CELLS: CHARACTERISTICS OF VIRUS INDUCTION FOLLOWING SENDAI VIRUS-MEDIATED FUSION WITH PERMISSIVE CELLS.

(E.) Vigier, P. (Inst. Radium, Faculty Sci., Orsay, France). *Int J Cancer* 11(2):473-483, 1973.

Virus induction in heterokaryons formed by non-permissive cells transformed by Rous sarcoma virus (RSV) and permissive chick embryo (CE) cells, in the presence of inactivated Sendai virus, was studied with a clone of hamster BHK21 fibroblasts transformed by Schmidt-Ruppin strain RSV (clone RS2) and subclones derived from this clone. The main results have been the following: (1) The number of heterokaryons giving rise to foci of Rous cells (focus-forming infective centers, or FFICs) in mixed cultures is proportional to the number of RS2 cells when the initial CE:RS2 cell ratio attains 30, and almost all heterokaryons are then formed from single RS2 cells. (2) Recovery of FFICs also depends on the dose of Sendai virus. (3) Following fusion of RS2 cells with cells from different chick embryos the recovery of FFICs varies in parallel with the number of foci produced by free virus in cultures of these embryos. (4) The latent period of virus induction from the time of cell fusion is about 24 hr and somewhat longer than that of viral replication in newly infected CE cells. The growth of induced virus is similar to that of virus produced after *de novo* infection. (5) Virus induction requires no DNA synthesis but is inhibited, reversibly, by treatment for a few hours with inhibitors of DNA-dependent RNA synthesis (actinomycin D) and protein synthesis (puromycin and cycloheximide). Hence, non-permissiveness of RS 2 cells may be due to a partial block of transcription of the viral replicative DNA (provirus) present in the cells, and induction in heterokaryons to switching from partial to full transcription and translation of the provirus. However, it is also possible that some function, or factor(s) of the CE cells is required for virus production in heterokaryons. (6) The study of a subclone of RS2 cells 100- to 1000-fold less inducible than other subclones suggesting that, in this subclone, non-permissiveness may be due to a permanent block of synthesis of all viral proteins, at the level of transcription of the provirus.

0886 CYTOTOXICITY OF ROUS SARCOMA CELLS BY LYMPHOID CELLS IN JAPANESE QUAILS.

(E.) Hayami, M. (U. Washington Med. Sch., Seattle), I. Hellström, K. E. Hellström and K. Yamanouchi. *Transplant Proc* 5(1):977-980, 1973.

Cell-mediated immunity to Rous sarcoma was studied in Japanese quails using the microcytotoxicity test. Spleen cells, but not thymus or bursa dependent cells, taken from quails with regressing Rous sarcoma virus (RSV)-induced tumors were cytotoxic against these tumor cells *in vitro*. The cytotoxic effect was due to cell-mediated immunity against tumor-specific cell-associated antigens and not to histocompatibiliry differences between the tumor cells tested. Spleen cell-mediated cytotoxicity was bursa independent and thymus dependent since it was abolished by thymectomy, but not by bursectomy. Spleen cells from quails with progressively growing tumors had no detectable cytotoxic effect. In addition, spleen cells from quails with progressively growing tumors could decrease the killin effect of spleen cells from untreated quails with regressing tumors but not from bursectomized regressors. These latter results could not be explained.

0887 ROUS SARCOMAS INDUCED BY HETEROTRANSPLANTATION OF VIROGENIC RAT TUMOUR CELLS IN MICE.

(E.) Bubenik, J. (Inst. Exp. Biol. Genetics, Czechoslovak Acad. Sci., Prague), M. Baresova, B. Micheel, V. Sovova, R. El Ridi and M. Keclikova. *Int J Cancer* 10(3):527-538, 1972.

The virogenic sarcoma RSL was induced in inbred Lewis rats by the Schmidt-Ruppin variant of Rous virus (SR-RSV). Heterotransplantation of RSL cells to newborn C57BL/10Sn mice gave rise to mouse sarcomas in approximately 10% of the recipients. The properties of one of these mouse sarcomas, designat as RSM, were compared with those of the original rat tumor line RSL. It was found that (1) RSL cells did not contain detectable amounts of infectious chicken, rat or mouse sarcoma virus. However in contact with permissive chicken fibroblasts the RSL cells produced malignant conversion of avian cells accompanied by the formation of infectious RSV. RSL cells carried a tumor-associated transpla tation antigen (TATA) of Rous sarcomas, a group-specific (gs) antigen of avian leukoviruses and a membrane antigen (MA), characteristic of Rous sarcomas, detectable by indirect immunofluorescence and cross-reacting with MA of mouse Rous sarcomas. The MA characteristic of tumors induced by mouse leukoviruses was not detected in the membrane of RSL cells. (2) RSM cells did not contain detectabl amounts of oncogenic avian, mouse or rat viruses in an infectious form. Neither contact of these cells with permissive chicken c/o fibroblasts nor heterotransplantation of RSM tumors to chickens led to malignant conversion of avian cells. Gs antigen of avian leukoviruses was not detectable in RSM cells. However, TATA and MA characteristic of Rous sarcomas were present. Neither MA nor TATA induced by mouse leukoviruses was detected in RSM cells. The results obtained indicate that the non-infectio RSV genome present in rat virogenic cells is able to induce tumors in a further mammalian species.

0888 GROWTH INHIBITION OF TRANSFORMED CELLS BY LIPOPOLYSACCHARIDES (LPS) FROM *S. MINNESOT* R MUTANTS.

(E.) Trudel, M. (Dept. Cell Biol., U. Sherbrooke, Canada), R. Lallier, C. Brailovsky and V. N. Nigam. *Proc Am Assoc Cancer Res* 13(March):124, 1972.

889 ESTABLISHMENT OF A CELL CULTURE FROM AN OWL MONKEY TUMOR INDUCED BY *HERPESVIRUS SAIMIRI* (HVS). *(E.)* Ablashi, D. V. (Natl. Cancer Inst., Bethesda, Md.), G. R. Armstrong, J. Heine and R. H. Adamson. *Proc Am Assoc Cancer Res* 13(March):124, 1972.

890 INTERACTIONS BETWEEN TWO DISSIMILAR AVIAN ONCOGENIC VIRUSES IN TISSUE CULTURE. *(E.)* Frankel, J. W. (Life Sci., Inc., St. Petersburg, Florida) and V. Groupe. *Proc Am Assoc Cancer Res* 13(March):122, 1972.

0891 TUMORIGENICITY AND ANTIGENICITY OF SV40-INFECTED CELLS. *(E.)* Wright, P. W. (Natl. Cancer Inst., Bethesda, Md.) and H. S. Smith. *Proc Am Assoc Cancer Res* 13(March):116, 1972.

0892 VIRUSES RESEMBLING C-TYPE PARTICLES IN HUMAN TUMORS. *(E.)* Stewart, S. E. (Sch. Med., Georgetown U., Washington, D.C.), G. Kasnic Jr. and C. Draycott. *Proc Am Assoc Cancer Res* 13(March):115, 1972.

0893 MAXILLOFACIAL LYMPHOMA IN GUATEMALA. *(E.)* Fortuny, I. E. (U. Minnesota Med. Sch., Minneapolis) and C. Vizcaino-Gamez. *Lancet* (7796): 198-199, 1973.

0894 EPSTEIN-BARR VIRUS: DETECTION OF GENOME IN SOMATIC CELL HYBRIDS OF BURKITT LYMPHOBLASTOID CELLS. *(E.)* Glaser, R. (M. S. Hershey Med. Ctr., Hershey, Pa.) and M. Nonoyama. *Science* 179(4072):492-493, 1973.

0895 VIRUS-LIKE PARTICLES IN ACUTE LYMPHOBLASTIC LEUKEMIA. *(E.)* Sun, C. N. (VA Hosp., Little Rock, Arkansas), G. E. Byre and H. Pinkerton. *Experientia* 29(1):100-101, 1973.

0896 GREY MATTER EDEMA: THE MARGINAL ZONE OF AUTOCHTHONOUS VIRALLY-INDUCED GLIOMAS. *(E.)* Sipe, J. C. (Sec. Neurol., U. Chicago, Ill.), N. A. Vick and D. D. Bigner. *J Neurol Sci* 17(2): 185-191, 1972.

0897 OBSERVATIONS OF APPARENT C-TYPE PARTICLES IN BABOON (*PAPIO CYNOCEPHAIUS*) PLACENTAS. *(E.)* Kalter, S. S. (Southwest Fdn. Res. Educ., San Antonio, Tex.), R. J. Helmke, M. Panigel, R. L. Heberling, P. J. Felsburg and L. R. Axelrod. *Science* 179(4080):1332-1333, 1973.

0898 POLYADENYLIC ACID IN VISNA VIRUS RNA. *(E.)* Gillespie, D. (Litton Bionetics, Inc., Bethesda, Md.), K. Takemoto, M. Robert and R. C. Gallo. *Science* 179(4080):1328-1330, 1973.

0899 ISOLATION OF A C-TYPE VIRUS FROM MOUSE MYELOMA CELLS. *(E.)* Karpas, A. (Dept. Med., U. Cambridge, England) and J. Cawley. *Eur J Cancer* 8(3):363-364, 1972.

0900 FOWL PLAGUE VIRUS ADAPTED TO HUMAN EPITHELIAL TUMOR CELLS AND HUMAN MYELOBLASTS *IN VITRO*. I. CHARACTERISTICS AND REPLICATION IN MONOLAYER CULTURES. *(E.)* Gerber, A. (Dept. Med., U. Zurich, Switzerland), C. Sauter and J. Lindenmann. *Arch Gesamte Virusforsch* 40:137-151, 1973.

0901 COMPARISON OF SV40 RNA FROM INFECTED CELLS AND SV40 cRNA MADE BY RNA POLYMERASE FROM *ESCHERICHIA COLI*. *(E.)* Peterson, E. E. (Dept. Virol. Inst. Hyg. U. Freiburg/Breisgau, W. Germany), N. Mueller, M. Heufer and G. Brandner. *Arch Gesamte Virusforsch* 39(4):381-388, 1972.

0902 INFLUENCE OF CELL DENSITY ON THE ACRIDINE ORANGE BINDING TO DEOXYRIBONUCLEOPROTEIN COMPLEX IN LEUCOCYTES FROM PATIENTS WITH INFECTIOUS MONONUCLEOSIS AND ACUTE LEUKAEMIA. *(E.)* Bolund, L. (Karolinska Inst., Stockholm, Sweden), L. E. De Bault, G. E. Foley, G. Gahrton, D. Killander, W. C. Moloney, R. Rigler, A. Skarin and B. Wahren. *Acta Haematol (Basel)* 48:227-233, 1972.

0903 ONCOGENIC ACTIVITY OF FELINE FIBROSARCOMA VIRUS IN NEWBORN PIGS. *(E.)* Pearson, L. D. (Comparative Oncology Lab., U. California, Davis), S. P. Snyder and C. D. Aldrich. *Am J Vet Res* 34(3): 405-409, 1973.

0904 BURKITT'S LYMPHOMA IN ETHIOPIA. *(E.)* Sudarsanam, T. (Ethio-Swedish Pediatric Clin., Addis Ababa, Ethiopia), D. Habte and T. Asfaw. *East Afr Med J* 49(7):502-508, 1972.

0905 A POXVIRUS IN A MARSUPIAL PAPILLOMA. *(E.)* Papadimitriou, J. M. (Dept. Path., U. Western Australia, Perth) and R. B. Ashman. *J Gen Virol* 16(1):87-89, 1972.

0906 INTRACRANIAL TRANSPLANTATION OF THE TUMOR INDUCED BY ADENOVIRUS TYPE 12 IN SYRIAN HAMSTER — AN EXPERIMENTAL BRAIN TUMOR MODEL. *(E.)* Tabuchi, K. (Okayama U. Med. Sch., Japan). *Acta Med Okayama* 25(6):605-613, 1971.

0907 MAMMARY NODULIGENIC ACTIVITY OF THE SPERM FROM VARIOUS HIGH MAMMARY TUMOR STRAIN MICE IN HISTOCOMPATIBLE RECIPIENTS OF DIFFERENT AGES. *(E.)* Squartini, F. (Inst. Path. Anat. Hist., U. Pisa, Italy) and G. B. Bolis. *Lav 1st Anat Istol Patol Perugia* 31(2):59-63, 1971.

0908 INFECTIOUS MONONUCLEOSIS AND RETICULUM CELL SARCOMA. *(E.)* Andersen, E. (Med. Dept., Gentofte Hosp., Copenhagen, Denmark) and A. Videbaek. *Scan J Naematol* 9(4):400-402, 1972.

0909 EFFECTS OF CYCLIC AMP ON SV3T3 CELLS IN CULTURE. *(E.)* Paul, D. (Armand Hammer Ctr. Cancer Biol., Salk Inst., San Diego, Calif.). *Nature* [*New Biol*] 240(101):179-181, 1972.

0910 RADIOPROTECTIVE EFFECT OF RAUSCHER LEUKEMIA VIRUS IN THE SJL/J MOUSE. II. DEVELOPMENT OF RADIORESISTANCE AND THE DISTRIBUTION OF MORTALITY. *(E.)* Markoe, A. M. (Clin. Radiation Therapy Res. Ctr., Pittsburgh, Pa.). *Radiat Res* 53(1):115-123, 1973.

0911 SV40 VIRUS TRANSFORMATION OF MOUSE 3T3 CELLS DOES NOT SPECIFICALLY ENHANCE SUGAR TRANSPORT. *(E.)* Romano, A. H. (Microbiol. Sec., U. Connecticut, Storrs) and C. Colby. *Science* 179 (4079):1238-1240, 1973.

0912 ELECTRON MICROSCOPIC STUDY OF FRIEND LEUKEMIA VIRUS (FLV) EXPOSED TO SPECIFIC ANTI-SERUM. *(E.)* Sato, T. (Sloan-Kettering Inst., New York, N.Y.), E. de Harven and C. Friend. *Proc Am Assoc Cancer Res* 13(March):111, 1972.

0913 MORPHOLOGY OF INTERNAL VIRAL COMPONENTS. *(E.)* Schidlovsky, G. (Pfizer Inc., Maywood, N.J.), K. Harewood and B. Cummings. *Proc Am Assoc Cancer Res* 13(March):111, 1972.

0914 REPAIR OF CHEMICALLY DAMAGED CELLULAR AND FOREIGN DNA IN MAMMALIAN CELLS. *(E.)* Satoh, T. (Temple U. Sch. Med., Philadelphia, Pa.). *Proc Am Assoc Cancer Res* 13(March):109, 1972.

0915 ISOLATION OF ONCOGENIC RNA VIRUS FROM SJL/J MICE. *(E.)* Chang, S. S. (Natl. Cancer Inst., Bethesda, Md.) and L. W. Law. *Proc Am Assoc Cancer Res* 13(March):112, 1972.

0916 SPONTANEOUS DIFFERENTIATION IN AN ONCORNAVIRUS CARRYING STABLE CELL LINE: FINE STRUCTURE. *(E.)* Arnold, W. J. (Michigan Cancer Fdn., Detroit). *Proc Am Assoc Cancer Res* 13(March): 112, 1972.

0917 COMPARING THE DNA'S SYNTHESIZED BY ENZYME FRACTIONS EXTRACTED FROM APHTHOUS FEVER VIRUS WITH THOSE PRODUCED BY ATYPICAL EPITHELIOMA CELLS OF THE RAT. *(Fr.)* Sakouhi, M. (Central Lab. Vet. Res. Paris, France), J. Chory, L. Dhennin and L. Dhennin. *C R Acad Sci* [*D*] *(Paris)* 276(3):411-414, 1973.

See also:

* (Rev): 0614, 0630, 0637, 0638, 0642
* (Phys): 0767
* (Immun): 0919, 0922, 0925, 0931, 0935, 0939,
0940, 0941, 0950, 0955, 0956, 0957,
0959, 0964, 0968, 0970, 0971, 0974,
0976, 0980, 0982, 0990, 1017, 1021
* (Epid-Biom): 1042, 1043

918 PRODUCTION OF ANTISERUM IN MICE TO HUMAN LEUKAEMIA-ASSOCIATED ANTIGENS. *(E.)*

ker, M. A. (Mount Sinai Sch. Med. City U. New York, .Y.) and R. N. Taub. *Nature* [*New Biol*] 241(107):93-94, 973.

ntisera to human leukemic blast cells were produced n CBA/J mice after first inducing tolerance to the atient's remission leucocytes with 200 mg/kg cyclo- nosphamide injected i.p. Intravenous injection of 0^8 remission leucocytes 4 hr later was follwed on ays 7 and 14 by s.c. and i.p. injections of 10^8 blast ells. Control antisera were obtained from mice in- ected with cyclophosphamide alone and then blast ells on days 7 and 14. Antisera from mice rendered olerant to remission leucocytes were more toxic to last cells than to remission cells in tests with an cute myeloblastic leukemia (AML) patient and an acute ymphoblastic leukemia (ALL) patient. Control antisera ere equally cytotoxic to blasts and normal leucocytes, s was anti-human thymocyte serum. Absorption of anti- era with remission leucocytes removed all cytotoxicity o normal cells and reduced but did not eliminate ytotoxicity to blast cells. Similarly, remission bone arrow cells from the ALL patient failed to absorb anti- last activity. Antisera against leukemic cells of the irst patient were selectively toxic to blast cells of he second patient, but the reverse cross-reactivity as not clearly selective. Rabbit complement was not easurably cytotoxic to either blast cells or remission eucocytes when used alone; rabbit anti-carcinoembryonic ntigen serum was more toxic to leukemic blast cells han remission cells of the same patient in both cases ested.

919 ANTIBODIES TO EBV RELATED ANTIGENS IN WEST MALAYSIAN CHILDREN. *(E.)*

Tan, D. S. K. Inst. Med. Res., Kuala Lumpur, Malaysia) and G. Henle. *ed J Malaya* 27(1):27-29, 1972.

Sera from West Malaysian children 1-10 yr old were titrated in the indirect immunofluorescence test for antibodies to Epstein-Barr viral capsid antigens (VCA) and antibodies to components D and R of the Epstein Barr Virus (EBV)-induced early antigens (EA) complex. Eighty-three percent of 1-2 yr old children already had antibodies to VCA and 90-100% of older children were positive. None had antibodies to the D component of the EA complex; 3% had antibodies to the R component. The failure to detect anti-D denotes that none of the children had undergone recent primary EBV infections. The high anti-VCA titers accompanied by low anti-R titers appear to reflect the extent of the EBV carrier state which becomes established with a high degree of regularity in the lymphoreticular system after primary EBV infection. The data explain why infectious mononucleosis is not observed in West Malaysian children: Few are still susceptible to EBV in adolescence when IM is the likely result of delayed primary EBV infections.

0920 CELL-MEDIATED IMMUNITY *IN VITRO* AGAINST SYNGENEIC MOUSE PLASMA TUMOUR CELLS. *(E.)*

Wagner, H. (Royal Melbourne Hosp., Victoria, Australia) and M. Röllinghoff. *Nature* [*New Biol*] 241(106): 53-54, 1973.

To sensitize thymus-derived (T) cells against tumor-associated transplantation antigens (TATA), cortisone-resistant (CR) BALB/c thymocytes were cultivated with irradiated syngeneic HPC-10 plasma cell tumor cells. The *in vitro* co-cultivation resulted in a cell-mediated cytotoxic immune response, as shown by lysis of ^{51}Cr-labeled HPC-108 target cells. Sensitized CR thymocytes lysed the HPC-108 cells up to 42% above background lysis in the presence of unsensitized allogeneic CBA CR thymocytes. Cytotoxic activity against HPC-6 tumor cells from NZB mice, P815 x-2 tumor cells from DBA/2 mice, and allogeneic EL4 tumor cells was also demonstrated but at much reduced levels. *In vitro* co-cultivation of NZB CR thymocytes and irradiated syngeneic HPC-6 plasma tumor cells also elicited a cell-mediated cytotoxic immune response. The cytotoxic activity was not as much restricted to the immunizing syngeneic tumor cells. These results agree with another recent *in vitro* study showing that θ bearing lymphocytes can adoptively transfer *in vitro* immunity to TATA of tumors. However, a possible participation of activated macrophages in the cytotoxic effector phase cannot be excluded in view of the observed nonspecific killing effect.

0921 RELATIONSHIP BETWEEN IMMUNE REACTIVITY AND DEVELOPMENT OF THYMIC LYMPHOMA BY N,N'-DIMETHYLNITROSOUREA IN C3Hf/Bi MICE. *(E.)*

Motoi, M. (Okayama U. Med. Sch., Japan), K. Yanagida and K. Ogawa. *Acta Med Okayama* 25(6):629-641, 1971.

Immunologic response was determined during the course of thymic lymphoma development in five to six wk old male C_3Hf/Bi mice receiving weekly s.c. injections of N,N'-dimethylnitrosourea (DMNU; 80 mg/kg body wt). Repeated antibody plaque assays were conducted using spleen cells and peripheral leukocytes taken from control and DMNU treated animals challenged five days previously by i.p. injection of sheep red blood cells. The results showed that the immunologic response of DMNU treated mice declined over an 8 wk period to less than 10% that of control mice. Serum hemagglutinin and hemolysin titers also decreased considerably. These changes were paralleled by a decrease in the peripheral leukocyte count, by atrophic histologic changes in the thymus and spleen, and by dystrophic and finally neoplastic thymic transformation at the end of 8 wk. The only change observed in lymph node sections was a slight decrease in the number of lymphocytes. Antibody production capacity declined before the appearance of thymic tumor cells and continued to decline during the course of tumor progression.

0922 GENETIC CONTROL OF THE GROUP-SPECIFIC ANTIGEN OF MURINE LEUKAEMIA VIRUS. *(E.)*

Taylor, B. A. (Jackson Lab., Bar Harbor, Maine), H. Meier and R. J. Huebner. *Nature* [*New Biol*] 241(110): 184-185, 1973.

A second example is reported of single gene control of the expression of group-specific antigen (gs-AG) in inbred mice. A number of congenic resistant (CR) lines were tested for their effects on gs-AG expression in the spleens of 5-7 wk old mice. Each tested line differed from their common inbred partner strain, C57BL/10Sn (B10), by a single histocompatability gene. In contrast to B10 and two CR lines, B10.129(5M) and B10.129(21M), which were gs-AG negative, spleens of B10.D2(58N) mice (58N) reacted consistently gs-AG positive in a direct complement fixation test. Genetic analysis of the difference in gs-AG titers between B10 and 58N suggested that 58N contained a single contaminant gene from strain DBA/2J that controls gs-AG expression. This was confirmed by segregation data for F_2 and backcross generations obtained by crossing the B10 and 58N strains. Joint segregation of gs-AG with β-chain-hemoglobin locus (*Hbb*) revealed no significant deviation from free recombination in both the F_2 and backcross generations, nor was any association with the *p* locus observed. In crossed strains, lack of gs-AG expression was dominant over expression and the presence of gs-AG was unaccompanied by detectable complete, replicating leukemia virus.

0923 EVIDENCE FOR NON-EXPOSED H-2 ANTIGENS IN IMMUNORESISTANT MURINE TUMOUR. *(E.)*

Friberg, S., Jr. (Karolinska Inst., Stockholm, Sweden) and B. Lilliehook. *Nature* [*New Biol*] 241 (108):112-114, 1973.

Cells from two ascites sublines of the spontaneous murine mammary adenocarcinoma TA3 of strain $A(H-2^a)$ were tested for antigenicity and antibody binding capacity. The lines were TA3/Ha, which shows wide homotransplantability and more than ten-fold reduction in the amount of expressed surface H-2 antigens in relation to other lines, and TA3/ST, which is regularly rejected by allogeneic mice. Viability of TA3/Ha and TA3/ST cells after disruption by sonication or lyophilization was less than 10%; the cells did not differ in adsorptive capacity after exposure to 10-60 s of 20 kcycles s^{-1} of sonic energy for various time periods. Incubation in 2 ml neuraminidase removed more than 80% of surface sialic acid from TA3/Ha and TA3/St cells without decrease of cell viability. Results from antibody binding experiments with $(ACA \times C57BL)F_1$ anti-C3H antiserum and $(ACA \times C3H)F_1$ anti-DBA/2 antiserum indicated that disrupted TA3/Ha cells bound several times more of the anti-C3H antibodies than living TA3/Ha cells. By contrast, neither sonication nor lyophilization altered the antigenicity of TA3/ST cells. The increase in specific antibody capacity of disrupted TA3/Ha cells demonstrates that these cells have considerable amounts of $H-2^a$ antigens synthesized but not exposed on the outer surface of the plasma membrane.

0924 CELL-MEDIATED AND SERUM BLOCKING REACTIVITY TO TUMOR ANTIGENS IN PATIENTS WITH MALIGNANT MELANOMA. *(E.)*

Heppner, G. H. (Roger Williams Gen. Hosp., Providence, Rhode Island), L. Stolbach, M. Byrne, F. J. Cummings, E. McDonough and P. Calalresi. *Int J Cancer* 11(2):245-260, 1973.

The reactivity of human lymphocytes, separated from peripheral blood by procedures involving sedimentation through plasmagel and settling on a glass surface, on survival or growth of melanoma tumor cells is reported. Assay of reactivity was by either a colony inhibition in dilute agar (GEL) technique or a microcytotoxicity assay (MC). In 54 experiments with lymphocytes from normal donors, inhibition of melanoma cell viability, as compared to growth in medium alone, was seen 25 (46.3%) times, stimulation was observed seven (12.9%) times and no effect 22 (40.7%) times. These results were not influenced by matching of the lymphocyte and tumor-cell donor for ABO blood group. Inhibition was seen less often, however, in tests with the GEL method than in those with the MC assay. Eighty-two experiments assessed the effect of lymphocytes from melanoma patients on melanoma cell viability. Inhibition to an extent greater than that of normal lymphocytes tested at the same time was seen 65 (79.3%) times. There was no difference in the frequency of detection of inhibition with lymphocytes from patients with local, regional, or widespread disease, although the percentage inhibition was less in the latter group. No differences were seen between patients with clinically active versus inactive disease. Only one of five patients who were within a week of death was reactive. Reactivity was detected as frequently with allogeneic as with autochthonous melanoma target cells. Serum blocking factor (SBF), capable of inhibiting *in vitro* cell-mediated reactivity to melanoma tumor cells, was assayed in serum from melanoma patients at various stages of disease. SBF was detected at some time in 30% of patients with local, 88.9% with regional, and 80.0% with widespread disease. It was found in 42.9% of those with clinically inactive and 70.6% with active melanoma. However, detection of SBF was found to fluctuate from sample to sample taken at different times during the course of disease in individual patients regardless of clinical status. The importance of sequential determinations of SBF activity for patient assessment is stressed.

0925 SURFACE ANTIGENS ON CAT LEUKEMIC CELLS INDUCED BY FELINE LEUKEMIA VIRUS: AREA DENSITY AND ANTIBODY-BINDING AFFINITY. *(E.)*

Boone, C. W. (Natl. Cancer Inst., Bethesda, Md.), F. Goodin and T. G. Kawakami. *J Virol* 11(4):515-519, 1973.

The binding of autologous bovine antibody to feline leukemia virus-induced cell surface antigens (FeCSA) on cat leukemia cells was studied by performing certain titration procedures with a mixture of immune and normal sera labeled with different iodine radioisotopes (paired-label technique). By using plots of titration data which conformed to linear equations derived from the mass action law, the following constants were determined. (i) The density of FeCSA was 2.03×10^6 sites per cell, or 5,230 sites per μm^2. (ii) The equilibrium constant of the FeCSA-antibody reaction was 2.67×10^7, from

ich the antibody binding affinity or standard
ee energy of the FeCSA-antibody bond was deter-
ned to be -10.48 kcal (-43,869.28 J) per mol.
en a cell titration of paired-labeled immune and
rmal serum protein is performed, the coefficient
determined by the cell titration, gives the
crograms of antibody present in 1 mg of serum
otein, or, if multiplied by the protein con-
ntration of the antiserum, the concentration of
tibody in micrograms per milliliter.

26 PAPILLOMA INDUCTION IN DIFFERENT AGED SKIN GRAFTS TO YOUNG RECIPIENTS.

(E.) Ebbesen,
(Inst. Med. Microbiol., U. Copenhagen, Denmark).
ture 241(5387):280-281, 1973.

rsal skin grafts from 2 or 14 months old BALB/c
ce were transferred to 2 months old syngeneic
ce. Twelve months later 25 μl of acetone con-
ining 40 μg/ml of 7,12-dimethylbenz(a)anthracene
MBA) was applied to the grafts. Papillomas first
peared on the grafts four weeks after DMBA
eatment and reached their maximum number three
eks later. Approximately 60% of grafts from
d donors and 25% of grafts from young donors
re papillomas. Two months after the application
DMBA, four carcinomas appeared on skin from
old donor and one on the skin from a young
nor. No tumors developed in 35 male and 30
male mice bearing grafts treated only with
etone, indicating an age-dependent susceptibility
skin to a chemical carcinogen.

27 SERUM ALPHA-FETO-PROTEIN VII. THE RANGE OF APPARENT SERUM VALUES IN NORMAL PEOPLE, EGNANT WOMEN, AND PRIMARY LIVER CANCER HIGH RISK PULATIONS.

(E.) Purves, L. R. (Atomic Energy
., Pretoria, South Africa), W. R. Branch, E. W.
ddes, C. Manso and M. Portugal. *Cancer* 31(3):
8-587, 1973.

radioimmunoassay for alpha-feto-protein (AFP) has
en used to compare the distribution of AFP levels
adults and children from areas of southern Africa
o have increased susceptibility to primary liver
ncer (PLC) and nonsusceptible children of other
ces from the same areas. The serum AFP values
pregnant women, ill Bantu patients, proven PLC
ses who were AFP-negative on immunodiffusion,
d patients with hepatitis have been determined.
ople from PLC high-risk areas appear to have
ised AFP levels more often than in other popu-
tions. There is a complete overlap between
rmal levels and levels in hepatitis and PLC.
e dynamics of the AFP response assists in dis-
nguishing hepatitis from PLC. The AFP in maternal
rum shows fluctuations, a fall before delivery,
d a spike of AFP on disruption of the placenta.

28 MEMBRANE FRACTIONS FROM RAT HEPATOMA. II. IMMUNOCHEMICAL CHARACTERIZATION OF TERGENT-SOLUBLE MEMBRANE ESTERASES, GLYCOSIDASES D LEUCYL-β-NAPHTHYLAMIDASE.

(E.) Blomberg, F. (Dept. Immunol., U. Stockholm, Sweden) and M. Raftell. *Biochim Biophys Acta* 291(2):431-441, 1973.

A plasma membrane fraction (tP) and two microsomal fractions (rough tR, smooth tSA) were isolated from cells of a 4-dimethylaminoazobenzene-induced rat hepatoma (D23). Material from fractions was reacted against anti-fraction antisera; immunodiffusion tests were performed and material was stained for active antigens associated with esterase, glycosidase and leucyl-β-naphthylamidase. The tP and tSa fractions precipitated esterase active antigens identical to those present in liver membranes. The esterase from tP hydrolyzed only substrate α-naphthyl acetate while the esterase from tSa also hydrolyzed longer fatty acid substrates. The three glycosidases studied (β-D-glucuronidase, β-D-galactosidase and *N*-acetyl-β-D-glucosaminidase) were all found in the tP fraction and with varying distributions in tumor microsomes. One leucyl-β-naphthylamidase active antigen was present in tP, tSa and tR; another immunologically different antigen found in liver microsomes only.

0929 IMMUNOLOGIC STUDIES OF HUMAN SEROUS CYSTADENOCARCINOMA OF OVARY. DEMONSTRATION OF TUMOR-ASSOCIATED ANTIGENS.

(E.) Bhattacharya, M. (Roswell Park Mem. Inst., Buffalo, N.Y.) and J. J. Barlow. *Cancer* 31(3):588-595, 1973.

Evidence is presented for the presence of a tumor-specific (associated) antigen(s) in human serous cystadenocarcinoma which is absent in normal ovarian tissue and other histologically similar organs. Antitumor antibodies were demonstrated by agar gel diffusion, immunoelectrophoresis, and immunofluorescence after extensive absorptions of the antitumor antisera to remove antibodies against normal ovarian and related organ components, blood group substances, nonspecific serum and tissue antigens, and fibrin. Fractionation and characterization of the tumor-specific antigen(s) suggests a high molecular weight mucoprotein with β-electrophoretic mobility which gave a single antigenic peak when fractionated on Sephadex G-200 columns. The tumor-specific antigen(s) was found to be immunologically unrelated to carcinoembryonic antigen (CEA) or the histocompatability antigens. The demonstration of antigenic cross-reactivity between pooled and individual tumor specimen extracts suggests a common tumor antigen(s) for ovarian serous cystadenocarcinoma.

0930 SPECIFIC INHIBITION OF SARCOMA-SPECIFIC CELLULAR IMMUNITY BY SERA FROM PATIENTS WITH GROWING SARCOMAS.

(E.) Cohen, A. M. (Natl. Cancer Inst., Bethesda, Md.), A. S. Ketcham and D. L. Morton. *Int J Cancer* 11(2):273-279, 1973.

Sera from sarcoma-free patients and patients bearing various soft tissue and skeletal sarcomas were assayed for their effect on *in vitro* lymphocyte-mediated cytotoxicity against an allogeneic osteosarcoma cell line. Sera from 14 of 16 sarcoma-bearing patients blocked cellular cytotoxicity compared to only four sera from 14 sarcoma-free patients. Three of the sarcoma-free patients with blocking sera developed recurrent cancer within one year following the assay. Only 1 of 8 sera from carcinoma-bearing patients inhibited cellular cytotoxicity against osteosarcoma cells, and only 2 of 11 sera from sarcoma-bearing patients which were 100% inhibitory against osteosarcoma cells were able to inhibit *in vitro* specific cytotoxicity against human lung carcinoma cells. Serum inhibition of sarcoma-specific cellular cytotoxicity antedated by as much as one year the development of metastatic sarcoma.

0931 DEPRESSED ALLOGRAFT IMMUNITY IN LEUKEMIA VIRUS INFECTED MICE.

(E.) Friedman, H. (Albert Einstein Med. Ctr., Philadelphia, Pa.), H. Melnick, L. Mills and W. S. Ceglowski. *Transplant Proc* 5(1):981-986, 1973.

The responsiveness of Friend leukemia virus (FLV)-infected Balb/c mice to allogenic skin grafts was studied. FLV-infected mice showed a marked depression of their ability to reject skin grafts from C3H or C57BL/6 donors, two strains differing at the H^2 locus. Delayed graft rejection time was maximum (25-50% longer than controls) when mice were infected with FLV ten days prior to grafting. No prolongation of donor graft survival occurred with any mouse infected with FLV after grafting. Prolongation of graft survival depended on the dose of FLV inoculated. Maximum prolongation occurred in mice infected with the largest dose of FLV which also produced the most rapid development of leukemia. *In vitro* macrophage migration-inhibition (MIF) tests used as an indicator of *in vivo* cell mediated immunity showed that spleen cells from mice infected with FLV 10-15 days prior to test grafting had a decreased ability to produce MIF. These results indicated that the allograft response was depressed in leukemic mice. Whether this was due to a direct effect of FLV infection on T lymphocytes or to secondary effects related to the disease process *per se* could not be determined.

0932 MAPPING AND MIGRATION OF LYMPHOCYTE SURFACE MACROMOLECULES.

(E.) Karnovsky, M. J. (Harvard Med. Sch., Boston, Mass.) and E. M. Unanue. *Fed Proc* 32(1):55-59, 1973.

The distribution of lymphocyte membrane receptor sites on treatment at 4 C with ligands, and their movement and redistribution on warming to 37 C has been studied. Bivalent anti-immunoglobulin and concanavalin A binding sites move rapidly to one pole of the cell and form a single large aggregate or cap which is frequently endocytosed. Antilymphocytic globulin binding sites are only capped on treatment with a double antibody (sandwich reaction). The H-2 binding sites cap only to a slight extent, even in a sandwich reaction. Freeze-etched preparations of cells treated with ferritin- or hemocyanin-labeled ligands show a correlation between the distribution of binding sites and their capacity to be moved by either a single or double antibody. Endocytosis does not necessarily follow on cap formation. This is related to the proximity of cross-linked sites to each other and to the cell membrane. In the case of immunoglobulin, cap formation is apparently accompanied by an exfoliation of the receptor from the cell membrane. Changes in intramembranous particles concomitant with capping of various entities have not been found. Metabolic inhibitors prevent capping, possibly by reducing cell motility. The relevance of the capping phenomenon to immunobiology and cell membrane organization is briefly discussed.

0933 FRACTIONATION OF PLASMA MEMBRANE-ASSOCIATED TUMOUR-SPECIFIC ANTIGEN FROM AN AMINOAZO DYE-INDUCED RAT HEPATOMA.

(E.) Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), J. R. Harris and M. R. Price. *Int J Cancer* 11(2): 385-397, 1973.

Tumor-specific antigens from an aminoazo dye-induced rat hepatoma were liberated in a soluble form following limited papain digestion of tumor cell membrane preparations. Fractionation of the soluble extract by DEAE-cellulose chromatography, gradient centrifugation and preparative electrophoresis yielded a major antigenic fraction, with an approximate molecular weight of 55,000, together with a range of other minor antigenic components of larger molecular weight. All preparations retained the capacity to inhibit the reaction of antibody in tumor-immune sera with the plasma membrane of viable hepatoma target cells in the indirect immunofluorescence test. It is considered that the defined antigen fractions isolated provide material suitable for analysing the nature of tumor antigen expression in chemical carcinogenesis and for evaluating the involvement of antigen-antibody interaction in tumor immunity.

0934 SEQUENTIAL STUDIES ON CELL-MEDIATED TUMOR IMMUNITY AND BLOCKING SERUM ACTIVITY IN TEN PATIENTS WITH MALIGNANT MELANOMA.

(E.) Hellström, I. (U. Washington Med. Sch., Seattle), G. A. Warner, K. E. Hellström and H. O. Sjogren. *Int J Cancer* 11(2):280-292, 1973.

Ten human patients with malignant melanoma were followed with respect to two *in vitro* parameters of tumor immunity, the ability of the patients' blood lymphocytes to destroy cultivated melanoma cells, and the ability of the patients' sera to block such destruction, and with respect to changes in the clinical status, such as increases or decreases in detectable tumor mass. Allogeneic and (to a much lesser extent) autochthonous melanoma cells were used as targets. The degree of lymphocyte-mediated tumor immunity was higher in patients with little or no residual tumor than in patients with large tumor loads, although the latter patients were also found to be reactive. Sera from patients with clinically detectable lanoma could block the cytotoxic lymphocyte fect, as could sera from patients who developed lanoma shortly after the serum harvest. Disappea- nce of blocking serum activity was seen to accom- ny clinical improvement. Sera from some patients thout clinically detectable tumor could potentiate e cytotoxic effect of lymphocytes from melanoma tients. The findings support the view that cell- diated tumor immunity and blocking serum activity, studied *in vitro*, are important correlates of e patients' immunological defense against their mors *in vivo*, and they suggest that monitoring these parameters may be prognostically useful. ey also suggest that procedures capable of im- oving cell-mediated tumor immunity without creasing blocking serum activity may be thera- utically beneficial.

35 COMMON AVIAN ONCORNAVIRUS-INDUCED TUMOR ANTIGENS IN DIFFERENT SPECIES AS REVEALED IMMUNO-FERRITIN TECHNIQUES. *(E.)* Gelderblom, (Robert Koch-Inst., Dept. Virol., Berlin, W. rmany) and H. Bauer. *Int J Cancer* 11(2):466- 2, 1973.

indirect immuno-ferritin technique was employed study whether avian tumor virus (ATV)-induced mor-specific surface antigens (TSSA) in different ecies were immunologically related. Two different n-producer, gs-negative cell lines derived from us sarcoma virus-induced tumors were investigated. en these lines were incubated in anti-avian rritin-tagged sarcoma virus antiserum prepared ainst different subgroups and were observed under e electron microscope, uniform labeling was en along the cell membranes. These findings ggested that both cell lines contained a common oup-specific virus-associated surface antigen though they were transformed by two different ral subgroups. These respective antigens also peared to cross-antigenic to the TSSA demonstrated eviously on ATV-transformed chick embryo cells. e common antigens do not represent any known ructural viral antigen.

36 A SIMPLIFIED ISOTOPIC ANTIGLOBULIN ASSAY: DETECTION OF TUMOR-CELL ANTIGENS. *.)* Burdick, J. T. (Natl. Cancer Inst., Bethesda, l.), A. M. Cohen and S. A. Wells, Jr. *J Natl ncer Inst* 50:285-289, 1973.

rapid, sensitive isotopic antiglobulin micro- say technique using tissue culture monolayers th a simplified washing procedure is described for e detection of tumor-specific cell-surface tigens. Briefly, the procedure involves washing target cell monolayers with balanced salt solu- on, incubation with either immune serum (IS) om isologous tumor-bearing mice or with isologous ntrol serum (CS), washing, and final incubation ith ^{125}I-labeled 7S goat antimouse γ-globulin rum. IS from several C57BL/6N male mice immunized repeated s.c. injection of 3-methylcholanthrene- duced tumor cells was absorbed to target tumor cells to a much greater extent than CS. The reaction was specific since absorption of IS was the same or less than CS when tested against normal mouse muscle cells, and since IS activity could be absorbed by tumor cells only. Maximum reactivity was obtained at dilutions of 1:32 or 1:64 with the difference in reactivity between CS and IS clearly demonstrable at 1:128.

0937 THE SPECIFICITY OF CARCINO-FOETAL ANTIGENS OF THE HUMAN DIGESTIVE-TRACT TUMORS. *(E.)* Martin, F. (Fac. Med., Dijon, France), M. S. Martin, M. Bordes and C. Bourgeaux. *Eur J Cancer* 8:315-321, 1972.

A specific anti-human carcinoembryonic antigen (CEA) serum was prepared in rabbits by inoculation with human colonic adenocarcinoma material. This serum reacted with a carcino-fetal antigen when tested in immunodiffusion studies with saline extracts of colonic tumors. The antigen could not be detected by reacting the anti-CEA serum with saline extracts of noncancerous human colon material. However, small but significant amounts of the carcino-fetal antigen were detected in perchloric extracts of normal colon by immunodiffusion (Ouchterlony test), precipitin-inhibition tests, immunofluorescence tests and radioimmunoassay. The carcino-fetal antigen is not, therefore, strictly cancer-specific; however, quantitative differences in CEA concentrations in normal and cancerous tissues indicate that the antigen is a useful tool for detection of digestive tract cancers. It was noted that α-fetoprotein of hepatoma and fetal sulphoglycoprotein of gastric carcinoma, two other human carcinofetal antigens, have also been detected in noncancerous tissue.

0938 SELECTIVE SENSITIVITY OF THYMUS-DERIVED CELLS TO PHYTOHAEMAGGLUTININ *IN VITRO*. *(E.)* Medzihradsky, J. (Cancer Res. Inst., Bratislava, Czechoslovakia), O. Babusikova and E. Konikova. *Neoplasma* 19(5):489-498, 1972.

Lymphoid cells of various origin were tested on their survival in tissue culture with, or without phytohemagglutinin (PHA) using standard dose of the drug. The number of viable cells which can be recovered from the cultures after 24-hour incubation was estimated. Lymph node cells harvested from adult thymectomized, lethally irradiated, and bone marrow-reconstituted rats showed higher survival in PHA cultures than did their sham-operated, irradiated, and marrow-injected control littermates. Bone marrow cells from normal rats were found to survive equally well when tested with or without PHA. The survival of thoracic duct cells or thymocytes from the same animals was, on the other hand, lower with PHA than without the drug. Lymph node cells altered by the regional graft-versus-host reaction which induce, as indicated from the high *in vitro* tritiated thymidine uptake, activation of proliferating blast cells, survived better with PHA than did the control node cells from untreated rats. Lymph node cells, thoracic duct cells, or peritoneal lymphocytes from rats bearing grafted syngeneic or methylcholanthrene-induced primary tumors were furthermore investigated. As compared with the tumor free controls, in 24-hour PHA cultures more viable cells can be recovered when cells derived from tumor-bearing animals were tested. The results are discussed in terms of the possible heterogeneity of the T-cells in the peripheral blood.

0939 THE EFFECT OF IMMUNOSUPPRESSION ON ONCOGENESIS BY MURINE SARCOMA VIRUS. *(E.)* Zisblatt, M. (Albert Einstein Coll. Med., Bronx, N.Y.) and F. Lilly. *Proc Soc Exp Biol Med* 141(3):1036-1040, 1972.

The role of immunocompetence in the resistance of CBA/Wh and A/HeJ mice to oncogenesis by s.c. inoculated murine sarcoma virus (MSV) was studied. Mice immunosuppressed by either i.p. inoculation of rabbit anti-mouse lymphocyte serum (ALS) or by whole body x-irradiation showed an increased incidence of MSV-induced tumors compared with normal controls. Also, both the mean and range of the latency periods until tumor appearance were usually lower in immunosuppressed than in immunocompetent mice. The length of survival of MSV-infected immunosuppressed mice was shorter than that of control mice and was inversely related to the virus inoculation dose. Resistance to tumor induction in immunosuppressed mice by MSV became apparent 3 wk after X-irradiation and was evidenced by a decreased tumor incidence and prolonged latency period. These resistant mice also showed an increased resistance to tumor development and to progressive tumor growth.

0940 ENZYMATIC SYNTHESIS OF DNA COMPLEMENTARY TO PURIFIED 14S MESSENGER RNA OF IMMUNOGLOBULIN LIGHT CHAIN. *(E.)* Diggelmann, H. (Swiss Inst. Exp. Cancer Res., Lausanne), C. H. Faust, Jr. and B. Mach. *Proc Natl Acad Sci USA* 70(3):693-696, 1973.

The poly A-containing 14S mRNA purified by successive sucrose density gradient centrifugation of mouse plasmacytoma (MOPC 41) immunoglobulin light chain was used to synthesize complementary DNA in an avian myeloblastosis virus RNA-dependent DNA polymerase system. The synthesis of DNA was completely dependent on addition of DNA and of oligo(dT) primer. Incorporation of ^{3}H-labeled deoxynucleotide into DNA also depended on the size of the primer, with smaller ones being more efficient, and on the quantity of primer used, an excess being slightly inhibitory. Analysis of the reaction product by $CsSO_4$ equilibrium density centrifugation showed that it was indeed DNA. Incorporation of label was not due to transcription of only the poly A region of the RNA template. Hybridization experiments showed specific annealing of the DNA product to 14S light chain mRNA template. Significant DNA synthesis was also found to occur when oligo(dG) was used as primer, implicating a second initiation point on the 14S mRNA molecule.

0941 DEMONSTRATION OF A CELL-SURFACE ANTIGEN ASSOCIATED WITH MURINE SARCOMA VIRUS BY IMMUNOELECTRON MICROSCOPY. *(E.)* Aoki, T. (Natl. Cancer Inst., Bethesda, Md.), J. R. Stephenson and S. A. Aaronson. *Proc Natl Acad Sci USA* 70(3): 742-746, 1973.

Various mouse cell lines transformed by several strains of murine sarcoma virus (MSV) were examined by immunoelectron microscopy for the presence of new virus-associated cell-surface antigen. The results showed the existence of a common antigen on the surface of nonproducer cells transformed by the Kirsten and Moloney strains of MSV. The antigen also showed cross reaction with MSV-transformed cell lines of rat and mouse origin. The antigen was distinct from previously described antigens found on the surface of cells infected with murine leukemia virus (MuLV), on the MuLV envelope, on the surface of spontaneously transformed mouse cell lines, or on the surface of mouse cells transformed by x-irradiation.

0942 TUMOR-SPECIFIC CELLULAR CYTOTOXICITY TO HUMAN SARCOMAS: EVIDENCE FOR A CELL-MEDIATED HOST IMMUNE RESPONSE TO A COMMON SARCOMA CELL-SURFACE ANTIGEN. *(E.)* Cohen, A. M (Natl. Cancer Inst., Bethesda, Md.), A. S. Ketcham and D. L. Morton. *J Natl Cancer Inst* 50(3):585-589, 1973.

Sarcoma-specific cellular cytotoxicity was evaluated in patients with skeletal and soft-tissue sarcomas. Peripheral blood lymphocytes from 14 of 18 sarcoma patients were cytotoxic to an allogeneic osteosarcoma cell line, but not to fibroblasts from the same patient. Lymphocytes from four patients after pelvic exenteration and from three melanoma patients were nonreactive. All pelvic surgery patients had received multiple blood transfusions. Patients with fibrosarcoma, liposarcoma, mesenchymoma, undifferentiated sarcoma, small cell sarcoma, and osteosarcoma had lymphocytes cytotoxic to osteosarcoma cells. The data support the concept of host transplantation resistance to a common human sarcoma antigen.

0943 CARCINOEMBRYONIC ANTIGEN IN EXTRACTS OF PULMONARY CANCERS. *(E.)* Sizaret, P. (Int. Agency Res. Cancer, Lyon, France) and F. Martin. *J Natl Cancer Inst* 50(3):807-810, 1973.

Carcinoembryonic antigen (CEA) was detected by direct immunodiffusion in aqueous extracts of 6 of 19 human lung carcinomas and in perchloric extracts of 4 of 5 apparently negative specimens. The presence of CEA in lung carcinomas was further supported by immunoelectrophoresis, precipitin-inhibition reaction, and radioimmunoassay. These results show that CEA is not restricted to fetal gut and digestive tract tumors.

0944 SOME RECENT STUDIES ON CELLULAR IMMUNITY TO HUMAN MELANOMAS. *(E.)* Hellström, I. (U. Washington Med. Sch., Seattle) and K. E.

lström. *Fed Proc* 32(2):156-159, 1973.

lifferences in cell-mediated immunity between ients with growing melanoma and patients clinily tumor free following therapy were demonstrated ng high lymphocyte doses in *in vitro* cytotoxicity s on melanoma target cells. However, when low hocyte doses were used, it was evident that noma patients were less reactive than tumor- patients. *In vivo* observations indicated that noma patients and tumor-free patients differed locking serum activity, and that this activity sometimes present before a tumor becomes cliniy manifest. Sera from melanoma-free patients gated the blocking effect of sera from cancer ents when added to it. Melanoma cells appeared ave more than one tumor-associated antigen, since hocytes from some patients were more reactive ome lines of cultivated melanoma cells than on rs while lymphocytes from other patients showed verse pattern. These findings suggest that hutumor antigens are targets for host defense nst neoplasms such as melanoma.

APPEARANCE IN TRYPSINIZED NORMAL CELLS OF REACTIVITY WITH ANTIBODY PRESUMABLY IFIC FOR MALIGNANT CELLS.

(E.) Tarro, G. . Clin. Inst. I, U. Naples, Italy). *Proc Nat Sci USA* 70(2):325-327, 1973.

al human disploid cells WI-38 (from embryonic) and MRC 5 (from fetal lung) were treated with sin for 2.5 min and reacted in complement-fixation s with anti-HeLa G immunoglobulin against malignant HeLa cells. Both normal cell lines showed ivity with the anti-HeLa immunoglobulin. Abion of the globulin with untrypsinized WI-38 s removed all reactivity, not only for trypsinized s but also for malignant human cells. That this ption effect was specific for WI-38 cells was ated by the fact that absorption of globulin rabbit and guinea pig cells did not affect the ivity of the globulin. It was thought that inization increased the number of combining s for the anti-HeLa antibody on the cells.

MEMBRANE FRACTIONS FROM RAT HEPATOMA. III. IMMUNOCHEMICAL CHARACTERIZATION OF DETERGENT-LE MEMBRANE PHOSPHATASES, ELECTRON TRANSPORT S AND CATALASE.

(E.) Raftell, M. (Dept. Immunol., ockholm, Sweden) and F. Blomberg. *Biochim ys Acta* 291(2):442-453, 1972.

sma membrane fraction (tP) and rough and smooth crosomal fractions (tR and tSa, resp.) were obd from cells of a rat D23 hepatoma originally ed by 4-dimethylaminoazobenzene. Fractions were ed to antisera against the various fractions mmunodiffusion tests were performed; membrane ions were stained for phosphatase, hydroxylase atalase. 5'-Nucleotidase (NMPase) was absent tR and tSa but tP gave a positive reaction with Pase active antigen; this antigen appeared to fferent from NMPase active antigens in liver. Fraction tP also contained nucleoside tri- and diphosphatase. Uridine diphosphatase active antigen characteristic for liver microsomes was found only in tR fractions, while both tR and tSa contained an additional antigen with similar activity. No D23 fraction showed any hydroxylating activity. However, some components of liver electron transport chains, NADPH-cytochrome *c* reductase, NADH- and NADPH-neotetrazolium (NT)-reductase and cytochrome b_5 were found in tR. All tumor fractions contained one of the two NADH-NT-reductase antigens common to liver microsomes and plasma membranes. One catalase active antigen was seen in all liver and tumor fractions.

0947 PURIFICATION OF HODGKIN'S DISEASE TUMOR-ASSOCIATED ANTIGENS.

(E.) Katz, D. H. (Harvard Med. Sch., Boston, Mass.), S. E. Order, M. Graves and B. Benacerraf. *Proc Nat Acad Sci USA* 70(2):396-400, 1973.

Tumor nodules were removed from spleens of Hodgkin's disease patients, homogenized and centrifuged. Supernatants were purified to yield Hodgkin's tumor-associated antigens. On Sephadex G-200 chromatography, antigenic material gave an elution pattern with three protein peaks of which only peak I contained antigenic activity. Hodgkin's tumor material showed an elution pattern similar to that of normal fetal liver. Material from peak I of Hodgkin's antigenic material was reacted with hyperimmune rabbit antisera and subjected to immunoelectrophoresis and Ouchterlony analysis. F and S antigens, associated with Hodgkin's disease, were detected in peak I material, as was a lymphcyte-associated antigen which, however, gave only a weak reaction. The tissue distribution pattern of the Hodgkin's tumor-associated antigens suggest that both originate in lymphoid tissue; the S antigen may be a dedifferentiation antigen expressed in immature lymphocytes, while the F antigen may be a product of reactive lymphocytes.

0948 CYTOTOXICITY OF MURINE PERITONEAL MACROPHAGES IN TUMOR ALLOGRAFT IMMUNITY.

(E.) den Otter, W. (Chester Beatty Res. Inst., Sutton, Surrey, England), R. Evans and P. Alexander. *Transplantation* 14(2):220-226, 1972.

The cytoxicity for the DBA/2 mouse lymphoma, SL2, of peritoneal macrophages taken from C57BL mice sensitized by a single i.p. injection of SL2 cells was studied *in vitro*. The growth of SL2 cells in macrophage monolayer cultures was selectively inhibited by sensitized but not by normal C57BL peritoneal macrophages. There was no evidence of phagocytosis of intact lymphoma cells, although ingested debris was observed. Sensitized macrophage monolayers maintained for one week before challange were as strongly cytotoxic as fresh monolayers. The activity was not due to contamination of the macrophage cultures by peritoneal lymphocytes. Contact between immune macrophages and target cells was necessary for cytotoxic action which was apparent within 0.5 to 12 hr. Exposure of sensitized macrophages to 0.1% trypsin solution for 2 hr resulted in complete loss of cytotoxicity. Exposure of ^{51}Cr-labeled SL2 lymphoma cells to sensitized macrophages resulted in loss of 50% of their radioactivity over a 9 hr period.

0949 SYNTHESIS, BIOCHEMISTRY AND DYNAMICS OF CELL SURFACE IMMUNOGIOBULIN ON LYMPPHOCYTES. *(E.)* Uhr, J. (New York U. Sch. Med., N.Y.) and E. S. Vitetta. *Fed Proc* 32(1):35-40, 1973.

The synthesis and intracellular transport of cell surface immunoglobulin were studied in pulse labeling experiments with Daudi cells, a line derived from a patient with Burkitt's lymphoma. In related experiments, radioiodination techniques were applied to characterize surface cell immunoglobulin of lymphocytes from BALB/c spleen cells and to determine the fate of surface immunoglobulin from splenocytes of A/J mice. The results indicate that μ- and light chains are synthesized on membrane-bound polyribosomes and the assembled monomer is transported to the Golgi complex. Binding occurs to the interior of the Golgi vesicle and the majority of sugar residues are then incorporated. Transport of immunoglobulin to the plasma membrane occurs in post-Golgi vesicles. The vesicle merges with the plasma membrane and membrane excision occurs, resulting in immediate release of "free" immunoglobulin and exteriorization of membrane-bound immunoglobulin, predominantly monomeric IgM. Subsequently cell surface immunoglobulin is released possibly attached to a fragment of the membrane. The finding that the small lymphocyte secrets 8S IgM, including cell surface IgM, suggests that this immunoglobulin functions to meet antigen and bring it to an immunogenic compartment.

0950 VSV PSEUDOTYPE PRODUCED IN CELL LINE DERIVED FROM HUMAN MAMMARY CARCINOMA. *(E.)* Zavada, J. (Slovak Acad. Sci., Bratislava, Czechoslovakia), Z. Zavadova, A. Malir and A. Kocent. *Nature* [*New Biol*] 240(99):124-125, 1972.

Wild-type vesicular stomatitis virus (VSV) was grown in three human cell lines: Lep (diploid human embryo lung), HeLa, and MaTu (from mammary carcinoma). VSV grew in all these cell cultures but only in MaTu did it produce a pseudotype from (VSV(MaTu)). The pseudotype was resistant to anti-VSV serum and had a restricted host range compared with VSV grown on Lep and HeLa cells. Pseudotype VSV was also produced with MaTu cells using *tl*-17, a thermolabile VSV mutant with altered neutralization antigens. These results indicate that MaTu cells may contain a viral antigen detectable by its appearance in pseudotype VSV. Attempts to demonstrate an oncornavirus in MaTu cells are continuing.

0951 BLOCKING OF LYMPHOCYTE-MEDIATED CYTOTOXICITY FOR RAT HEPATOMA CELLS BY TUMOUR-SPECIFIC ANTIGEN-ANTIBODY COMPLEXES. *(E.)* Baldwin. R. W. (Cancer Res. Campaign Labs., U. Nottingham, England), M. R. Price and R. A. Robins. *Nature New Biol* 238(84):185-186, 1972.

Transplanted rat D23 hepatoma cells were exposed to serum taken from rats after surgical removal of hepatomas or to post excision serum incubated with solubilized hepatoma D23-specific antigen. Serum alone did not block lymph node cell (LNC)-mediated inhibition of D23 cell growth, but serum mixed with D23 antigen acquired the capacity to block LNC cytotoxicity. The optimal effect (95.4% blocking) was obtained using 0.8 mg antigen/ml serum. Addition of increasing amounts of antigen to serum reduced blocking activity. Similarly, addition of excess antigen to tumor-bearer serum abolished its blocking activity. These experiments support the concept that antigen-antibody complexes are involved in the abrogation of lymphocyte toxicity for tumor cells.

0952 INCREASED PROPORTION OF COMPLEMENT-RECEPTOR LYMPHOCYTES IN THE PERIPHERAL BLOOD OF PATIENTS WITH CHRONIC LYMPHOCYTIC LEUKEMIA. *(E.)* Pincus, S. (New York U. Sch. Med., N.Y.), C. Bianco and V. Nussenzweig. *Blood* 40(3):303-310, 1972.

Lymphocytes from normal adults and from chronic lymphocytic leukemia (CLL) patients were tested to determine the proportion of complement-receptor lymphocytes (CRL) in these subjects. Lymphocytes for testing were prepared by a method that achieved 70% recovery of lymphocytes from peripheral blood. Lymphocytes were incubated with antibody and mouse complement (EAC) and rosettes of EAC-CRL were counted. Blood, lymph node and tonsil lymphocytes from normals showed 7.1 ± 3.8%, 31.0 ± 6.9% and 59.6 ± 13.2% CRL, resp. The percentage of CRL in blood of CLL patients was 50.7 ± 25.0%. In blood of two patients virtually all lymphoid cells formed rosettes with EAC.

0953 EFFECT OF STIMULATION OF THE HOST DEFENSE SYSTEM BY COENZYME Q_{10} ON DIBENZPYRENE-INDUCED TUMORS AND INFECTION WITH FRIEND LEUKEMIA VIRUS IN MICE. *(E.)* Bliznakov, E. G. (New England Inst., Ridgefield, Conn.). *Proc Natl Acad Sci USA* 70(2):390-394, 1973.

$C_{57}Bl_6$ mice were given s.c. injections of 0.5 ml dibenzpyrene 7, 14 or 21 days before i.v. administration of a 500 μg/ml concentration of coenzyme Q_{10}. The percentage of mice with tumors induced by dibenzpyrene was reduced by coenzyme Q_{10}. In addition, numbers of mice surviving dibenzpyrene were increased and tumor sizes among those developing tumors were reduced. These effects of coenzyme Q_{10} were dose-dependent. In related experiments, coenzyme Q_{10} was administered (250 μg/ml, i.v.) 7, 14 or 21 days after infection of Swiss Webster mice with Friend leukemia virus (FLV). Coenzyme Q_{10} decreased FLV-induced splenomegaly and hepatomegaly and increased the number of surviving infected mice. These effects were also dose-dependent.

54 MEMBRANE FRACTIONS FROM RAT HEPATOMA I. ISOLATION AND CHARACTERIZATION. *(E.)*
ftell, M. (Dept. Immunol., U. Stockholm, eden) and F. Blomberg. *Biochim Biophys Acta* 1(2):421-430, 1973.

lls of the 4-dimethylaminoazobenzene-induced t hepatoma D_{23} were broken down into three crosomal subfractions: "rough" (tR) and mooth" (tSa and tSb). Plasma membranes raction tP) were also isolated. Under the ectron microscope the tR fraction showed a mbrane made up of ribosome-covered tubules, ile vesicles of the tSa and tSb fractions re smooth. Membranes of the various fractions re further characterized by observing the tivity of the plasma membrane marker 5'- cleotidase (NMPase) with AMP as substrate. b and tP had the highest levels of NMPase tivity; however NMPase was always lower in mor cell fractions than in liver cell fractions. glucose-6-phosphatase or succinate-cytochrome reductase was detected in any membrane fraction. Pase, UDPase and ATPase were elevated tenfold the tSb fraction compared with other tumor d liver fractions. Freezing decreased ADPase, Pase and ATPase in all membrane fractions.

55 *IN VITRO* ACTIVATION OF CELLULAR IMMUNE RESPONSE TO GROSS VIRUS-INDUCED LYMPHOMA.
.) Ortiz de Landazuri, M. (Natl. Cancer Inst., thesda, Md.) and R. B. Herberman. *J Exp Med* 6(5):969-983, 1972.

U rats were immunized against the Gross virus-duced WFU lymphoma and lymphoid cells were rvested at intervals after immunization. Cell diated cytotoxicity of immune lymphocytes was sted on ^{51}Cr-labeled target lymphoma cells. leen cells harvested 40 days after immunization re never cytotoxic unless incubated *in vitro* r 24 hr at 37 C before exposure to target cells. leen cells harvested ten days after immunization re cytotoxic without preincubation. Lymphoid lls other than spleen cells were cytotoxic ter preincubation, with the exception of bone arrow cells. Reinoculation of tumor cells to immunized rats increased the cytotoxicity spleen cells harvested after reinoculation. periments designed to elucidate the mechanism *in vitro* activation of cytotoxicity in leen cells showed that activation was tempera- re-dependent; cells incubated at 4 C developed cytotoxicity. Immune serum against the mphoma sometimes suppressed cytotoxicity of leen cells. Passage of immune cells through nylon column before preincubation prevented totoxic activation; this suggested that cell rface factors were involved in the activation spleen cell cytotoxicity.

56 *IN VITRO* CYTOTOXICITY BY A NONTHYMUS-PROCESSED LYMPHOCYTE POPULATION WITH ECIFICITY FOR A VIRALLY DETERMINED TUMOR CELL SURFACE ANTIGEN. *(E).* Lamon, E. W. (Karolinska Inst., Stockholm, Sweden), H. M. Skurzak, E. Klein. *J Exp Med* 136(5):1072-1079, 1972.

BALB/c mice were immunized against Moloney sarcoma virus (MSV) and lymphocytes from immune mice were collected from spleens and lymph nodes. Collected lymphocytes were separated into thymus-processed cells (T-cells) and nonthymus-processed cells (i.e., B-cells) by treating them with an anti-T-cell serum produced in rabbits against mouse thymus cells. The cytotoxicity of T-cell-deficient lymphocyte populations was tested on Ha2 cells from an MSV-induced mouse tumor, on D56 cells from MSV-transformed mouse cells, and on D56-M cells. Ha2 cells produced MSV and Moloney leukemia virus (MLV) and possessed the MLV-determined surface antigen; D56 cells contained the MSV genome but did not possess a distinctive virus-associated surface antigen. D56-M cells were D56 cells superinfected with MLV; in contrast to D56 cells, they possessed the MLV-associated surface antigen. On these targets, the cytotoxicity of non-T lymphocytes was specific for the MLV-determined cell surface antigen. MSV-immune non-T lymphocytes significantly reduced the number of surviving Ha2 and D56 cells compared with non-T lymphocytes from unimmunized mice. Also, immune lymphocytes killed only those cells which possessed the MLV-associated surface antigen (Ha2 and D56-M) and spared MLV antigen-free D56 cells.

0957 SOLUBLE MEMBRANE ANTIGENS OF LIP AND CERVICAL CARCINOMAS: REACTIVITY WITH ANTIBODY TO HERPESVIRUS NONVIRION ANTIGENS. *(E.)*
Hollinshead, A. C. (George Washington U. Med. Ctr., Washington, D.C.) and G. Tarro. *Science* 179(4074):698-700, 1973.

Soluble membrane antigens separated from lip and cervical carcinomas, but not antigens from normal vaginal tissues or intestinal carcinomas, reacted in complement-fixation tests with guinea pig antiserum to herpes nonvirion antigens. The reaction was highly specific since neither guinea pig serum obtained before hyperimmunization with the herpesvirus nonvirion antigen nor antiserum from guinea pigs immunized with comparable uninfected cell extracts reacted with these tumor soluble antigens. Since the soluble membrane antigens could be specific markers for the presence of viral genome in tumor cells, the findings could support an etiological role of herpesvirus in selected human malignancies.

0958 THE EFFECT OF SPLENECTOMY ON FIBROSARCOMA "METASTASES" IN LUNGS OF MICE. *(E.)*
Milas, L. (Rudjer Boskovic Inst. Central Inst. Tumors Allied Dis., Zagreb, Yugoslavia) and H. Mujagic. *Int J Cancer* 11(1):186-190, 1973.

Cells of a methylcholanthrene-induced fibrosarcoma, injected i.v. into syngeneic C57Bl mice, generated more pulmonary metastases in splenectomized mice than in sham-operated mice. However, immunization by three consecutive injections of heavily irradiated tumor cells prior to injection of viable tumor cells induced significantly higher resistance in splenectomized than nonsplenectomized mice. Thus, 5 X 10^1 tumor cells generated 7.4 ± 1.1 tumor nodules in the lungs of splenectomized and immunized mice compared with 16.4 ± 1.5 nodules in immunized sham-operated mice. The effect of splenectomy was specific (thymectomy had no effect on the number of pulmonary metastases) and was greater if fewer tumor cells were injected.

0959 SEROLOGICAL STUDIES IN A STUDENT POPULATION PRONE TO INFECTION WITH HUMAN PAPILLOMA VIRUS.

(E.) Cubie, H. A. (Dept. Bacteriol., U. Edinburgh, Scotland). *J Hyg (Camb)* 70(4):677-690, 1972.

Blood sera from 467 students were tested for circulating antibodies to human wart virus using the immunoelectroosmophoresis method. Students with a past history of plantar warts or plantar and other warts were virus-antibody-positive in 44.3% and 50.0% of cases, resp., while those with no wart history were serum positive in 18.1% of cases. The virus antibody was apparently of the IgG class. Most subjects evidently did not develop anti-wart virus antibody until the virus infection had been present for a few months. Antibodies persisted for up to nine years after infection. There were a few instances of multiple infection; these demonstrated that reinfection was possible.

0960 NORMAL DIFFERENTIATION OF MYELOID LEUKAEMIC CELLS INDUCED BY A DIFFERENTIATION-INDUCING PROTEIN.

(E.) Fibach, E. (Weizman Inst. Sci., Rehovot, Israel), T. Landau and L. Sachs. *Nature [New Biol]* 237(77):276-278, 1972.

Cells of a mouse myeloid leukemia were treated with the differentiation-inducing protein MGI. MGI induced normal differentiation of leukemic cells to mature macrophages and granulocytes. Induction of differentiation did not require the addition of a co-factor since the leukemic cells produced a co-factor. Addition of adenine, cyclic AMP, dibutyryl cyclic AMP, uridine, guanine or thymine did not increase the differentiation or the number of colonies formed by MGI-treated leukemic cells.

0961 THE RESTRICTION OF THE RELEASE OF MIGRATION INHIBITORY FACTOR TO CERTAIN PHASES OF THE CELL CYCLE OF MURINE LEUKEMIA.

(E.) Thomas, D. B. (Middlesex Hosp. Med. Sch., London, England). *Eur J Immunol* 2(5):478-481, 1972.

Cell cultures of the murine leukemia line L1210 and of the murine mastocytoma line P815Y were synchronized by the double thymidine block technique, and the production by these cells of a migration inhibitory factor (MIF) was studied by observing the migration of guinea pig macrophages in L1210 and P815Y cultures. Both cell lines inhibited macrophage migration by 40-60%. The inhibitory material was eluted as a broad peak on Sephadex G-100, with a Kav of zero to 0.76, indicating considerable heterogeneity. Release of the inhibitory factor by L1210 and P815Y cells occurred in the G_1 and early S phases of the cell cycle.

0962 THYMUS-DERIVED ROSETTE-FORMING CELLS IN VARIOUS HUMAN DISEASE STATES: CANCER, LYMPHOMA, BACTERIAL AND VIRAL INFECTIONS, AND OTHER DISEASES.

(E.) Wybran, J. (Sch. Med., U. California, San Francisco) and H. H. Fudenberg. *J Clin Invest* 52(5):1026-1032, 1973.

Lymphocytes that bind *in vitro* to sheep erythocytes in a rosette formation are thymus-derived. A modified technique that does not detect the total number of rosette-forming cells (RFC) was used to study normal subjects and various disease states. RFC as detected by this technique represent the population(s) of T cells involved in the active aspects of cellular immunity. Of 100 healthy subjects, 95 had more than 15% RFC (mean 28.4 ± 6.5%). One hundred-four patients with solid tumors were studied, who were classified according to clinical status and stage of therapy. Of 19 newly diagnosed patients, 13 had less than 15% RFC. Of 44 untreated patients, undergoing relapse, 32 had less than 15% RFC. In both categories, patients with metastases had fewer RFC than patients with localized disease. Eleven patients were studied 2 wk after cessation of therapy; four of them showed less than 15% RFC. Only one of 30 patients in remission had less than 15% RFC. In seven patients followed for various periods of time, the numbers of RFC correlated generally with clinical status. Eleven patients with chronic lymphatic leukemia had very low percentages of RFC. Twenty-one of 21 patients with symptoms of viral upper respiratory diseases had less than 15% RFC. RFC returned to normal values between five days and 7 wk after disappearance of clinical symptoms. Twenty patients with bacterial infections had normal numbers of RFC. Of 25 patients with miscellaneous nonimmunologically related disease two had low numbers of RFC. It is suggested that the percentage of RFC may be valuable in evaluating not only immunological defenses but also the status of patients with solid tumors, lymphomas, viral diseases and, perhaps, bacterial infections.

0963 SEROEPIDEMIOLOGIC STUDIES OF HERPESVIRUS TYPE 2 AND CARCINOMA OF THE CERVIX. III. HOUSTON, TEXAS.

(E.) Adam, E. (Baylor Coll. Med., Houston, Texas), R. H. Kaufman, J. L. Melnick, A. H. Levy and W. E. Rawls. *Am J Epidemiol* 96(6):427-442, 1972.

Neutralizing antibody activity to herpesvirus type 1 and type 2 was quantified in sera from 85 women with cervical cancer and 367 control women. The

an titers of the antibody activity to herpesvirus
pe 2 were higher among women with cancer than
iong control women. Titers to type 2 virus in-
eased with age up to the fourth decade of life
iong control women but no age dependency was ob-
rved among women with cancer. These observations
iggest that a larger percentage of women with
rvical cancer have higher type 2 antibody titers
an control women but the antibodies were acquired
irlier in life. To evaluate the relation between
pe 2 antibody activity and attributes associated
th an increased risk of developing cervical cancer,
II/I index threshold was chosen at the level of
ie maximal case-control differences. The distri-
ition of II/I values exceeding the threshold levels
iong cases and controls was examined according to
e at first pregnancy, age at first marriage,
imber of live births, number of marriages and
imber of sex partners. The occurrence of II/I
ilues exceeding the threshold value among control
men with attributes associated with an increased
sk of developing cancer was greater than among
men without these attributes; however, significan-
y fewer of these women had positive values than
men with cancer. In addition, significantly
re cases had positive values than pair matched
ntrols. These findings suggest that the associa-
on between herpesvirus type 2 and cervical cancer
not simply one of covariability of sexual promis-
iousness.

64 SOME CHARACTERISTICS OF THE A ANTIGEN IN MAREK'S DISEASE VIRUS-INFECTED CELL LTURES.

(E.) Settnes, O. P. (U. Copenhagen,
nmark). *Acta Pathol Microbiol Scand* [*B*] 80(6):
7-822, 1972.

e A antigen in Marek's Disease Virus infected cell
ltures was found to be stable at 37 C or lower
r long periods, to be rapidly inactivated at
mperatures at 75 C to 100 C, unaffected by ether
chloroform, tolerate pH 1.7 for 4½ hr, but become
activated by pH 12.1 in 4½ hr. Trypsin inactivates
e antigen activity in 40-60 minutes at 37 C and
remains in the supernatant after centrifugation
1000,000 x G for 120 minutes. A antigen prepara-
ons were found inhibitory to the replication of
V in cell cultures and to VSV plaque formation
CK cultures, but not in L cells. The possibility
at the A antigen is an interferon-associated
otein is discussed.

65 CORRELATION BETWEEN CHANGES IN ANTITUMOR IMMUNE PARAMETERS AND TUMOR GROWTH *IN VIVO* RATS.

(E.) Bansal, S. C. (Fred Hutchinson Cancer
s. Ctr., Seattle, Washington) and H. O. Sjögren.
d Proc 32(2):165-172, 1973.

perimental tumors in animals and spontaneous
nors in humans exhibit weak antigenic responses
the host of origin. Inquiry was made into
veral facets of these immune responses at dif-
rent time points during tumor growth in primary
d isografted polyoma tumor-bearing W/Fu rats. The
incipal findings suggest an early appearance and maintenance of strong cellular immunity, but when the host is bearing a widespread tumor, there is a significant depression of cellular immunity. No change was found in the number of circulating lymphocytes during the progressive growth of tumor in the host, but there was a significant increase in number of circulating lymphocytes per milliliter in tumor-bearing rats as compared with normal controls. It has been demonstrated that the serum of tumor-bearing animals and humans can block the specific cytotoxic effect of lymphocytes on target cells *in vitro*. The present studies indicate an early appearance of blocking activity after tumor isograft, 2-3 days before palpable tumor nodule. This blocking activity is not only maintained but also increases with the progressive growth of the tumor; however, it rapidly disappears after tumor excision. The present model provides strong evidence that blocking antibodies are important in tumor growth and that, by means of unblocking serum and splenectomy, the blocking activity can be counteracted in the serum of rats bearing established primary polyoma tumors and thereby tumor growth is inhibited.

0966 *IN VIVO* AND *IN VITRO* ASSAYS OF CELLULAR IMMUNITY TO HUMAN TUMOR ANTIGENS.

(E.) Herberman, R. B. (Natl. Cancer Inst., Bethesda, Md.). *Fed Proc* 32(2):160-164, 1973.

Skin tests for delayed hypersensitivity and a lymphocyte cytotoxicity assay have been used to study the cellular immune response of patients to tumor-associated antigens. In Burkitt's tumor and in acute leukemia, the skin tests have detected antigens that appear to be tumor specific. Reactivity of patients has correlated well with the clinical course of the disease. In intestinal cancer, a carcinoembryonic antigen has been found that is distinct from the carcinoembryonic antigen of Gold. In malignant melanoma and in carcinoma of the breast, skin reactions have been elicited by extracts of normal tissues as well as by tumor preparations. The lymphocyte cytotoxicity assay has been useful in studying patients with leukemia and lymphoma. Patients have been found to react against autologous tumor cells but not against remission or normal cells. No positive reactions have been seen thus far in Hodgkin's disease. Comparative tests of skin with *in vitro* assays were performed in patients with acute leukemia. No correlation was found between the results of the different tests. The possible explanations for this and the limitations in evaluating the results of assays of cellular immunity are discussed.

0967 CELL-MEDIATED IMMUNE MECHANISMS OF TUMOR CELL DESTRUCTION.

(E.) Perlmann, P. (Wenner-Gren Inst., U. Stockholm, Sweden), C. O'Toole and B. Unsgaard. *Fed Proc* 32(2):153-155, 1973.

The specific destruction *in vitro* of various target cells may in certain instances require thymus-derived lymphocytes. This cytotoxicity is inhibited by humoral antibodies. However, humoral antibodies to surface antigens of target cells have also been shown to induce cytotoxicity of various effector cells, including thymus independent lymphocytes with affinity for antigen-antibody complexes. The cytotoxicity of such lymphocytes may be inhibited by complexes made up of antibodies to unrelated antigens. Knowledge of these different mechanisms in human neoplasia is fragmentary. In human urinary bladder carcinoma, circulating lymphocytes specifically cytotoxic for bladder carcinoma cells *in vitro* have been found. This cytotoxicity is well correlated to the clinical course of the disease. It reflects the presence in the patients of either a growing tumor or, after radiotherapy, of tumor-associated material. It is diminished or absent in patients with large tumors or distant metastases. This cytotoxic reaction can be inhibited by glycoprotein extracts of bladder tumor. The extent of participation of thymus-dependent lymphocytes in the reaction varies in different cases, suggesting that humoral antibodies may have both enhancing and inhibitory effects in this disease.

0968 ALTERATION OF IMMUNOCOMPETENCE BY MAMMARY TUMOR VIRUS.

(E.) Griswold, D. E. (Roger Williams Gen. Hosp., Providence, R.I.), G. H. Heppner and P. Calabresi. *J Natl Cancer Inst* 50(4): 1035-1038, 1973.

Methylated bovine serum albumin (MBSA)-induced hypersensitivity and antisheep red blood cells (SRBC) hemolysin responses were studied in C3H/HeJ mice harboring mammary tumor virus (MTV) and an MTV-negative companion strain, C3HeB/FeJ. Only C3HeB/FeJ mice produced appreciable hypersensitivity to MBSA, as measured by paw edema. Production of 7S hemolysin plaque-forming cells (hpfc) to SRBC was comparable in the two lines, but C3HeB/FeJ mice produced higher numbers of 19S hpfc to SRBC. Foster-feeding of C3HeB/FeJ mice on MTV-infected C3H/HeJ mothers resulted in animals with diminished capacity to produce an MBSA-induced hypersensitivity but without impairment of SRBC-induced hemolysin production. Virus infection increased the ability of female mice to produce 7S hemolysin. Thus the effect of MTV on immunocompetence is more complex than a simple immunosuppression.

0969 SERUM GLOBULIN CHANGES IN TUMOR-BEARING HAMSTERS.

(E.) Hinrichs, D. J. (Dept. Bacteriol. Public Hlth, Washington State U., Pullman), J. W. Templeton, L. E. Irish and D. R. Burger. *Oncology* 27(1):64-68, 1973.

Serum changes in 25 hamsters implanted with a hamster-derived melanoma No. 3 were analyzed by polyacrylamide gel disc electrophoresis. Initial serum globulin changes occurred 10-14 days post implantation when primary tumor size averaged 1.5-2 cm^3. Final changes occurred only after substantial metastatic spread of the tumor (days 21-28). The most dramatic changes were observed in the α-globulin region but changes in other fractions were also noted. Serum region 2ab decreased from 13.2% in the initial changes stage to 4.9% after metastatic spread, while region 4 increased from 8.8 to 12.1%.

0970 SPECIFIC TRANSPLANTATION ANTIGENS OF MURINE NEOPLASMS INDUCED BY VISNA AND PROGRESSIVE PNEUMONIA VIRUSES.

(E.) Law, L. W. (Natl. Cancer Inst., Bethesda, Md.) and K. K. Takemoto. *J Natl Cancer Inst* 50(4):1075-1079, 1973.

The growth of transplanted sarcomas originally induced in A/LN and BALB/c mice by visna virus #1, visna virus #2 and progressive pneumonia virus (PPV) was significantly inhibited in syngeneic recipients immunized by foot pad inoculation and amputation or treatment with irradiated, disrupted or freeze-thawed cells. The most marked inhibition was observed with tumor visna #2 in BALB/c mice which indicated the existence of strong membrane-bound tumor antigens. In contrast to most virus-specified transplantation antigens, the new antigens did not show cross-reactivity. The antigens resemble in certain respects the new class of tumor antigen described in murine mammary adenocarcinomas; they are resistant to X-irradiation, and irreversibly damaged tumor cells bearing the antigens can induce a specific immunity.

0971 ISOLATION OF PLASMA MEMBRANE ANTIGENS FROM EPSTEIN-BARR VIRUS-INFECTED LYMPHOID CELLS.

(E.) Boone, C. W. (Natl. Cancer Inst., Bethesda, Md. P. Gerber and P. B. Brandchaft. *J Natl Cancer Inst* 50(4):841-848, 1973.

The plasma membrane fraction of Epstein-Barr virus (EBV)-infected lymphoid cells was isolated on sucrose density gradients in an attempt to also isolate EBV-coded membrane antigen (MA). The surface HL-A and xenoantigens of these cells were readily isolated in this way. The location of antigens on the gradients was monitored by bound radioiodine-labeled proteins from MA-reactive sera and normal sera introduced by incubation of intact cells with labeled proteins bef fractionation (paired-label technique). Though the cells used were 40% positive for MA by membrane immunofluorescence before fractionation, no detectabl MA remained associated with the plasma membrane after fractionation. However, a peak of antibody-bound material appeared in the denser region of the gradien which contained EBV by electron microscopy. No detectable soluble MA-antibody complexes were released during the isolation procedure. In contrast to the stronger bonds which hold HL-A and xenoantigens to the plasma membrane, the MA-membrane bond appears to be weak enough to be broken during hypotonic treatment and mechanical rupture of the cells. It is concluded that the majority of cells in an EBV carri culture that are positive for MA by immunofluorescen may be carrying EBV and MA-positive membranous debri adsorbed to their surfaces derived from the perinucl and endoplasmic membranes of degenerating infected cells.

0972 MELANOMA-SPECIFIC ANTIBODIES PRODUCED IN MONKEYS BY IMMUNIZATION WITH HUMAN MELANOMA CELL LINES.

(E.) Metzgar, R. S. (Duke U. Med. Ctr., Durham, N.C.), P. M. Bergoc, M. A. Moreno and H. F. Seigler. *J Natl Cancer Inst* 50(4):1065-1068, 1973.

monkeys immunized s.c. with a melanotic tissue-
ture cell line from a cerebral metastasis (SMB)
an amelanotic line derived from a lymph node
astasis (WHRD) produced antibodies which appeared
be specific for melanoma antigen(s). The anti-
a, after absorption with human erythrocytes,
mal peripheral blood leukocytes and Hela cells,
led to react with normal human fibroblasts and
phocytes or with nonmelanoma malignant cell lines
gave strong reactivity by cytotoxicity and
ed agglutination with SMB, WHRD and two other
anoma cell lines.

3 TUBERCULIN REACTION IN CANCER PATIENTS, "MANTOUX RELEASE," AND LYMPHOSUPPRESSIVE-
MULATORY FACTORS. *(E.)* Steward, A. M. (Sligo
nty Hosp., Ireland). *J Natl Cancer Inst* 50(3):
-632, 1973.

Mantoux response was evaluated in two groups
patients with carcinoma. In 47 patients with
mary carcinoma of the gastrointestinal tract,
ast, and prostate, the Mantoux responses were
ressed before primary tumor resection, compared
h 25 patients tested after resection and with
normal controls. Fifty-nine patients with
nchial carcinoma had normal or stronger reactions
n controls. Old Tuberculin 1:1000 and later
ified Protein Derivative 1:5000 were used. The
toux response increased 2-6 weeks after primary
or resection in 6 of 7 patients with colon and
tal cancer, in 16 of 24 bronchial cancer, and
2 after estrogen treatment for prostate cancer.
s increase in response appeared to be a "release"
preexisting sensitivity, suppressed in the pre-
ce of the tumor, rather than a conversion from
"intrinsic" negativity to positivity. Mantoux
ease indicated a change in immunologic status
er treatment and did not necessarily indicate
ative surgery. The suppression of response was
ociated with a circulating immunosuppressive
tor in 11 of 16 patients examined: Serum
pressed tuberculin-induced lymphocyte DNA
thesis, but in higher dilution DNA synthesis was
mulated. This appeared to be one cause of Mantoux
ression, and changing titer following tumor
ection appeared to be one cause of Mantoux
ease. This clinical picture may resemble the
specific immunosuppression seen in mice during
erimental tumor induction, but a more specific
ationship cannot be discounted.

4 MOUSE STRAIN-RELATED DIFFERENCES IN THE BIOLOGIC AND IMMUNOLOGIC RESPONSES TO A
INE SARCOMA VIRUS. *(E.)* Gazdar, A. F. (Natl.
er Inst., Bethesda, Md.), E. K. Russell and R. B.
erman. *J Natl Cancer Inst* 50(4):971-978, 1973.

ortant differences were present in the biologic
immunologic responses of 4-week-old BALB/c, C3H,
(NZW X NZB)F_1(B/W) mice to a murine sarcoma virus
-MSV) isolated from a B/W mouse. Although the
e strains rapidly developed large soft-tissue
ors at the virus inoculation site, the tumors re-
ssed in C3H mice but grew progressively in BALB/c mice. These 2 strains showed excellent correlation among tumor regression, virus titers, and plasma levels of virus-neutralizing and cytotoxic antibodies. The biologic responses of B/W mice to the virus were complex and were not correlated with the humoral immune responses. Tumors in B/W mice persisted for long periods, despite histologic evidence of rejection and high levels of cytotoxic antibodies. Cellular immune responses to the tumor were relatively weak in all three strains. B/W mice developed massive splenomegaly, with extensive inflammatory fibrosis and vascular proliferation replacing the normal architecture. The biologic responses of NZB and B/W mice to Gz-MSV were similar. Splenectomy did not prevent the death of virus-inoculated BALB/c, NZB, and B/W mice but it prolonged the survival time of B/W mice.

0975 THYMUS AND BONE MARROW DERIVED LYMPHATIC LEKAEMIA IN MICE. *(E.)* Haran-Ghera, N. (Dept. Chem. Immunol., Weizman Inst. Sci., Rehovot, Israel) and A Peled. *Nature* 241(5389):396-398, 1973.

Lymphatic leukemia was induced in intact, thymectomized irradiated or reconstituted (by injection of 10^6 isologous normal bone marrow cells) SJL/J mice by 7,12-dimethylbenz(a)anthracene (DMBA, 1 mg) administered four times at weekly intervals by stomach tube. Incidence of lymphatic leukemia ranged from 52% in intact mice to 70% in thymectomized mice receiving 550 R whole body irradiation. Average latent period was 130-160 days. Thymus-dependent lymphatic leukemia was induced by irradiation or by a leukemogenic virus. Thymic lymphomas occurred in 40% of SJL/J mice exposed to four weekly doses of 170 R whole body irradiation and in 70% of mice inoculated in one thymus lobe with 0.02 ml of virus, prepared from thymic lymphoid leukemia induced in syngeneic mice by serial passage of a virus isolated from irradiated nonleukemic bone marrow, and subsequently exposed to 400 R whole body irradiation. Average latent periods were 200 days for irradiation-induced lymphomas and 150 days for virus-induced lymphomas. Tests for surface antigens indicated that the DMBA-induced lymphatic leukemia in intact or thymectomized hosts was of B lymphocyte origin, while the radiation-and virus-induced lymphatic leukemia was of T lymphocyte origin.

0976 MURINE LEUKEMIA AND SARCOMA VIRUSES: FURTHER STUDIES ON THE ANTIGENS OF THE VIRAL ENVELOPE. *(E.)* Gomard, E. (Hosp. St. Louis, Paris, France), J. C. Leclerc and J. P. Levy. *J Natl Cancer Inst* 50(4):955-961, 1973.

The antigens of the envelope (VEA) of murine leukemia sarcoma viruses were studied by *in vivo* neutralization of nine different pseudotypes of the murine sarcoma virus (MSV) with specific antisera prepared in rats or mice. Antisera directed against 15 different murine leukemia viruses as well as against the related MSV pseudotypes were tested. Four categories of viruses were separated by mouse antisera: 1) Moloney, Rauscher, Friend, Rich, Buffet viruses, and the LLV, 2) Graffi, Mazurenko, and Stepina-Zilber viruses, 3)

Gross, AKR, and Kaplan viruses, 4) Tennant virus. The first two categories included agents which induced the FMRGi antigen on the host cell membrane, but they could be separated according to the antigens of the viral envelope; a strong antigenic specificity differentiated between the two. The agents of the third category, which induced G antigen on the host cell membrane, were poorly immunogenic for mice, but an antigen specific for this subgroup could be detected by an antiserum prepared by intrathymus inoculation. It is proposed to name the main antigens of the three virus groups VEA I, II, and III. Some weak cross-reactions were found among the viruses of all three categories--probably due to minor antigens. The Tennant virus behaved in a way that did not permit its classification in one of the three categories. With rat antisera, a group-specific antigen of the viral envelope (gs VEA) was detected in all murine leukemia sarcoma viruses.

0977 IMMUNOCHEMICAL AND IMMUNOLOGICAL STUDIES OF HUMAN BRAIN TUMOURS. *(Fr.)* Delpech, B. (Becquerel Cancer Campaign Reg. Ctr., Rouen, France), A. Delpech, J. Clement and R. Laumonier. *Int J Cancer* 9(2):374-382, 1972.

Brain astrocytoma extracts (obtained from surgically removed preparations), treated with normal antibrain immune sera or with antiglioblastoma immune sera revealed the presence of antigens common to different organs as well as traces of brain specific fetal antigen. No carcinoembryonic type antigen could be detected but large amounts of lactoferrin were present in the examined extracts. Application of an antiglioblastoma immune serum revealed traces of a splenic and hepatic antigen whoses origin might be revealed only by further cytological studies. Immunofluorescence failed to detect autologous antitumor antibodies, whereas typical fluorescence was observed in tumor cells upon treatment with antinuclear active sera.

0978 A MICROASSAY FOR THE QUANTITATION OF CYTOTOXIC ANTITUMOR ANTIBODY: USE OF ^{125}I-IODODEOXYURIDINE AS A TUMOR CELL LABEL. *(E.)* LeMevel, B. P. (Natl. Cancer Inst., Bethesda, Md.) and S. A. Wells, Jr. *J Natl Cancer Inst* 50(3):803-806, 1973.

A microassay was developed for the detection of cytotoxic tumor-specific antibody against ^{125}I-iododeoxyuridine (IDU)-labeled sarcoma cells induced by 3-methylcholanthrene in C57BL/6N mice. From 1-2 X 10^3 tumor cells were grown in wells and labeled with 0.25-0.5 μCi IDU/well after a 6-hour incubation with immune sera (IS) and complement. Serum cytotoxicity is then determined by radioactive counting in a spectrometer. That the cytotoxic reactions are specific is demonstrated by the fact that IS were markedly cytotoxic (50-90%) at high dilutions against immunizing tumor cells, but only minimally cytotoxic at low dilutions against unrelated MCA tumor cells.

0979 HOST IMMUNITY TO GROWING SARCOMAS: TUMOR-SPECIFIC SERUM INHIBITION OF TUMOR-SPECIFIC CELLULAR IMMUNITY. *(E.)* Cohen, A. M. (Natl. Cancer Inst., Bethesda, Md.). *Cancer* 31(1):81-89, 1973.

Host immunity to growing antigenic sarcomas was studied in a methylcholanthrene-induced guinea pig tumor system (MCA-25/Strain 2 GP) and in patients with skeletal and soft tissue sarcomas. Tumor-specific cellular immunity was assayed by cell-mediated cytotoxicity. Sera from animals and patients with growing sarcomas were evaluated for specific inhibition of this cellular immunity. Groups of guinea pigs received an intramuscular injection of 1x10^5 MCA-25 sarcoma cells. Splenic lymphocytes from these animals and from control animals were assayed for cytotoxicity against MCA-25 target cells and also against MCA-A target cells. Cell-mediated toxicity was present against the MCA-25 target cells but not against MCA-A cells. No significant difference in the degree of cell-mediated cytotoxicity was found among the following groups of animals: the first and second week of tumor growth, when the tumor was palpable, following amputation of the tumor, or following hyperimmunization with multiple intradermal injections of MCA-25 tumor cells. Sera from animals with palpable MCA-25 tumors inhibited MCA-25 specific cellular cytotoxicity, but sera 2 wk after tumor removal were noninhibitory. This inhibition was apparently mediated by peripheral enhancing antibody. Human sarcoma-specific cellular cytotoxicity was present in most sarcoma-bearing patients. Sera from 14/16 sarcoma-bearing patients inhibited sarcoma-specific cellular immunity, but not cellular immunity against carcinoma. Of 11 sarcoma sera tested that completely inhibited sarcoma-specific cellular cytotoxicity, only two of these inhibited cell-mediated cytotoxicity against lung carcinoma. The data indicate that the presence or absence of the serum inhibitor was clearly related to the clinical status of the patient. The presence of inhibitors in sera following surgery usually antedated the clinical recurrence of sarcoma. These observations demonstrate an immunological phenomenon which may explain progressive sarcoma growth in the presence of tumor-specific cellular immunity.

0980 PROTECTION AGAINST GRAFFI'S LEUKEMIA IN MICE TREATED WITH LEUKEMIC CELLS PHOTO-INACTIVATED BY PSORALEN. *(E.)* Musajo, L. (Inst. Chem. Pharm., U. Padua, Italy) and F. Bacchichetti. *Eur J Cancer* 8:397-398, 1972.

Leukemic cells, originally induced by Graffi's virus in C57BL mice, produced 80 and 100% mortality in syngeneic mice when injected s.c. in doses of 10^6 and 10^7 cells/mouse, resp. Pretreatment of C57BL mice with four to six s.c. injections of leukemic cells irradiated at 365 nm in the presence of 10 μg psoralen afforded complete protection against challenge with leukemic cells. No deaths occurred among 10 animals given a 10^6 dose or among 63 animals given a 10^7 dose. The resistance induced by treatment with leukemic cells photo-inactivated with psoralen appeared to be lasting. A 100% survival, observed for 60 days, was obtained when treated animals were reinjected 60 days after the first challenge.

ANTIGENICITY OF DIETHYLNITROSAMINE-INDUCED RAT HEPATOMAS. *(E.)* Garisoain, M. J.
lix Huarte Ctr. Biol. Investigations, U. Navarre,
lona, Spain) and J. M. Arcos. *Acta Oncol (Madr)*
:40-47, 1970.

antigenicity of hepatomas arising in male Wistar
fed diethylnitrosamine (DEN, 42 mg/l) in
r drinking water for 20-22 wk was studied. The
ence of tumor antigens was analyzed by direct
moelectrophoresis of hepatoma homogenates against
-normal liver and against anti-hepatoma immune
m prepared in rabbits. The studies revealed the
ence of a single precipitation band correspond-
to an antigen present in hepatoma but not normal
t liver cells. This antigen could also be
cted in liver from 11-18 day-old embryos and
newborns. This carcino-embryonic antigen,
gnated DEN-86, contained no lipids or carbohy-
es and did not possess esterase activity. OEN-
as unaffected by heating at 80 C for 60 min
00 C for 20 min, and was resistant to ultra-
d treatment for 60 min. DEN-86 was present in
nuclear fraction but could not be detected in
microsomal or soluble fractions.

TUMOUR PRODUCTION IN IMMUNOSUPPRESSED RATS WITH CELLS TRANSFORMED *IN VITRO* BY ADENO-
S TYPE 2. *(E.)* Gallimore, P. H. (Med. Sch., U.
ingham, England). *J Gen Virol* 16:99-102, 1972.

day-old Hooded Lister rats were inoculated i.p.
cells from two adenovirus 2 (Ad 2)-transformed
es from Wistar White rat embryo fibroblasts
10^6 cells/rat). Rabbit anti-rat thymocyte
m (ATS) was administered on days 10-13 (0.2 ml
/rat/day). Tumors were palpable by day 20 in
treated animals injected with cells from one of
transformed clones (A2/REF/1) and by day 31 in
ted animals injected with cells from the second
sformed clone (A2/REF/2). Deaths from invasive
rs occurred as early as day 34. Post-mortem
inations revealed many tumors in the peritoneal
ty, infiltration of the liver, and secondary
rs in the lung. Frozen sections of tumors and
s derived from tumor growths grown *in vitro*
positive for AD 2 T-antigen. No tumors appear-
n rats injected with transformed cells but
ted with normal rat serum.

IMMUNOGENICITY OF NEURAMINIDASE TREATED $6C_3$HED LYMPHOMA IN C_3H_{HeJ} AND C_3H_f MICE.
Bekesi, J. G. (Roswell Park Mem. Inst.,
alo, N.Y.), L. Walter and J. F. Holland.
Am Assoc Cancer Res 13(March):113, 1972.

DISTRIBUTION OF SARCOMA ANTIGEN (S1). *(E.)* Hirshaut, Y. (Sloan-Kettering Inst.
Hosp., New York, N.Y.), D. M. Tsien, R. C.
ove and E. S. Essner. *Proc Am Assoc Cancer*
13(March):114, 1972.

CHARACTERIZATION AND AMINO-TERMINAL AMINO ACID SEQUENCES OF A TUMOR ASSOCIATED CARCINOEMBRYONIC ANTIGEN PROTEIN. *(E.)* Terry, W. D. (Natl. Inst. Hlth., Bethesda, Md.), P. Henkart, J. E. Coligan and C. W. Todd. *Proc Am Assoc Cancer Res* 13(March):116, 1972.

0986 IgA PLASMOCYTOMA WITH A TOTAL IgM-ANTIBODY DEFICIENCY SYNDROME AND CRYOGLOBULINEMIA. *(Ger.)* Gay, S. (K. Marx U. Inst. Path. Leipzig, E. Germany) and D. Weinhold. *Z Innere Med* 27(19):863-866, 1972.

0987 THE APPLICATION OF α_1-FETOPROTEIN IN THE DIAGNOSIS OF PRIMARY HEPATOCELLULAR CARCINOMA. *(Ger.)* Müller, G.-M. (Central Inst. Occupational Med., Berlin, E. Germany) and G. Geserick. *Z Innere Med* 27(24):1084-1087, 1972.

0988 IMMUNOLOGICAL STUDIES IN UGANDAN PATIENTS WITH HEPATOCELLULAR CARCINOMA. *(E.)* Primack, A. (Uganda Cancer Inst., Kampala), C. L. Vogel and L. F. Barker. *Br Med J*(5844):16-19, 1973.

0989 BLOOD CARCINOEMBRYONIC ANTIGEN, REGAN ISOENZYME, AND HUMAN CHORIONIC GONADOTROPHIN IN PRIMARY MEDIASTINAL CARCINOMA. *(E.)* Bagshawe, K. D. (Charing Cross Hosp., Fulham, London), G. T. Rogers, F. Searle and H. Wilson. *Lancet* (7796):210-211, 1973.

0990 IMMUNOLOGIC MANIPULATION OF METASTASES DUE TO HERPESVIRUS TRANSFORMED CELLS. *(E.)* Duff, R. (M. S. Hershey Med. Ctr., Hershey, Pa.), E. Doller and F. Rapp. *Science* 180(4081): 79-81, 1973.

0991 AGGLUTINATION OF LEUKEMIC MYELOBLASTS BY NEURAMINIDASE. *(E.)* Sauter, C. (Inst. Med. Microbiol., U. Zürich, Switzerland), J. Lindenmann and A. Gerber. *Eur J Cancer* 8(4): 451-453, 1972.

0992 THE INDUCTION OF IMMUNOPROTECTION BY MOUSE ASCITES TUMOR CELLS ATTENUATED IN TISSUE CULTURE. *(E.)* Morgan, J. F. (Dept. Biochem. Cancer Res., U. Saskatchewan, Canada) and C. P. Eng. *Eur J Cancer* 8(3):293-298, 1972.

0993 ELECTROPHORETICALLY HOMOGENEOUS MYELOMA LIGHT CHAIN mRNA AND ITS TRANSLATION *IN VITRO*. *(E.)* Tonegawa, S. (Basel Inst. Immunol., Switzerland) and I. Baldi. *Biochem Biophys Res Commun* 51(1):81-87, 1973.

0994 A UNIQUE IgG MYELOMA IMMUNOGLOBULIN—MOLECULAR WEIGHT AND OTHER PROPERTIES. *(E.)* Bobrzecka, K. (Inst. Med. Biochem., Med. Sch., Cracow, Poland), L. Konieczny, J. Rybarska, J. Bogdal and I. Urasinski. *Biochim Biophys Acta* 295(2):564-575, 1973.

0995 A PROTEIN RELATED TO IMMUNOGLOBULIN LIGHT CHAIN DETECTED IN MOUSE MYELOMA CELLS. *(E.)* Jones, G. H. (Dept. Zoology, U. Michigan, Ann Arbor). *Biochem Biophys Res Commun* 51(1):88-93, 1973.

0996 TRANSFER RIBONUCLEIC ACIDS FROM MOUSE PLASMACYTOMA TUMORS PRODUCING κ AND λ IMMUNOGLOBULIN CHAINS. *(E.)* Bridges, K. R. (Dept. Zoology, U. Michigan, Ann Arbor) and G. H. Jones. *Biochemistry* 12(6):1208-1212, 1973.

0997 A TRANSPLANTABLE HUMAN BLADDER CANCER IN THE UNCONDITIONED SYRIAN HAMSTER. *(E.)* Elhilali, M. M. (U. Hosp. Ctr., Sherbrooke, Canada), S. K. Nayak and C. Brailovsky. *Invest Urol* 10(3):230-234, 1972.

0998 RESIDUAL T CELLS IN CHRONIC LYMPHOCYTIC LEUKAEMIA. *(E.)* Goldstone, A. H. (Dept. Endocrinol., Royal Infirm., Edinburgh, Scotland), L. Kerr, S. J. Urbaniak, A. Farmer and W. J. Irvine. *Lancet* (7801):481-482, 1973.

0999 IMMUNOGLOBULIN SYNTHESIS AND SECRETION BY CELLS IN THE MOUSE THYMUS THAT DO NOT BEAR θ ANTIGEN. *(E.)* Vitetta, E. S. (New York U. Sch. Med., New York), J. W. Uhr and E. A. Boyse. *Proc Natl Acad Sci USA* 70(3):834-838, 1973.

1000 IMMUNITY IN THE ACUTE LEUKAEMIAS. *(E.)* Parker, J. E. (British Columbia, Canada). *Lancet* (7804):679, 1973.

1001 MOLECULAR WEIGHT OF A HUMAN J CHAIN. *(E.)* Schrohenloher, R. E. (Div. Clin. Immunol., U. Birmingham, Ala.), J. Mestecky and T. H. Stanton. *Biochim Biophys Acta* 295(2):576-581, 1973.

1002 CELL PROLIFERATION IN TRANSPLANTED RAT TUMORS: INFLUENCE OF THE HOST IMMUNE SYSTEM. *(E.)* Rajewsky, M. F. (Max-Planck Inst. Virus Res., Tübingen, Germany) and A. Grüneisen. *Eur J Immunol* 2(5):445-447, 1972.

1003 REGULATION OF NORMAL AND LEUKAEMIC LYMPHOCYTE TRANSFORMATION AND RECIRCULATION BY SODIUM PERIODATE OXIDATION AND SODIUM BOROHYDRIDE REDUCTION. *(E.)* Zatz, M. M. (Albert Einstein Coll. Med., Bronx, N.Y.), A. L. Goldstein, O. O. Blumenfeld and A. White. *Nature* [*New Biol*] 240(103):252-255, 1972.

1004 A DIFFUSION CHAMBER METHOD FOR THE DETECTION OF LYMPHOCYTE-MEDIATED CYTOTOXICITY OF ^{125}IUdR-LABELED TUMOR CELLS. *(E.)* Kreider, J. W. (Dept. Path., Coll. Med., Pennsylvania St. U., Hershey), W. H. Shoff and N. Lengle. *Transplantation* 14(2):278-283, 1972.

1005 CELL-MEDIATED IMMUNITY IN PATIENTS WITH MALIGNANT MELANOMA. *(E.)* Mackie, R. M. (Dept. Dermatol., U. Glasgow, Scotland), W. G. S. Spilg, C. E. Thomas and A. J. Cochran. *Br J Derm* 87:523-532, 1972.

1006 INCREASED PHOSPHOFRUCTOKINASE CONCENTRATIO IN HEPATOMA 3924-A: ENZYMIC AND IMMUNOLOGICAL EVIDENCE. *(E.)* Dunaway, G. A., Jr. (Indiana U. Sch. Med., Indianapolis), H. P. Morris and G. Weber. *Life Sci* [*II*] 11(18):909-918, 1972.

1007 REGIONAL ILEITIS, LEUKAEMIA, AND HUMORAL IMMUNE DEFICIENCY IN A FAMILY. *(E.)* Park J. E. (British Columbia, Canada) and J. W. Thomas. *Lancet* (7785):1038, 1972.

1008 MALIGNANCY ASSOCIATED WITH ANTINUCLEAR ANTIBODIES. *(E.)* Zeromski, J. O. (Dept. Path. Anat., Med. Sch., Poznan, Poland), M. K. Gorny and K. Jarczewska. *Lancet* (7785):1035-1036, 1972.

1009 *IN VIVO* AND *IN VITRO* PRODUCTION OF HUMAN CHORIONIC GONADOTROPIN AND ALPHA-FETOPROTEIN BY VIRILIZING HEPATOBLASTOMA. *(E.)* Braunstein G. D. (Natl. Cancer Inst., Bethesda, Md.), W. E. Bridson, A. Glass, E. W. Hull and K. R. McIntire. *J Clin Endocrinol Metab* 35(6):857-862, 1972.

1010 "NONSECRETORY" PLASMA CELL MYELOMA: OBSERVATIONS ON SEVEN CASES WITH ELECTRON MICRO SCOPIC STUDIES. *(E.)* Azar, H. A. (Dept. Path., Columbia U., New York, N.Y.), E. C. Zaino, T. D. Pham and K. Yannopoulos. *Am J Clin Pathol* 58(6):618-629, 1972.

1011 DNA REPAIR IN HUMAN LEUKAEMIC LYMPHOCYTES *(E.)* Huang, A. T. (Duke U. Med. Ctr., Durham, N. Carolina), W. B. Kremer, J. Laszlo and R. B. Setlow. *Nature* [*New Biol*] 240(99):114-115, 1972.

1012 HUMAN TUMOURS GROWN IN MICE. *(E.)* Castr J. E. (MRC Clin. Res. Ctr., Middlesex, England). *Nature* [*New Biol*] 239(90):83-84, 1972.

1013 BINDING OF CONCANAVALIN A TO RAT, NORMAL HUMAN, AND CHRONIC LYMPHATIC LEUKEMIA LYMPHOCYTES. *(E.)* Novogrodsky, A. (Weizmann Inst Sci., Rehovot, Israel), M. Biniaminov, B. Ramot and E. Katchalski. *Blood* 40(3):311-316, 1972.

1014 IMMUNOLOGICALLY ACTIVE HETEROSACCHARIDES OF CARCINOEMBRYONIC ANTIGEN OF HUMAN DIGESTIVE SYSTEM. *(E.)* Banjo, C. (Montreal Gen. Hosp., Canada), P. Gold, S. O. Freedman and J. Krup *Nature* [*New Biol*] 238(84):183-185, 1972.

5 TRANSFER OF INCREASED RESISTANCE TO BO-
IV CARCINOMA BY SERUM IN THE SYNGENEIC
TEM. *(E.)* Wotke, J. (Fac. Med., J. E. Purkyne
Brno, Czechoslovakia) and A. Rejthar. *Scr Med*
5):293-298, 1972.

6 STUDIES ON TUMOR-HOST RELATIONSHIP. V.
EFFECT OF *MYO*-INOSITOL ON TUMOR CELL GROWTH
VIVO AND ON THE RESISTANT ACTIVITY OF THE HOST.
) Tanino, T. (Inst. Med. Sci., U. Tokyo, Japan),
Narumi and T. Tsumita. *Jap J Exp Med* 42(6):575-
, 1972.

7 THE PRESENCE OF GROUP-SPECIFIC ANTIGENS
AND C PARTICLES IN THE LIQUID PHASE OF
ITIC TUMORS OF THE MOUSE. *(It.)* Ribacchi, R.
ncer Res. Dept. U. Perugia, Italy). *Lav Ist Anat*
ol Patol Perugia 31(3):91-102, 1971.

3 TUMOR SPECIFIC HOST RESISTANCE FOR
TRANSPLANT C57BL MOUSE LYMPHOMAS. *(E.)*
uyama, Y. (U. Kentucky, Lexington) and J. Feola.
c Am Assoc Cancer Res 13(March):112, 1972.

9 LYMPHOCYTE ATPase ACTIVITY IN DETECTION
OF SOLID MALIGNANT TUMORS - DOUBLE BLIND
DIES. *(E.)* Dimitrov, N. V. (Philadelphia Gen.
p., Pa.), J. Ellegard and I. Brodsky. *Proc Am*
oc Cancer Res 13(March):111, 1972.

0 SERUM FACTORS SPECIFICALLY ANTAGONISTIC
TO OR SYNERGISTIC WITH CYTOTOXIC
PHOCYTES IN PATIENTS WITH NEOPLASTIC DISEASE.
) Sinkovics, J. G. (U. Texas M. D. Anderson
., Tumor Inst., Houston), N. Ahmed, J. R.
iness and W. J. Reeves. *Proc Am Assoc Cancer*
13(March):109, 1972.

PREVALENCE IN ENGLAND OF ANTIBODY TO
HUMAN POLYOMAVIRUS (B.K.). *(E.)* Gardner,
. (Virus Reference Lab., London, England).
ed J (5845):77-78, 1973.

See also:

- * (Rev): 0601, 0609, 0613, 0619, 0627, 0630, 0631, 0653
- * (Chem): 0675, 0729
- * (Viral): 0784, 0829, 0832, 0836, 0840, 0849, 0856, 0861, 0864, 0868, 0877, 0891, 0912
- * (Epid-Biom): 1043

1022 LONGITUDINAL BONE MARROW CHROMOSOME STUDIES IN POTENTIAL LEUKEMIC MYELOID DISORDERS. *(E.)* Lisker, R. (Natl. Inst. Nutrition, Mexico City, Mexico), A. Cobo de Gutierrez and M. Valazquez-ferrari. *Cancer* 31(3):509-515, 1973.

The possible relationship between bone marrow cell chromosome abnormalities and the subseqent development of leukemia was investigated. All 18 patients with pancytopenia, who were followed between 6 months and 13 yr, had normal karyotypes and no evidence of leukemia. Ten patients with various unusual myeloproliferative syndromes were followed for 6 months to 5 yr. Of these, six patients had chromosome abnormalities, including two with clonal evolution. Three patients died with no evidence of leukemia, one developed a form of chronic eosinophilic leukemia, and two are alive without evidence of leukemia. It was thus concluded that the presence of bone marrow chromosome abnormalities may not necessarily predispose to the development of acute leukemia.

1023 FORMATION OF PEROXIDES IN CELLULAR MEMBRANE PHOSPHOLIPIDS AND ITS ROLE IN THE PATHOGENESIS OF MALIGNANT GROWTH. *(Rus.)* Kagan, V. Ye., (Lomonosov State U. Moscow, USSR), M. V. Sitkovskiy, V. S. Danilov and Yu. P. Kozlov. *Dokl Akad Nauk SSSR* 208(3):733-735, 1973.

The dynamics of hydroperoxide content changes occurring in liver cell membrane phospholipids during the malignant growth process were studied in random-bred mice and rats. Mice, inoculated i.p. with 10^6 cells of Ehrlich ascites carcinoma, presented maximum hydroperoxide accumulation rates at the cephalin and lecithin levels during the period of exponential tumor growth 6 to 8 days postinoculation. Peroxidation products increased at higher rates in the cephalin than in the lecithin fraction. Rats receiving solid lymphosarcoma inocula elicited 1.5, 6, 2, and 2.5 fold hydroperoxide levels in their nuclear, mitochondrial, microsomal, and lysosomal lecithins, resp., 9 days post innoculation, with respect to controls. Nuclear and microsomal cephalin hydroperoxides were 6-fold, and mitochondrial and lysosomal cephalin hydroperoxides reached 3-fold levels with respect to controls. Accumulation of free radical oxidation products in tumor-bearing animal phospholipids might cause structural alterations of the cell and intracellular membrane formations, and thus be responsible for a wide range of processes involved in the pathogenesis of malignant growth.

1024 MUTAGENIC DNA POLYMERASE IN HUMAN LEUKEMIC CELLS. *(E.)* Springgate, C. F. (Inst. Cancer Res., Philadelphia, Pa.) and L. A. Loeb. *Proc Natl Acad Sci USA* 70(1):245-249, 1973.

DNA was removed from lymphocytes from normal donors and from acute lymphatic leukemia patients. The exactness of DNA replication by leukemic and normal cell extracts was compared using poly(dA-dT)·poly(dA-dT) as template in the presence of highly radioactive dGTP or dCTP and unlabeled dATP and dTTP. With this template, leukemic preparations polymerized ten times more dCTP than did normal cells. The observed differences in the exactness of the poly(dA-dT)·poly(dA-dT) synthesis did not appear to be due to nonspecific factors such as inhibitors or activators present in excess in normal or leukemic preparations. The most direct explanations for the differences in the precision of homopolymer replication between normal and leukemic extracts are (1) the presence of a viral DNA polymerase in leukemic cells or (2) the presence of an altered cellular polymerase that i faulty in base selection.

1025 INITIAL CHANGES OF LEUKEMIA IN RATS FED 2,7-DIACETYLAMINOFLUORENE. *(E.)* Takayama, S. (Tokyo Med. Dental Sch., Japan) and S. Kamata. *Acta Pathol Jap* 22(3):477-487, 1972.

Eighty female Wistar rats were fed a diet containing 0.025% 2,7-diacetylaminofluorene. Blood samples were examined at intervals for six months; the appearance of erythroblasts or leukocytosis in blood was assumed to indicate the initial stage of leukemia. Leukemia developed in 28 of 80 rats. In all 28 cases, erythroblasts were present in blood. Solitary lesions were found in bone marrow of eight of the leukemic rats. Nodular foci of these lesions, confined to the bone marrow, were thought to be the site of origin of the leukemias.

1026 ANDROGENIC CAUSE OF BREAST CARCINOMA AND ENDOMETRIAL HYPERPLASIA. *(E.)* Grattarola, R. (Natl. Tumor Inst., Milan, Italy). *Cancer Cytol* 12(2):25-28, 1972.

Evidence is presented which suggests that mammary cancer and endometrial hyperplasia is associated with overproduction of androgenic hormones, rather than overproduction of estrogens. While estrogen excretion values were similar in a group of five normal patients and in a group of eight "at risk" patients with atypical endometrium, androgen excretion was significantly elevated in the "at risk" group. Marked increases in androgenic activity were also observed in ten pre- and postmenopausal breast cancer patients with atypical endometrium.

1027 PATHOGENESIS OF C-CELL NEOPLASIA IN THYROID GLAND: C-CELL PROLIFERATION IN A CASE OF CHRONIC HYPERCALCAEMIA. *(E.)* Ljungberg, O. (Malmo Gen. Hosp., Sweden) and J. F. Dymling. *Acta Pathol Microbiol Scand [A]* 80(5): 577-588, 1972.

A 64-year-old male was diagnosed as having primary hyperparathyroidism on the basis of long-standing hypercalcemia. An enlarged parathyroid was surgically removed from the patient and a subtotal thyroidectomy was performed. The enlarged parathyroid consisted entirely of waterclear cells;

calcitonin-producing cells (C-cells) were en, and no normal parathyroid tissue was found. yroid specimens from both lobes contained veral benign follicular adenomas. A hyperplastic cumulation of C-cells was observed in a small ea of the thyroid parenchyma from one lobe. The yroid lesion was assumed to have resulted from cell proliferation perhaps caused in part by ronic hypercalcemia.

28 ABNORMAL IRON INCORPORATION, SURVIVAL, PROTOPORPHYRIN CONTENT AND FLUORES- NCE OF ONE RED CELL POPULATION IN PRELEUKEMIC DEROBLASTIC ANEMIA. *(E.)* Riedler, G. F. antonal Hosp., U. Zurich, Switzerland) and P. W. raub. *Blood* 40(3):345-352, 1972.

d blood cells of a 77-yr-old man with sideroblas- c anemia were examined; the patient eventually veloped myelomonoblastic leukemia. On multilayer ntrifugation, two major red cell populations were und, a light fraction (comprising about 8% of red lls) and a heavy fraction (comprising about 81% red cells). Light cells showed a smaller an corpuscular volume and a lower mean corpuscu- r hemoglobin than heavy cells. Light cells corporated 3.5% of ^{59}Fe in ten days and heavy lls incorporated 31% of the ^{59}Fe dose. Light lls had a half-life in ^{51}Cr labeling of 8.2 days ile heavy cells had a half-life of 20 days. ght cells had a higher reticulocyte count, fluoro- e count and protoporphyrin content than heavy lls. These findings suggest markedly reduced heme- nthetase activity in the abnormal red cell popu- tion. The development of leukemia in the tient supports the recent hypothesis that both deroblastic anemia and leukemia in the sample tient may be the result of mutations on the level the multipotent bone marrow stem cell.

9 EFFECTS OF MARIJUANA AND TOBACCO SMOKE ON DNA AND CHROMOSOMAL COMPLEMENT IN IAN LUNG EXPLANTS. *(E.)* Leuchtenberger, C. iss Inst. Exp. Cancer Res., Lausanne), R. chtenberger, U. Ritter and N. Inui. *Nature* (5397):403-404, 1973.

omparison was made between the DNA content in aphases and telophases, and number of chromosomes fibroblastic cells, of human lung explants before after exposure to marijuana and tobacco smoke. r to 28 days after exposure to fresh smoke there a significant decrease in number of cells with chromosomes, 4 DNA and 2 DNA. In addition, the iability of cells with deviating DNA content and omosomal numbers was greater than that observed unexposed cultures. The difference was especial- marked after marijuana cigarette smoke. The fact t the alterations of DNA and chromosomal complement sisted for prolonged periods indicates that they not lethal to cells.

0 ETIOLOGY AND TREATMENT OF DYSHORMONAL MAMMARY GLAND HYPERPLASIA. *(Rus.)* Popova, O. B. (City Oncol. Dispensary and Hosp., USSR Acad. Sci.) and I. A. Fridman. *Vopr Onkol* 18(12):78-79, 1972.

1031 COMPARATIVE MORPHOGENESIS OF MALIGNANT SKIN MELANOMA. *(E.)* Blinova, G. A. (Prof. Petrov Res. Inst. Oncol., Leningrad, USSR). *Tumori* 57(6):361-376, 1971.

1032 THE INCIDENCE OF MAST CELLS IN SELECTED ORAL LESIONS. *(E.)* Jandinski, J. J. (Tufts U. Sch. Dental Med., Boston, Mass.), S. Sonis and H. C. Doku. *Oral Surg* 34(2):245-248, 1972.

1033 SIDEROBLASTIC ANEMIA IN MULTIPLE MYELOMA: A PRELEUKEMIC CHANGE. *(E.)* Khaleeli, M. (U. Utah Coll. Med., Salt Lake City), W. M. Keane and G. R. Lee. *Blood* 41(1):17-25, 1973.

1034 THE CONDITION OF STEROID HORMONE EXCRETION IN PATIENTS WITH PRECANCEROUS DISEASE AND CANCER OF THE STOMACH. *(Ukr.)* Slynchak, S. M. (Sci. Res. Inst. Oncol. Radiol., Kiev, USSR) and V. V. Shapoval. *Dopov Akad Nauk Ukr RSR Ser B Kh* 83-85, 1973.

1035 THE DIAGNOSTIC SIGNIFICANCE OF SEX CHROMATIN IN HORMONE-DEPENDENT PRENEOPLASTIC AND NEOPLASTIC PROCESSES. *(Rus.)* Ganina, K. P. (Inst. Oncol. Probl. Kiev, USSR). *Vopr Onkol* 18(11):46-50, 1972.

1036 PATHOLOGICAL PROCESSES OF THE UTERINE CERVIX. *(Rus.)* Cherkasskiy, L. A. (Orenburg Med. Inst., USSR) and O. P. Mazurovskaya. *Vopr Onkol* 18(1):40-46, 1972.

1037 A CONTRIBUTION TO THE MORPHOGENESIS OF CANCER OF THE CORPUS UTERI. *(Rus.)* Cherkasskiy, L. A. (Orenburg Med. Inst., USSR) and L. D. Plesovskikh. *Vopr Onkol* 19(1):52-58, 1973.

1038 HORMONAL ALTERATIONS OCCURRING IN PATIENTS WITH DYSHORMONAL HYPERPLASIA OF THE MAMMARY GLANDS. *(Rus.)* Turkevich, N. M. (Inst. Oncol. Probl., Kiev, USSR), R. N. Akimova, A. N. Gorevaya, M. P. Ishchenko, N. S. Kartavova, L. N. Koretskaya, N. M. Makarchuk and Ye. M. Samundzhan. *Vopr Onkol* 19(1):31-35, 1973.

1039 THE INCIDENCE AND SIGNIFICANCE OF EARLY CARCINOMAS IN ENDOMETRIAL POLYPS. *(E.)* Salm, R. (Dept. Histopath., Royal Cornwall Hosp., Truro, England). *J Pathol* 108(1):47-53, 1972.

See also:

* (Rev): 0608, 0646
* (Chem): 0677, 0727, 0759
* (Phys): 0776

1040 EPIDEMIOLOGY OF ACUTE MYELOMONOCYTIC LEUKEMIA IN CHILDREN. *(E.)* Li, F. P. (Childrens Cancer Res. Fdn., Boston, Mass.), N. Jaffe, W. J. Mitus, W. C. Moloney and J. F. Fraumeni Jr. *Cancer* 31(3):516-519, 1973.

Acute myelomonocytic leukemia was diagnosed in 73 children at the Children's Cancer Research Foundation and the Children's Hospital Medical Center in Boston, 1949-1971. Although the disorder may occur within the first year of life, no association was found with inherited disorders or with prenatal exposure to potential carcinogens. Three patients had a well-documented history of antecedent aplastic anemia, suggesting that prolonged myelodepression is an etiologic factor or precursor state in myelomonocytic leukemia. In addition, eight others appeared to have antecedent anemia, thrombocytopenia, or pancytopenia for months to years, a finding previously described in adults with myelomonocytic leukemia.

1041 COLORECTAL CANCER IN ROCHESTER, MINNESOTA 1940-1969. *(E.)* Lanier, A. P. (Mayo Clin. Mayo Fdn., Rochester, Minn.), A. R. Wychulis, M. B. Dockerty, L. R. Elveback, O. H. Beahrs and L. T. Kurland. *Cancer* 31(3):606-615, 1973.

All residents of Rochester, Minn., with a diagnosis of cancer of the colon or rectum during the 30-year period, 1940 through 1969, were identified from the records of the Mayo Clinic and the other resources in the Rochester epidemiologic central file. The medical records and microscopic slides on these patients were reviewed, and 439 met the necessary residency and diagnostic criteria. Adjusted average annual incidence rates per 100,000 for the entire time period for cancer of the colon were 33.0 for males and 24.6 for females and for rectal cancer, 16.3 for males and 13.7 for females. Age-specific incidence rates increase progressively with age for cancer in both sites. Age-adjusted rates for both colon and rectal cancer were consistently higher for males than for females in the three decades studied. However, for both sites, the male predominance appeared only in those patients more than 60 years of age. No significant increases in incidence rates in either sex for cancer of the colon or rectum over the 30-year period was demonstrated.

1042 MALIGNANT LYMPHOMAS OF AFRICAN CHILDREN. *(E.)* Stewart, A. (Dept. Social Med., U. Oxford, England), J. N. P. Davies, G. Dalldorf and F. E. Barnhart. *Proc Natl Acad Sci USA* 70(1):15-17, 1973.

Comparative data on cancer incidence in Africa and in England and Wales is adduced to bear out a hypothesis about the etiology of lymphomas in African children. According to the theory, repeated attacks of malaria during infancy have several consequences for African children who are incubating malignant disease: (1) the malaria may make survival difficult and thus account for the relative deficit of leukemias and neural tumors among young children; (2) the masking effect of true incidence may be disproportionately large on cancers with short latent periods, such as leukemia and neuroblastoma; and (3) the acquisition of resistance to malaria among the survivors may determine the switch from leukemia to the multi-focal, restricted conditions of the Burkitt lymphoma type prevalent in African children.

1043 SERO-EPIDEMIOLOGY OF REOVIRUS TYPE 3 INFECTIONS IN FOUR AREAS OF UGANDA WITH VARYING INCIDENCE OF BURKITT'S TUMOUR. *(E.)* Munube, G. M. R. (East African Virus. Res. Unit, Entebbe, Uganda), T. M. Bell, P. M. Tukei and P. G. Smith. *East Afr Med J* 49(5):369-375, 1972.

Sera (840) collected from school children in four districts of Uganda were assayed in neutralization tests for anti-reovirus 3 antibodies to determine the incidence of reovirus 3 infection in the four areas. Reovirus 3 antibodies were found in sera from all areas, regardless of incidence of Burkitt lymphoma. However, the highest percentage of children with antibodies to the virus (82%) was found in Terego County, the area with the highest incidence of Burkitt lymphoma and where space-time clustering of Burkitt lymphoma cases has been demonstrated. There was no marked difference in percentage of antibody-containing sera between male and female children.

1044 EPIDEMIOLOGY OF URINARY BLADDER CANCER IN OKLAHOMA. *(E.)* Asal, N. R. (U. Oklahoma Hlth. Sci. Ctr., Oklahoma City) and S. W. Ferguson. *J Okla State Med Assoc* 65(10):409-417, 1972.

Data on mortality from urinary bladder cancer in Oklahoma from 1956-1970 were analyzed according to sex, race, and year of death. Among males, death rates for bladder cancer increased from 4.5/100,000 in 1956-1960 to 5.9/100,000 in 1966-1970. Female mortality did not increase. While 92.5% of bladder cancer deaths occurred among whites, only 1.0% occurred among American Indians. Although counties showing high and low bladder cancer mortality were well distributed throughout Oklahoma, mean death rates by county were found to increase with increasing degree of urbanization of counties. This increase, which was highly significant among males, suggests that an industrial hazard affecting bladder cancer incidence is operating in urban areas.

1045 MORTALITY FROM CANCER OF THE CERVIX IN LOUISIANA, 1960-1970. *(E.)* Newell, G. R. (Sch. Pub. Hlth. Tropical Med., Tulane U., New Orleans, La.) and E. K. Stancliff. *J La State Med Soc* 124(8):280-286, 1972.

A study on cervical cancer mortality in Louisiana

owed that the death rate for this condition in
70 was 5.5/100,000 among white women and 15.4/
0,000 among nonwhite women. Cervical cancer
rtality among white women had declined steadily
nce 1960, but no improvement was seen in the
ath rate among nonwhite women. A steady increase
mortality in age groups 25-34 and 65 and over
s seen. Cervical cancer mortality in Louisiana
owed a good correlation with poverty indices
cluding household without plumbing and female
ed of family. Among nonwhite women, there
re fewer deaths among widows and more deaths
ong single women and divorcees than among white
men.

46 TRENDS IN STOMACH CANCER INCIDENCE FOR MALE COHORTS IN FINLAND.

(E.) Hakama,
(Finnish Cancer Registry, Helsinki). *Ann Clin*
4(5):300-303, 1972.

e incidence of stomach cancer is higher in Finland
an in most other European countries. However,
e rates are decreasing with calendar time as a
sult of changes in etiological factors, e.g.,
nsumption of cereal. Data from the Finnish Cancer
gistry show that these changes have most affected
horts born around 1900 and that the estimated
sk of stomach cancer varied up to three-fold for
horts of Finnish males born only 20 years apart.
is suggests that stomach cancer will be a rare
sease even in Finland after the death of the
dest cohorts.

47 INCIDENCE OF CANCER IN NEGROES IN WASHINGTON, D.C. AND SELECTED AFRICAN CITIES.

) Kovi, J. (Howard U. Coll. Med., Washington,
C.) and M. Y. Heshmat. *Am J Epidemiol* 96(6):
-413, 1972.

results of a 5-year (1965-1969) survey of cancer
ong Negroes in 11 major hospitals in Washington,
., are reported. In males, the commonest
es of cancer, in order of decreasing frequency
e prostate, lung, esophagus, and colon. In
males, breast, cervix uteri, and colon were the
st common sites. Among Negroes the overall in-
ence of malignant disease in Washington, D.C.
considerably higher than in Africa. The aver-
annual incidence rate for males was 220.4/
,000 and for females 194.3. Incidence rates for
h males and females reached a peak in the 70-79
group, and then leveled off. Negroes in Wash-
gton, D.C., and in Alameda County, California,
vealed a pattern of cancer risk for leading
mor sites, except cervix uteri, much closer to
ites in Alameda County, California, than to Negroes
various parts of Africa, south of the Sahara.
fact, American Negroes had higher standardized
cidence rates for cancer of the esophagus, stomach,
ncreas, prostate and cervix uteri, but lower for
east, than Caucasians in Alameda County, Cali-
rnia. Among Negroes in Africa, only the South
rican Bantu showed a trend in susceptibility to
ncer comparable to American Negroes and whites.
ese findings emphasize the importance of environ-
ntal influences in the development of cancer in man.

1048 TIME-SPACE CLUSTERING AMONG CASES OF ACUTE LEUKEMIA IN TWO GEORGIA COUNTIES.

(E.) Evatt, B. L. (Public Hlth. Serv., Atlanta, Ga.), G. A. Chase and C. W. Heath, Jr. *Blood* 41(2):265-272, 1973.

Statistical analyses were made of the time-space configuration of 406 acute leukemia cases in DeKalb and Fulton Counties. Georgia, over the 13-yr period 1956-1968. Separate analyses were performed for each of three age groups, 0-14 yr, 15-49 yr, and 50 yr-plus, cases being assigned to 2- and 3-yr time segments and to census tract units according to date of onset and place of residence at diagnosis. Pearson chi-square statistics and Haldane's moments were used as a measure of homogeneity in case configurations. All calculations took into account age-specific populations for each census tract unit corrected for shifting population density. Evidence of inhomogeneity of time-space configurations, based on case clustering, was found among cases in the 0-14 age group but not at older ages. Clustering was present in both counties whether examined separately or combined, and it was more clearly seen in case distributions in DeKalb County. Analyses done for various periods of time showed that degree of clustering depended greatly on placement of time boundaries.

1049 ALARMING INCREASE OF THE CANCER MORTALITY IN THE U.S. BLACK POPULATION (1950-1967).

(E.) Henschke, U. K. (Howard U. Coll. Med., Washington, D.C.), L. D. Leffall, Jr., C. H. Mason, A. W. Reinhold, R. L. Schneider and J. E. White. *Cancer* 31(4):763-768, 1973.

The U.S. cancer mortality per 100,000 for both sexes rose, from 1950 to 1967, for blacks from 147 to 177, an increase of 20%, while it remained unchanged for whites at 150. The female cancer mortality rate declined for blacks from 146 to 142, a decrease of only 3%, while it declined for whites from 139 to 126, a decrease of 9%. The male cancer mortality rate rose for blacks from 147 to 220, an increase of 50%, while it increased for whites from 158 to 181, an increase of only 16%. In 1950, the cancer mortality rate for both sexes was 2% lower for blacks than for whites, but, by 1967, it had become 18% higher. Of the 58 most frequent U.S. sex-specified cancer types, 29 increased slower in whites, 9 decreased faster in whites, 14 showed no significant differences, and only 4 (malignant melanoma and reticulum cell sarcoma in both sexes) increased faster in whites. Environmental factors are the most likely causes for this alarming rise of cancer in U.S. blacks.

1050 RELATION OF ABNORMAL CYTOLOGICAL SMEARS AND CARCINOMA OF CERVIX UTERI TO HUSBAND'S OCCUPATION.

(E.) Wakefield, J. (U. Hosp. South Manchester, Manchester, England), R. Yule, A. Smith and A. M. Adelstein. *Br Med J* 5859(2):142-143, 1973.

An analysis of the cytological records of almost 300,000 women in the Manchester, England area showed that rates of positive or suspicious smears from population screening are highly correlated with cervical cancer mortality rates when both are distributed according to the husband's occupation. The correlation applied to 26 occupational groupings and 61 individual occupational units with more than 1000 women. These findings provide strong evidence that the condition revealed by an abnormal cytological smear is a stage in the development of invasive cancer of the uterine cervix.

1051 LEUKOCYTE LABELING WITH ^{51}CHROMIUM. IV. THE KINETICS OF CHRONIC LYMPHOCYTIC LEUKEMIC LYMPHOCYTES. *(E.)* Scott, J. L. (Wadsworth VA Hosp., Los Angeles, Calif.), R. McMillan, J. V. Marino and J. G. Davidson. *Blood* 41(1):155-162, 1973.

1052 QUANTITATIVE MORPHOLOGY OF CANCER CELLS. *(E.)* Meyer-Arendt, J. R. (Dept. Physics, Pacific U., Forest Grove, Oregon) and D. M. Humphreys. *Acta Histochem (Jena)* 44(1):41-48, 1972.

1053 *IN VIVO* TUMOR CELL KINETIC STUDIES: USE OF LOCAL THYMIDINE INJECTION FOLLOWED BY FINE-NEEDLE ASPIRATION. *(E.)* Muggia, F. M. (Albert Einstein Coll. Med., Bronx, N.Y.) and V. T. DeVita. *J Lab Clin Med* 80(2):297-301, 1972.

1054 COAL MINING AND CANCER OF THE STOMACH. *(E.)* Matolo, N. M. (U. Utah Coll. Med., Salt Lake City), W. M. Gorishek, V. Moslander and J. A. Dixon. *Rocky Mt Med J* 69(12):44-49, 1972.

1055 HYDROXYUREA EFFECTS IN THE C3H MOUSE. II. MAMMARY TUMOR CELL KINETICS. *(E.)* Dethlefsen, L. A. (U. Utah Med. Ctr., Salt Lake City) and R. M. Riley. *Cell Tissue Kinet* 6(2):173-184, 1973.

1056 CYTOKINETICS OF THE S-180 ASCITES TUMOR SYSTEM. *(E.)* Schiffer, L. M. (Allegheny Gen. Hosp., Pittsburgh, Pa.), J. S. R. Nelson, B. Dilettuso, D. Migliorato and W. Randolph. *Cell Tissue Kinet* 6(2):165-172, 1973.

1057 CHANGING CYTOKINETIC PATTERNS OF HUMAN LEUKAEMIC LYMPHOBLASTS DURING THE COURSE OF THE DISEASE, STUDIED *IN VIVO*. AN ADDENDUM ON MATHEMATICAL TREATMENT OF THE DATA. *(E.)* Karle, H. (U. Hosp. Copenhagen, Denmark), P. Ernst, S.-A. Killmann, N. R. Hartmann and E. E. Scheufens. *Br J Haematol* 24(2):231-244, 1973.

1058 KINETICS OF LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKEMIA. I. EQUILIBRIUM BETWEEN BLOOD AND A "READILY ACCESSIBLE POOL". *(E.)* Manaster, J. (Tumor Res. Ctr. Free U. Brussels, Belgium), J. Frühling and P. Stryckmans. *Blood* 41(3):425-438, 1973.

1059 CANCER MORTALITY IN NEW ZEALAND: 4. OTHER PRIMARY SITES. *(E.)* Donovan, J. W. (Dept. Math. Stat., U. Sydney, Australia). *NZ Med J* 75(478): 144-148, 1972.

1060 LEUKAEMIA IN THE RHODESIAN AFRICAN. *(E.)* Jeffrey, C. (Dept. Med., U. Coll. Rhodesia, S. Africa) and M. Gelfand. *J Trop Med Hyg* 75(9):176-179, 1972.

1061 TRENDS IN REDUCING THE MORBIDITY AND MORTALITY FROM CANCER OF THE UTERINE CERVIX. *(Rus.)* Charkviani, L. I. (Sci. Res. Inst. Oncol. Tbilisi, USSR). *Vopr Onkol* 19(1):95-101, 1973.

1062 PROGNOSIS OF MORBIDITY AND MORTALITY FROM CANCER OF THE UTERINE CERVIX. *(Rus.)* Glebova, M. I. (USSR Min. Pub. Hlth., Moscow). *Vopr Onkol* 18(11):35-39, 1972.

1063 POPULATION SIZE AND COMPUTATION OF THE TIME REQUIRED FOR THE GENERATION OF EHRLICH ASCITES CARCINOMA CELLS. *(Rus.)* Il'yina, K. P. (Central Sci. Res. Inst. Roentgenol. Radiol. Leningrad, USSR) and K. Merkle. *Vopr Onkol* 19(1):70-74, 1973.

1064 SPONTANEOUS MAMMARY GLAND TUMOR GROWTH KINETICS IN C3H/He MICE. *(Rus.)* Matiyenko, N. A. (Inst. Cytology Genetics, Novosibirsk, USSR). *Dokl Akad Nauk SSSR* 208(4):963-965, 1973.

1065 RARITY OF TESTIS CANCER IN NEGROES. *(E.)* Tulinius, H. (Intl. Agency Res. Cancer, Lyons, France), N. Day and C. S. Muir. *Lancet* (7793):35-36, 1973.

1066 MORTALITY FROM MALIGNANT TUMORS IN THE MONGOLIAN PEOPLE'S REPUBLIC. *(Rus.)* Dorzhgotov, B. (No affiliation), R. Gur and S. Dorzh. *Vopr Onkol* 19(1):63-65, 1973.

1067 CANCER IN MEXICO. STATISTICS. CAMPAIGN. *(Sp.)* Zuckermann, C. (Natl. Campaign Agains Cancer, Mexico). *Acta Oncol* 9(1):48-55, 1970.

1068 CANCER MORTALITY AMONG THE AMERICAN INDIANS: AN 18 YEAR SURVEY. *(E.)* Creagan, E. T. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 13(March):64, 1972.

See also:

* (Rev): 0602, 0606, 0610, 0625, 0636, 0640, 0643
* (Chem): 0705, 0737
* (Immun): 0963

69 ACID MUCOPOLYSACCHARIDES IN TUMOURS OF THE MYELIN SHEATH CELLS, THE OLIGODENDROGLIOMA ID THE NEURILEMMOMA. *(E.)* Smith, B. (St. Bartholo- w's Hosp., London, England) and M. Butler. *Acta uropathol (Berl)* 23(2):181-185, 1973.

ıe intracellular distribution of acid mucopoly- ıccharides was studied by histochemical methods ;ing paraffin-embedded sections of human oligo- ·ndrogliomas, neurilemmomas and astrocytomas. :aining with alcian blue (pH 1) and high iron .amine, two stains which are relatively specific ›r sulfated mucopolysaccharides, produced staining ı cell walls, cyst fluid or cytoplasm of the ıree tumor types. The staining was unaffected › neuraminidase but was completely abolished by ·aluronidase, thus tentatively identifying the :ainable material as chondroitin sulfate. It ıs postulated that this mucopolysaccharide may ınction in relation to nerve impulse conduction : insulation, possibly by facilitating move- ɜnt of potassium ions across the cell membrane.

70 CYCLIC AMP-INDUCED DIFFERENTIATED MOUSE NEUROBLASTOMA CELLS LOSE TUMOURGENIC IARACTERISTICS. *(E.)* Prasad, K. N. (U. Colorado ·d. Ctr., Denver). *Cytobios* 6(23):163-166, 1972.

ıe tumorigenicity of uncloned neuroblastoma cells ıs studied following s.c. injection into male 'J mice. Untreated, undifferentiated neuroblastoma ·lls produced tumors in all 30 animals following ıoculation. However, cells treated *in vitro* .th prostaglandin E_1 in combination with dibutyryl ·clic AMP or 8-benzylthio cyclic AMP, substances ıich induce morphological differentiation, failed › induce tumors by 60 days following inoculation. :perimental animals showed a longer latent period ıd lower tumor incidence than the controls. Agents ıich increase the endogenous level of cyclic AMP ıy, therefore, be potentially important drugs ı the treatment of neuroblastoma.

71 ASTROCYTOMA ARISING IN MENINGEAL FIBRO-SARCOMA. *(E.)* Henry, J. M. (Armed Forces ıst. Path., Washington, D. C.) and J. E. Leestma. *ɜta Neuropathol (Berl)* 23(4):334-337, 1973.

rare case of mixed glioma-sarcoma arising in a rimary meningeal fibrosarcoma was observed in a 5-year-old Negro male. The mixed tumor, which ıs located in the right middle cerebral fossa t the sphenoid ridge, grossly resembled a fibro- arcoma. Microscopic observation revealed a arcomatous element which contained well-defined ɜsts of loose, randomly oriented neoplastic glial ells. There was minimal mitotic activity. The esion was interpreted as representing a fibro- arcoma of meningeal origin in association with secondary glioma, a situation analogous to hat previously reported by Rubinstein and unlike he more common situation reported by Feigin in hich sarcoma arises from hyperplastic endothelium ithin a primary glioblastoma. It is suggested that the term "gliosarcoma" be reinstated to describe these mixed tumors.

1072 HISTOCHEMICAL OBSERVATIONS ON NDPase AND TPPase IN CEREBRAL TUMOURS. *(E.)* Schiffer, D. (Clin. Nervous Mental Dis., U. Turin, Italy). *Acta Neuropathol (Berl)* 23(2):112-117, 1973.

Slices of fixed tissue from biopsies of 24 human cerebral tumors were studied by a histochemical method for thiamine pyrophosphatase (TTPase) and nucleoside diphosphatase (NDPase), two marker enzymes for the Golgi apparatus. Using TPP as substrate, hydrolysis in the presence of Ca^{++} and, especially, Mg^{++} was of moderate intensity such that small structures tentatively identified as the Golgi apparatus could be discerned in the cytoplasm of glioma tumor cells. However, hydrolysis of TPP in the presence of Mn^{++} and of inosine diphosphate (IDP) in the presence of Ca^{++}, Mg^{++} or Mn^{++} gave such intense and diffuse cellular staining that no intracellular organelles were apparent. Intracellular staining of non-glial tumor cells with either TPP or IDP as substrate was very weak or absent. Histochemical localization of TPPase may thus be of practical importance in the study of the Golgi apparatus of tumor cells.

1073 SIMILARITY OF THE ESTROGEN RECEPTORS IN HUMAN AND RAT MAMMARY CARCINOMA. *(E.)* McGuire, W. L. (Dept. Med., U. Texas, San Antonio) and M. Delagarza. *J Clin Endocrinol Metab* 36(3): 548-552, 1973.

The properties of human and rat mammary tumor cytoplasmic estradiol binding protein (EBP) have been compared. Sephadex column chromatography of human tumor cytosol preparations preincubated with estradiol showed that human tumor EBP, as had previously been shown for rat tumor EBP, could be demonstrated by this method. Low-salt sucrose density gradient centrifugation of human tumor cytosol preincubated with ^{3}H-estradiol revealed radioactive peaks migrating as 8S and 4S particles. All the label in the 8S peak and most of the label in the 4S peak were eliminated by competition with unlabeled estradiol, indicating that both peaks contained EBP. These properties were identical to those of rat mammary tumor EBP. Like rat tumor EBP, human mammary tumor EBP was precipitated by protamine sulfate. Human tumor EBP binding was specific for estrogens as progesterone, testosterone, hydrocortisone and dihydrotestosterone failed to compete significantly with estradiol.

1074 DEOXYRIBONUCLEOTIDE POOLS DURING LIVER REGENERATION. *(E.)* Söderhäll, S. S. (Karolinska Inst. Stockholm, Sweden), A. Larsson and K. L. Skoog. *Eur J Biochem* 33(1):36-39, 1973.

Intracellular pools of deoxyribonucleoside triphosphates were analyzed in Sprague-Dawley rats after partial hepatectomy. During the first 12 hr after the operation, the liver content of dATP, dCTP, dGTP and dTTP remained constant at 0.3, 1.3, 0.3 and 0.4 pmol/μg DNA, resp. When the remaining liver tissue started to proliferate the four nucleotide pools increased and reached maxima at 24-27 hr, i.e., the time of peak DNA synthesis as measured by incorporation of labeled thymidine. Mitotic activity reached a maximum of 8% at 32 hr. Ribonucleotide reductase activity began to increase at 15 hr and was maximal at 50 hr, i.e., when the four triphosphate pools and rate of DNA synthesis had declined. This indicated a feed-back control of the enzyme.

1075 CORRELATION BETWEEN TUMOR INVASIVENESS AND CONTENT OF A-TYPE PARTICLES IN EHRLICH ASCITES TUMOR: A MODEL OF QUANTITATIVE ELECTRON MICROSCOPY. *(E.)* Kodama, T. (Aichi Cancer Ctr. Res. Inst., Nagoya, Japan) and M. Kodama. *J Natl Cancer Inst* 50(3):707-712, 1973.

The ultrastructure of Ehrlich ascites tumor in dd female mice in relation to tumor invasiveness was investigated using an electron microscope for the study of cytoplasmic structure and a superscope for measurement of cell and nuclear diameters. In each of four hypotetraploid clones and the one hyperdiploid stock tumor, the incidence of intracisternal A particles in tumor cells increased with tumor invasiveness (an inverse of survival time of mice), and the correlation was statistically significant. The number of A particles in a single tumor cell, calculated by stereologic rearrangement of data, ranged from 101 for Ehrlich 2N tumor to 15,600 for clone 4 (4N). A-type particle incidence in normal thymus, liver, and lactating mammary gland was significantly lower than in the 2N tumor. The three-dimensional conversion of the electron micrographs was based on the assumed geometric thickness of the section, which actually accounted for the particle counts observed.

1076 PRODUCTION OF HUMAN CHORIONIC GONADOTROPIN *IN VITRO* BY A CELL LINE DERIVED FROM A CARCINOMA OF THE LUNG. *(E.)* Rabson, A. S. (Natl. Cancer Inst., Bethesda, Md.), S. W. Rosen, A. H. Tashjian, Jr. and B. D. Weintraub. *J Natl Cancer Inst* 50(3):669-674, 1973.

An epithelial cell line was derived from tumor tissue of a lung carcinoma patient who had evidence of human chorionic gonadotropin (hCG) secretion. *In vitro* production by cell cultures of a biologically active gonadotropin immunochemically indistinguishable from hCG was demonstrated by radioimmunoassay. Sections of cell pellets have morphological features similar to those of the original tumor. The cells grow both in suspension and adherent to plastic surfaces, and continue to produce hCG after 19 months in culture.

1077 FAMILIAL OCCURRENCE OF EXTRAMAMMARY PAGET'S DISEASE. *(E.)* Kuehn, P. G. (Hartford Hosp., Conn.), R. Tennant and A. R. Brenneman. *Cancer* 31(1):145-148, 1973.

Two cases of extramammary Paget's disease occurring within the same family are reported. The first was in a 66-yr-old Caucasian male who was admitted to Hartford Hospital in 1949 for investigation of a chronic eczematous lesion of the right scrotal area with palpable nodes in the right groin. Biopsy resulted in a diagnosis of extramammary Paget's disease and the lesion was excised surgically. Local recurrence was discovered one year later followed by metastases to the tibia and brain. The patient expired after two years of illness. The patient's son presented in 1971 at the age of 58 with a weeping raised red eczematoid lesion of the right scrotal area which had been present for a year. Skin biopsy again produced a diagnosis of extramammary Paget's disease. The tumor was excised surgically, and to date there has been no recurrence. In both cases, the lesion consisted of clusters of epithelial cells with clear cytoplasm and fine vesicular nuclei located within the confines of the epidermis. A search of the literature revealed no previous record of such a familial occurrence of extramammary Paget's disease. This finding suggests the possibility of a genetic basis for the disease.

1078 CATENATED OLIGOMERIC CIRCULAR DNA MOLECULES FROM MITOCHONDRIA OF MALIGNANT AND NORMAL MOUSE AND RAT TISSUES. *(E.)* Wolstenholme, D. R. (Div. Biol., Kansas State U., Manhattan), J. D. McLai K. Koike and E. L. Jacobson. *J Cell Biol* 56(1):247-255, 1973.

Mitochondrial DNA (mtDNA) was isolated from cells of solid or ascites hepatomas and normal liver of mice and rats. The frequencies of multiple circular forms of DNA molecules were determined by electron microscopy. More than 90% of at least one preparation of mtDNA from malignant and normal tissues was in circular form. Forms comprising two attached 5 μm circles were found in all tissues examined. Between 6-8% of normal mouse liver mtDNA was in this form compared with 15-22% of Ehrlich mouse hepatoma mtDNA. In addition, up to 19% of Ehrlich hepatoma mtDNA was in the form of molecules comprising 3-7 joined 5 μm circles. Such higher oligomers were not found or were rare in mtDNA from normal mouse liver. MtDNA from Novikoff and Chang rat hepatomas were distinguished from normal rat liver mtDNA by the presence of tetrameric and pentameric forms.

1079 MONOBLAST OF ACUTE MONOBLASTIC LEUKEMIA. *(E.)* Schumacher, H. R. (Harrisburg Hosp., Pa.), I. E. Szekely and S. A. Park. *Cancer* 31(1): 209-220, 1973.

An ultrastructural study was conducted on marrow myeloblasts obtained from 14 normal hospital personnel, on leukemic myeloblasts from six patients with acute myeloblastic or myelomonoblastic leukemia, and on monoblasts from 14 patients with various types of acute monoblastic anemia.

asurements of nuclear length, nuclear area, total
l area, cytoplasmic area and total cell length
wed no difference among the three cell types
h respect to these parameters. Both the
ukemic myeloblasts and monoblasts possessed a
ber of morphologic characteristics which
tinguished them from normal myeloblasts. These
cluded nuclear pockets, deep nuclear invaginations,
ranuclear particles, nucleosomes, dilated
inuclear spaces, centrioles located in nuclear
kets, striking accumulation of microfibrils,
rupted mitochondria containing 1000 Å virus-
e particles, smaller nuclear and mitochondrial
rticles, intramitochondrial 25 Å fibers (possibly
A), glycogen and myelin whorls. The Golgi zone
the leukemic monoblasts was more frequently
monstrable and larger than that of the other two
ll types. The endoplasmic reticulum (ER) was
st developed in the leukemic myeloblasts, with
ese cells containing a fair amount of smooth ER
ich was not present in normal myeloblasts.
ukemic monoblasts contained a significantly
eater quantity of polyribosomes than either
e leukemic or normal myeloblasts.

30 VARIATIONS IN INTERFERON PRODUCTION BY
LYMPHOCYTES FROM PATIENTS WITH CHRONIC
MPHATIC LEUKEMIA. *(E.)* Mackenzie, A. M. R.
adcliffe Infirm., Oxford, England). *J Clin
thol* 25(9);768-771, 1972.

terferon production by leukocytes was assayed
14 chronic lymphatic leukemia patients and
und to be significantly lower in patients
an in healthy subjects. In addition, a
gnificant inverse correlation was found between
ripheral blood lymphocyte count and interferon
oduction in leukemia patients. These results
ovide indirect evidence that thymus-dependent
) lymphocytes are more competent producers of
terferon than non-thymus dependent (B)
mphocytes.

81 CLINICAL AND EXPERIMENTAL TUMORS OF THE
KIDNEY IN TISSUE CULTURE AND IN THE
ICK EMBRYO. *(E.)* Leighton, J. (Med. Coll.
nnsylvania, Philadelphia), S. Mansukhani and G.
Fetterman. *Eur J Cancer* 8(3):281-285, 1972.

periments with *in vitro* cultivation of Wilm's
mor and MDCK, a line of cells derived from a
rmal dog kidney, are described. Fresh Wilm's
mor material as well as freeze-stored material
ew on chick chorioallantois (CAM) when tumor
terial was mixed with normal chick embryo
ssues. Wilm's tumor on CAM grew as anaplastic
lls in nodules containing tumor stroma and
bules. MDCK cells grew as papillary adenocar-
nomas on CAM. MDCK cells on CAM showed one of
o different patterns of cell polarization; the
ttern depended on the cell microenvironment.

82 FURTHER STUDIES ON THE FREQUENCY OF CONSTITUTIONAL CHROMOSOME ABNORMALITIES IN PATIENTS WITH MALIGNANT DISEASE. *(E.)* O'Riordan, M. L. (Western Gen. Hosp., Edinburgh, Scotland), A. O. Langlands and D. G. Harnden. *Eur J Cancer* 8(4):373-379, 1972.

Venous blood samples from 3068 cancer patients were examined karyologically. Patients included individuals with tracheo-bronchial cancer, mammary cancer, oral cancer and cancers of other sites. Twenty patients were found to have constitutional chromosome aberrations; eight patients had abnormal sex chromosomes and 12 had autosomal abnormalities. Of autosomal aberrations, five were Robertsonian translocations. No one site of cancer monopolized chromosomal abnormalities and the incidence of aberrations in cancer patients was similar to that in the general population.

1083 RELATIONSHIP BETWEEN RNA METABOLISM AND CELL CYCLE IN ACUTE LEUKEMIA. *(E.)* Quaglino, D. (Inst. Med. Path., U. Modena, Italy), G. Emilia, A. de Pasquale, E. Ferreira and C. Mauri. *Acta Haematol (Basel)* 47(6):326-334, 1972.

Cell size measurements and measurements of RNA synthesis (measured as uptake of ^{3}H-uridine by cells) were performed on cultured normal cells and cells from eight acute leukemia patients (bone marrow cultures). In normal lymphocytes, ^{3}H-uridine incorporation and extent of cytoplasmic labeling by radioactive precursor depended on cell size, smaller cells being less heavily labeled than larger blast cells. However, normal blasts showed only a narrow range of variation in cell size and uridine incorporation. In contrast, leukemic blast cells showed ample variations both in cell size and in uridine incorporation. Larger leukemic blast cells showed a greater degree of nuclear-cytoplasmic transfer of ^{3}H-uridine and also a higher rate of ^{3}H-thymidine incorporation than smaller leukemic blasts.

1084 *IN VITRO* GROWTH OF LUCKÉ RENAL TUMOR CELLS. *(E.)* Zambernard, J. (Albany Med. Coll., N.Y.). *J Natl Cancer Inst* 50(2):557-578, 1973.

Cells from three primary Lucké renal tumors (LRT or Leopard frog renal adenocarcinoma) were established *in vitro*. Cells from one tumor were subcultured through 10 generations without any signs of cellular degeneration. This success was attributed to the use of highly purified trypsin (2X crystallized, dialized, salt-free, and lyophilized) for dissociating primary tumor cells and subsequent confluent cell monolayers.

1085 DIHYDROOROTATE DEHYDROGENASE IN LIVER AND MORRIS HEPATOMAS. *(E.)* Sweeney, M. J. (Lilly Res. Lab., Eli Lilly Co., Indianapolis, Ind.) and D. H. Hoffman. *Cancer Res* 33(2):394-396, 1973.

Dihydroorotate dehydrogenase (DHODH) activity in the 3683, 3924A, 7288C, and 5123D Morris hepatomas was one-half that of normal rat liver. However, the DHODH activity in Morris hepatoma 7800 was the same as that of rat liver. No correlation existed between the DHODH activities and the growth rates of the hepatomas. The K_m's of the DHODH of the hepatomas ranged from 2.5 to 11.5 X 10^{-5} M and were similar to the K_m's of 4.2 X 10^{-5} M for liver from Buffalo rats and 8.25 X 10^{-5} M for liver from ACI rats. Both rat livers and the five hepatomas had a pH optimum of 8.05 ± 0.1 for DHODH. The activity of DHODH from liver was not influenced by fasting, refeeding, or partial hepatectomy in Buffalo and ACI rats, except for an increase in the livers of fasting ACI rats.

1086 CONTROL OF NORMAL DIFFERENTIATION OF MYELOID LEUKEMIC CELLS TO MACROPHAGES AND GRANULOCYTES.

(E.) Fibach, E. (Weizmann Inst. Sci., Rehovot, Israel), M. Hayashi and L. Sachs. *Proc Natl Acad Sci USA* 70(2):343-346, 1973.

Cultured myeloblasts from a myeloid leukemia of SL mice were treated with MGI, a protein required for induction of macrophage and granulocyte development. MGI in culture medium increased cloning efficiency of leukemic cells from 2% to 15%. About 60% of the colonies formed after MGI contained only undifferentiated blast cells (D- colonies); 40% of colonies (D+) contained differentiating cells. D+ colonies differentiated to either macrophages or granulocytes. In general, D+ colonies produced D+ clonal progeny and D- colonies produced D- clonal progeny. In some cases, however, 40 cell generations after clonal isolation, D+ clones segregated D- progeny and vice versa.

1087 GLYCOLIPID SYNTHESIS AND TUMORIGENICITY OF CLONES ISOLATED FROM THE NIL 2 LINE OF HAMSTER EMBRYO FIBROBLASTS.

(E.) Sakiyama, H. (Dept. Biol., Massachusetts Inst. Tech., Cambridge) and P. W. Robbins. *Fed Proc* 32(1):86-90, 1973.

A number of clones isolated from the Nil 2 hamster line were found to vary in morphology, saturation density, and glycolopid composition. The tumorigenicity of these clones was examined by injecting cells subcutaneously into golden hamsters. Nil 2C1 contained Forssman glycolipid (GL-5), globoside (GL-4), trihexosyl ceramide (GL-3), and hematoside (GM_3). Nil 2C2 contained GL-5 and GM_3 while Nil 1C1 contained GM_3 only. These glycolipids increased on confluence in Nil 2C1 and Nil 2C2 cells. The amount of GM_3 increased at confluence in Nil 1C1 cells. Nil 2C2 cells were most and Nil 1C1 cells were least oncogenic. Tumors produced by these clones were recovered and grown *in vitro*. The glycolipid patterns were examined again. Tumor lines derived from Nil 2C1 lost GL-3 and the amount of GL-5 and GL-4 decreased significantly. In tumor lines derived from Nil 1C1 cells, the amount of GM_3 decreased. Moreover, in both cases the glycolipids no longer showed the density-dependent increase in confluent cultures. In the case of Nil 2C2 no significant change in the glycolipid pattern was found in tumor lines. Tumor lines were injected back into hamsters to examine their tumorigenicity. Tumor cells induced by Nil 1C1 and Nil 2C1 increased in tumorigenicity significantly. The tumorigenicity of the tumor cells induced from Nil 2C1 cells was not changed. The agglutinability of the clones and tumor lines by concanavalin A and wheat-germ agglutinin was examined. No correlation between agglutinability and tumorigenicity was found.

1088 ENZYMATIC ACTIVITIES MODIFIED DURING MULTIPLICATION AND DIFFERENTIATION OF NEUROBLASTOMA CELLS.

(E.) Ciesielski-Treska, J. (Neurochem. Ctr., CNRS, Strasbourg, France), P. Mandel, G. Tholey and B. Wurtz. *Nature New Biol* 239(93):180-181, 1972.

The activities of fructose disphosphate aldolase (ALD), glucose-6-phosphate dehydrogenase (G-6PD), lactate dehydrogenase (LDH), glutamate dehydrogenase (GDH), and malate dehydrogenase (MDH) were determined in fresh neuroblastoma C-1300 cells from A/J mice, in proliferating cells, and in differentiating cells. The activities of all five enzymes were higher in cells grown in the presence of serum than in cells not grown in culture. Withdrawal of serum from the culture medium was followed by growth in most cells. These morphological modifications were accompanied by a decrease of LDH activity and increases of MDH and GDH activities. No significant changes of ALD and G-6PD activities were observed. The similarity of ALD and G-6PD activities suggests that glucose metabolism occurs at a similar rate whether cells multiply or differentiate. However, the decrease of LDH activity and the increase of MDH and GDH activities suggest that the Krebs cycle is more active. These results are thus in good agreement with the higher oxygen uptake in differentiating cells.

1089 CHARACTERIZATION OF TUMOR CELL POPULATIONS BY DNA-MEASUREMENTS.

(E.) Pfitzer, P. (Inst. Path., U. Düsseldorf, W. Germany) and H. D. Pape. *Acta Cytol (Baltimore)* 17(1):19-25, 1973.

Nuclear DNA content in AFi-H cells derived from a human fibrosarcoma, primary oral cavity tumors and diffusions with metastatic tumor cells was determined by scanning cytophotometry. The chromosomal stemline of the AFi-H line was 63 and the karyotype contained at least 11 marker chromosomes. The DNA content of interphase nuclei followed a bimodal distribution with a very large spread. Bipolar mitoses showed two maxima representing ana- and metaphases. Five different DNA distribution profiles were obtained for the nine primary tumors of the oral cavity. These were characterized by polyploid values, by a large spread, by aneuploidy or by a combination of these characteristics. Only one case showed none of them. Generally nuclear size increased with polyploidy, but there was no clear separation of size classes corresponding to DNA values. The same characteristics applied to ten diffusions in

h polyploidy was even more pronounced. No efflu-
n had less than three maxima which in a few cases
ained 32 C. All effusions due to systemic
gnant disease had 4 C nuclei.

0 DNA CONTENT AND CHROMATIN PATTERN ANALYSIS
ON CERVICAL CARCINOMA *IN SITU*. *(E.)*
enger, E. (Dept. Path., U. Freiburg, W. Germany),
. Moore, H. Naujoks, G. Schlüter and W. Sandritter.
Cytol (Baltimore) 17(1):27-31, 1973.

ee cases of cervical carcinoma *in situ* with pre-
inantly positive sex chromatin, negative sex
omatin or coarsely granulated abnormal cell nuclei
subjected to Feulgen DNA cytophotometry and
lear image analysis. Both the positive and
ative sex chromatin abnormal populations showed
aneuploid histogram of Feulgen-DNA distribution.
lear image analysis indicated that chromatin
itive and chromatin negative nuclei possessed
ore or less equal proportion of condensed chroma-
with a tendency toward peripheral positioning.
contrast, clumped chromatin nuclei showed a
nificantly higher proportion of chromatin dense
ions and a tendency toward central positioning
this chromatin.

1 DIFFERENCES IN THE METHYLATION OF TRANSFER
RIBONUCLEIC ACID *IN VITRO* BY THE MITOCHONDRIAL
) CYTOPLASMIC TRANSFER RIBONUCLEIC ACID METHYLASES
HeLa CELLS. *(E.)* Klagsbrun, M. (Natl. Inst.
hritis, Metabolism, Digestive Dis., Bethesda, Md.).
iol Chem 248(7):2606-2611, 1973.

methylation of *Escherichia coli* B transfer RNA
vitro by HeLa mitochondrial and cytoplasmic ex-
cts was examined. The following differences were
nd. (*a*) The cytoplasmic extract methylated *E.*
i B tRNA to a much greater extent than did the
ochondrial extract. (*b*) The two extracts methylated
fferent sites on *E. coli* B tRNA, thereby generating
fferent methylated oligonucleotide sequences. (*c*)
methylated base composition of *E. coli* B tRNA
hylated *in vitro* indicated that 2-methyladenine
A methylase activity was uniquely associated with
mitochondria, while N^2-dimethylguanine tRNA
hylase activity was uniquely associated with the
oplasm. The presence of 2-methyladenine tRNA
hylase activity in HeLa mitochondria and its
ence in HeLa cytoplasm was taken as evidence for
existence of at least one mitochondrial-specific
hylase. In addition, since 2-methyladenine is
monly found in prokaryote tRNA but not in eukaryote
toplasmic tRNA, it was suggested that the presence
2-methyladenine tRNA methylase activity in mito-
ndria was evidence that mitochondria might have
olved from prokaryote cells.

2 METABOLISM OF ANDROGENS BY HUMAN BREAST
TISSUE. *(E.)* Miller, W. R. (Royal Infirm.,
inburgh, Scotland), D. McDonald, A. P. M. Forrest
A. A. Shivas. *Lancet* (7809):912-913, 1973.

idence for the conversion of dehydroepiandrosterone (DHA) through testosterone to 5α-dihydrotestosterone was sought in three breast cancers, two fibroadenomas, one fibroadenosis and six specimens of normal breast tissue from the same patients. Significant *in vitro* activity in all tissues was demonstrated by measuring the incorporation of 7-^{3}H DHA and 4-^{14}C testosterone into metabolites. Both benign and malignant tumors transformed DHA to testosterone and reduced testosterone to 5α-dihydrotestosterone. 5α-Reductase activity was markedly increased in the fibroadenomas, and to a lesser extent in the normal breast associated with them, over that in the tumors and normal tissues from other patients.

1093 ULTRASTRUCTURAL TRANSFORMATION OF A MURINE LEYDIG-CELL TUMOR AFTER GONADOTROPIN ADMINISTRATION. *(E.)* Neaves, W. B. (U. Texas Southwestern Med. Sch., Dallas). *J Natl Cancer Inst* 50(4):1069-1073, 1973

The fine structure of a transplantable Leydig-cell tumor (M548) was examined by electron microscopy before and after s.c. injection of tumor-bearing C57BL/6J mice with 50 IU chorionic gonadotropin. Under normal *in vivo* conditions, tumor cells were poorly differentiated and contained small quantities of smooth endoplasmic reticulum (SER), an organelle notably abundant in mature testicular Leydig cells. After gonadotropin administration, SER became the most prominent organelle in many tumor cells. Contorted, branching tubules and fenestrated cisternae of smooth SER filled large areas of peripheral cytoplasm and caused the transformed tumor cells to resemble typical mouse Leydig cells. This finding suggests that appropriate hormones can cause relatively undifferentiated neoplasms to develop structural and functional characteristics of their cells of origin.

1094 PARALLEL CORRECTION OF CANCEROUS GROWTH AND OF A GENETIC DEFECT OF CELL-TO-CELL COMMUNICATION. *(E.)* Azarnia, R. (U. Miami Sch. Med., Fla.) and W. R. Loewenstein. *Nature* 241(5390): 455-457, 1973.

Noncoupling cells from a cancerous epithelial strain derived from a rat liver tumor (A cells) were fused by Sendai virus with normal and/or mutant fibroblast cells derived from the same tumor and with cells from a normal liver epithelial line. Junctional communication (coupling) in the primary products of cell fusion and their descendents was measured electrically and by fluorescent tracer diffusion. The primary hybrids, like their normal parents, were coupled, contact inhibited, and nontumorigenic. Growth of noncoupling parental A cells was not contact inhibited (cells grew to densities of 10^6 cells/sq cm in culture), and the cells were highly tumorigenic (inocula of 10^2 cells produced fatal tumors in Buffalo rats). The junctional defect was also corrected in descendents of the primary hybrids, including eight clones of cancer cell/normal (mutant) fibroblast hybrids and five clones of cancer cell liver epithelial cell hybrids. Correction of the junctional defect, which was paralleled by the correction of growth defect, appeared due to the normal cell genome in hybrids and not simply to cell cultivation or cell fusion. These results suggest that defective junctional coupling may be an etiological factor of certain kinds of cancer.

1095 HUMAN SPLEEN INHIBITOR OF LEUKAEMIC CELL GROWTH.

(E.) Lozzio, B. B. (U. Tennessee Mem. Res. Ctr. Hosp., Knoxville), C. B. Lozzio and E. G. Bamberger. *Experientia* 29(1): 87-89, 1973.

Spleens were removed from two chronic myelocytic leukemia (CML) patients and a splenic inhibitor (SI) fraction was isolated from the spleen cells. The SI appeared to be a small peptide or glycopeptide. It did not contain proteins, hexosamines and pentoses, and had a molecular wt of less than 1000 daltons. At SI doses above 1.0 mg/ml, the SI had a lethal effect on cultured CML cells, a 5 mg/ml dose killing all CML cells by 48 hr after SI treatment. The SI diminished DNA synthesis in CML cells 24 hr after treatment.

1096 PYRUVATE DEHYDROGENASE, THE CITRATE CONDENSING ENZYME AND THE UTILIZATION OF ^{14}C-LABELED LACTATE, PYRUVATE AND ALANINE BY SLICES OF LACTATING MAMMARY GLAND AND ADENOCARCINOMA OF MOUSE MAMMARY GLAND.

(E.) Kopelovich, L. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.). *Proc Soc Exp Biol Med* 142(3):954-962, 1973.

The oxidative decarboxylation patterns of lactate, pyruvate and alanine were studied in tissue slices of lactating C3H mouse mammary gland and mammary adenocarcinoma and were correlated with activity of the enzymes directly responsible for the entry of the substrates into the Krebs cycle. $^{14}CO_2$ production from exogenously supplied ^{14}C-labeled lactate, pyruvate and alanine was greater in the lactating gland than in adenocarcinoma. Addition of glucose to the incubation medium greatly stimulated $^{14}CO_2$ production by lactating mammary gland tissue but decreased production by mammary adenocarcinoma. The rates of formation of fatty acids from lactate, pyruvate and alanine were also greater in lactating mammary gland. Chromatographic analysis of ^{14}C-lactate-derived amino acids showed that formation of alanine, glutamate and aspartate was considerably greater in adenocarcinoma slices than in lactating mammary glands. *In vitro* enzyme assays with fractionated cell homogenates indicated that the activity of pyruvate dehydrogenase activity was three-fold and citrate condensing enzyme activity was seven-fold higher in lactating mammary gland than in mammary adenocarcinoma. The lower enzyme activity in the neoplasm was not due to an inhibitor.

1097 SUBCELLULAR PARTICLES IN TUMORS--V. BEHAVIOR OF MITOCHONDRIA OF HEPATOMA HC AND MORRIS HEPATOMAS 7316A, 7794B AND 16 IN DENSITY-GRADIENT CENTRIFUGATION.

(E.) Wattiaux-de Coninck, S. (Fac. U. Notre Dame Paux, Namur, Belgium), M. Collot, R. Wattiaux and H. P. Morris. *Eur J Cancer* 8(4): 415-420, 1972.

The distribution of mitochondrial enzymes has been investigated after isopycnic centrifugation of mitochondrial fractions of HC hepatoma and Morris hepatomas 7794B, 7316A and 16. Two kinds of density gradient were used, one consisting of glycogen dissolved in aqueous sucrose of different molalities and the second of aqueous sucrose alone. In sucrose gradient experiments, the distributions of cytochrome oxidase, monoamine oxidase and glutamate dehydrogenase were established. Isopycnic centrifugation was performed at two speeds, 39,000 rev/min and 65,000 rev/min. At 39,000 rev/min, a similar distribution was seen for the three enzymes in each hepatoma; however, the distribution exhibited marked differences from one hepatoma to the next. The distributions of the three mitochondrial enzymes of hepatomas 16 and HC remained practically unchanged when centrifugation was performed at 65,000 rev/min instead of 39,000 rev/min. In contrast, the higher centrifugation speed did induce changes in the distributions of hepatoma 7316A and 7794B enzymes. In glycogen gradient experiments, cytochrome oxidase distribution was studied. The distribution indicates that the behavior of mitochondria of hepatoma 7794B, 7316A and HC in such gradients is qualitatively similar to that of rat liver mitochondria. However, the parameters that characterize the intramitochondrial spaces, calculated from the distribution pattern, differ to a variable extent from those calculated for rat liver particles.

1098 TURNOVER OF LABELED METHYL GROUPS IN HISTONES.

(E.) Byvoet, P. (Dept. Path., U. Florida, Gainesville), G. R. Shepherd, J. M. Hardin and B. J. Noland. *Proc Am Assoc Cancer Res* 13(March):59, 1972.

1099 THE SUBCELLULAR DISTRIBUTION OF ETHER LIPIDS IN TUMORS.

(E.) Lee, T.-C. (Med. Division, Oak Ridge Natl. Lab., Tenn.), C. Piantodosi and F. Snyder. *Proc Am Assoc Cancer Res* 13(March):58, 1972.

1100 PARTICLES, ABOUT 65 mμ IN DIAMETER, IN THE NUCLEI OF HUMAN BONE TUMOR CELLS.

(E.) Lloyd, E. (Argonne Natl. Lab., Ill.) and F. Mackevicius. *Proc Am Assoc Cancer Res* 13(March):55, 1972.

1101 THE SIGNIFICANCE OF PRIMITIVE CELLS (? LYMPHOBLASTS) IN MARROW ASPIRATES OF CHILDREN WITH NEUROBLASTOMA.

(E.) Evans, A. E. (Children's Hosp. Philadelphia, Pa.) and K. Hummeler. *Proc Am Assoc Cancer Res* 13(March):54, 1972.

1102 ACID PHOSPHATASE ISOZYMES IN CANCER OF THE PROSTATE.

(E.) Reif, A. E. (Tufts U. Sch. Med., Boston, Mass.), R. M. Schlesinger

l C. A. Fish. *Proc Am Assoc Cancer Res* 13(March): , 1972.

03 CHANGES IN CELL MEMBRANE PERMEABILITY BEFORE, DURING AND AFTER THE ESTABLISHMENT LEUKEMIC AND NONLEUKEMIC HUMAN BLOOD CELL LINES. *)* Rosenfeld, C. (Paul-Brousse Hosp., Villejuif, ance) and J. P. Wastiaux. *Proc Am Assoc Cancer* ; 13(March):47, 1972.

04 NEW MACROMOLECULAR LIPIDS FROM L1210 LEUKEMIC CELLS. *(E.)* Yesair, D. W. ist. Chem. Natural Substances, Gif-sur-Yvette, ance), E. Lederer and C. J. Kensler. *Proc Am soc Cancer Res* 13(March):45, 1972.

05 PRIMARY EXPLANTATION AND CONTINUOUS CULTIVATION OF HUMAN MAMMARY CARCINOMA. *(E.)* sfargues, E. Y. (Inst. Med. Res., Camden, N.J.), G. Coutinho and D. H. Moore. *Proc Am Assoc ncer Res* 13(March):42, 1972.

06 SELENIUM IN THE BLOOD OF NORMALS AND CANCER PATIENTS. *(E.)* Shamberger, J. (Cleveland Clin. Fdn., Ohio), E. F. Rukovena l C. E. Willis. *Proc Am Assoc Cancer Res* (March):41, 1972.

07 GLYCOGEN SYNTHETASES AND PHOSPHORYLASES IN RAT HEPATOMAS. *(E.)* Sato, K. emple U. Sch. Med., Philadelphia, Pa.), S. inhouse and H. P. Morris. *Proc Am Assoc Cancer* ; 13(March):27, 1972.

08 BIOCHEMICAL STUDIES OF HUMAN BREAST CANCER. *(E.)* Hilf, R. (U. Rochester d. Sch., N.Y.), J. L. Wittliff, W. D. Rector d R. A. Orlando. *Proc Am Assoc Cancer Res* (March):24, 1972.

09 ALTERATION OF RNA METABOLISM IN VITAMIN A DEFICIENT TRACHEAL EPITHELIUM. *(E.)* ufman, D. G. (Natl. Cancer Inst., Bethesda, Md.), S. Baker, M. B. Sporn and U. Saffiotti. *Proc Assoc Cancer Res* 13(March):23, 1972.

10 ELEVATION OF PROLINE HYDROXYLASE ACTIVITY IN SPONTANEOUS PRIMARY MAMMARY TUMORS OF BRED MICE. *(E.)* Cutroneo, K. R. (Med. Coll. orgia, Augusta) and A. G. Liebelt. *Proc Am soc Cancer Res* 13(March):22, 1972.

11 SPECIFIC ESTROGEN-BINDING CAPACITY OF AN ESTROGEN-RESPONSIVE MAMMARY CARCINOMA THE RAT. *(E.)* Gardner, D. G. (U. Rochester h. Med. Dentistry, New York), W. L. Battema and J. Gilbert. *Proc Am Assoc Cancer Res* 13(March): , 1972.

1112 CELL KINETIC STUDIES IN PATIENTS WITH SMALL-CELL CARCINOMA OF THE LUNG. *(E.)* Muggia, F. M. (VA Hosp., Washington, D.C.), S. K. Oster and H. H. Hansen. *Proc Am Assoc Cancer Res* 13(March):16, 1972.

1113 CYCLIC AMP AND THE DIFFERENTIATION OF NEUROBLASTOMA CELL CULTURE. *(E.)* Prasad, K. N. (Dept. Radiol., U. Colorado Med. Ctr., Denver) *Proc Am Assoc Cancer Res* 13(March):16, 1972.

1114 EFFECTS OF COLONY STIMULATING FACTOR (CSF) UPON MACROMOLECULAR SYNTHESIS IN MOUSE BONE MARROW CULTURES. *(E.)* Ross, D. D. (Emory U., Atlanta, Ga.) and D. P. Groth. *Proc Am Assoc Cancer Res* 13(March):14, 1972.

1115 RIBONUCLEASE ACTIVITY IN CROWN GALL OF TOBACCO. *(E.)* Simard, A. (Cancer Inst., Montreal, Canada). *Proc Am Assoc Cancer Res* 13(March):41, 1972.

1116 BIOCHEMICAL ORGANIZATION OF NOVIKOFF HEPATOMA MITOCHONDRIA. *(E.)* White, M. T. (Dept. Molecular Biol. Biochem., U. California, Irvine) and K. K. Tewari. *Proc Am Assoc Cancer Res* 13(March):36, 1972.

1117 LONG TERM GROWTH OF CHICK EMBRYO CELLS. *(E.)* Gey, G. O. (Johns Hopkins U., Finney Howell Lab., Baltimore, Md.), M. Svotelis, F. B. Bang and M. Foard. *Proc Am Assoc Cancer Res* 13(March):35, 1972.

1118 CULTIVATION OF CELLS FROM A PLEURAL EFFUSION DERIVED FROM A HUMAN BREAST CARCINOMA. *(E.)* Vazquez, J. (Michigan Cancer Fdn., Detroit), S. Albert, A. Long and H. Soule. *Proc Am Assoc Cancer Res* 13(March):34, 1972.

1119 FETAL-TYPE ISOENZYMES IN RAT TUMORS. *(E.)* Knox, W. E. (New England Deaconess Hosp., Boston, Mass.), F. Farron and H. H. T. Hsu. *Proc Am Assoc Cancer Res* 13(March):34, 1972.

1120 POOL SIZE ATP, ADP, AND AMP DURING THE MAMMALIAN CELL CYCLE. *(E.)* Murphee, S. (U. Texas M. D. Anderson Hosp., Houston) and E. C. Moore. *Proc Am Assoc Cancer Res* 13(March): 28, 1972.

1121 EFFECT OF NORMAL IODINE DIET ON THE THYROID GLAND OF IODINE DEFICIENT RATS: AN ELECTRON MICROSCOPIC STUDY. *(E.)* Saadi, A. A. (William Beaumont Hosp., Royal Oak, Mich.). *Proc Am Assoc Cancer Res* 13(March):28, 1972.

1122 SYNTHESIS OF α-FETOPROTEIN BY LIVER, YOLK

SAC AND GASTROINTESTINAL TRACT IN THE HUMAN CONCEPTUS. *(E.)* Gitlin, D. (U. Pittsburgh Sch. Med., Pa.), A. Perricelli and G. M. Gitlin. *Proc Am Assoc Cancer Res* 13(March):12, 1972.

1123 HLA-2 AND HLA-12 PHENOTYPES IN PATIENTS WITH LYMPHATIC MALIGNANCIES. *(E.)* Fortuny, I. E. (U. Minnesota Hospitals, Minneapolis), F. Dick, A. Theologides and E. Yunis. *Proc Am Assoc Cancer Res* 13(March):10, 1972.

1124 POLYADENOSINE (POLY A) CONTENT OF HUMAN LEUKEMIC CELLS. *(E.)* Diez, J. (Harvard Med. Sch., Boston, Mass.), L. S. Desai, T. Liu, G. Yerganian and G. E. Foley. *Proc Am Assoc Cancer Res* 13(March):9, 1972.

1125 TYROSINASE ACTIVITY AND ITS ENDOGENOUS INHIBITION IN HUMAN MELANOMA. *(E.)* Chen, Y. M. (Michigan Cancer Fdn., Detroit). *Proc Am Assoc Cancer Res* 13(March):6, 1972.

1126 A STATISTICAL ANALYSIS OF CHROMOSOME CHANGES IN 73 CASES OF HUMAN CANCER. *(E.)* Miles, C. P. (Dept. Path., U. Utah, Salt Lake City). *Proc Am Assoc Cancer Res* 13(March): 6, 1972.

1127 ADENINE PHOSPHORIBOSYLTRANSFERASE ACTIVITIES OF TUMORIGENIC AND NONTUMORIGENIC MOUSE CELLS. *(E.)* Blair, D. G. R. (Dept. Cancer Res., U. Saskatchewan, Saskatoon, Canada) and J. F. Morgan. *Proc Am Assoc Cancer Res* 13(March):5, 1972.

1128 FIXATION OF PATHOLOGICAL LESIONS IN ADVANCED INBRED CHINESE HAMSTERS. *(E.)* Yerganian, G. (Children's Cancer Res. Fdn., Boston, Mass.), H. J. Gagnon and W. E. O'Connor, Jr. *Proc Am Assoc Cancer Res* 13(March):4, 1972.

1129 EFFECT OF LOW CARBOHYDRATE DIET ON *IN VIVO* GROWTH OF WALKER 256 INTRAMUSCULAR CARCINOMA IN RATS. *(E.)* Gold, J. (Syracuse Cancer Res. Inst., N.Y.). *Proc Am Assoc Cancer Res* 13(March):4, 1972.

1130 PROSPECTIVE STUDY OF THE CANCER FAMILY SYNDROME. *(E.)* Lynch, H. T. (Creighton U. Sch. Med., Omaha, Neb.) and A. J. Krush. *Proc Am Assoc Cancer Res* 13(March):3, 1972.

1131 INCORPORATION OF 5-BROMODEOXYURIDINE (BUDR) INTO HEPATOMA CELLS: INHIBITION OF SYNTHESIS OF TYROSINE AMINOTRANSFERASE (TAT) AND TUMOR FORMATION. *(E.)* Dethlefsen, L. A. (Dept. Biochem., Biophys., U. California, San Francisco) and G. M. Tomkins. *Proc Am Assoc Cancer Res* 13(March):1, 1972.

1132 DEACTIVATION OF TUMOR CELLS BY BLOCKING OF SULFHYDRYLS. *(E.)* Apffel, C. A. (Pondville Hosp., Walpole, Mass.). *Proc Am Assoc Cancer Res* 13(March):1, 1972.

1133 QUANTITATION OF β-GLUCURONIDASE IN GROWING AND REGRESSING MAMMARY CARCINOMAS. *(E.)* Lanzerotti, R. H. (Natl. Inst. Hlth., Bethesda, Md.), Y. S. Cho-Chung and P. M. Gullino. *Proc Am Assoc Cancer Res* 13(March):23, 1972.

1134 METHYLATION OF MOUSE LIVER, SKIN, AND SKIN TUMOR RIBOSOMAL RNA. *(E.)* Colburn, N. H. (Dept. Biol. Sci., U. Delaware, Newark), S. A. Hartman, M. C. Leo and M. A. Della Fera. *Proc Am Assoc Cancer Res* 13(March):61, 1972.

1135 RNA POLYMERASE OF HUMAN TUMOR CELLS. *(E.)* Austin, G. E. (U. Pennsylvania Sch. Med., Philadelphia). *Proc Am Assoc Cancer Res* 13(March):61, 1972.

1136 SPECIFICITY OF ACETYLATION OF HISTONES F3 AND F2a1 FROM NORMAL AND LEUKEMIC LYMPHOCYTES. *(E.)* Miller, D. M. (Dept. Biochem., Duke U., Durham, N.C.), W. F. Marzluff, L. A. Sanders and K. S. McCarty. *Proc Am Assoc Cancer Res* 13(March):64, 1972.

1137 ORNITHINE CARBAMOYLTRANSFERASE (OCT) IN NORMAL, NEOPLASTIC, REGENERATING AND DIFFERENTIATING LIVER. *(E.)* Weber, G. (Indiana U. Sch. Med., Indianapolis), S. F. Queener and H. P. Morris. *Proc Am Assoc Cancer Res* 13(March): 65, 1972.

1138 RIBONUCLEOTIDE REDUCTASE ACTIVITY AND DNA SYNTHESIS IN EHRLICH ASCITES TUMOR CELLS. *(E.)* Cory, J. G. (U. South Florida, Tampa) and T. W. Whitford, Jr. *Proc Am Assoc Cancer Res* 13(March):72, 1972.

1139 REGULATION OF NORMAL AND LEUKEMIC DNA SYNTHESIS BY A SERUM FACTOR. *(E.)* Burke, P. J. (Johns Hopkins U. Sch. Med., Baltimore, Md.), C. H. Diggs and A. H. Owens, Jr. *Proc Am Assoc Cancer Res* 13(March):73, 1972.

1140 ELEVATED SERUM DOPAMINE-β-HYDROXYLASE (DβH) ACTIVITY IN PATIENTS WITH NEUROBLASTOMA TUMORS. *(E.)* Freedman, L. S. (New York U. Med. Ctr., N.Y.), M. Goldstein, C. N. Ores, A. C. Bohuon and F. Guerinot. *Proc Am Assoc Cancer Res* 13(March):75, 1972.

1141 *IN VITRO* AND *IN VIVO* DIFFERENTIATION OF A MURINE NEUROBLASTOMA. *(E.)* Ruffner, B. W., Jr. (U. Virginia Sch. Med., Charlottesville). *Proc Am Assoc Cancer Res* 13(March):77, 1972.

142 TRANSFER RNA PATTERNS IN TUMOR CELLS. *(E.)* Griffin, A. C. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), W. T. riscoe, W. Taylor and D. D. Black. *Proc Am Assoc Cancer Res* 13(March):78, 1972.

143 APPARENT FETAL FORMS OF AMINO ACID NAPTHYLAMIDASE IN HUMAN LIVER METASTASES. *(E.)* Smith, E. E. (Boston U. Sch. Med., Mass.). *Proc Am Assoc Cancer Res* 13(March):79, 1972.

1144 HYPERFIBRINOGENEMIA AND THROMBOSIS IN PATIENTS WITH NEOPLASIA. *(E.)* Brugarolas, A. (Roswell Park Mem. Inst., Buffalo, N.Y.), A. Mittelman and E. E. Sproul. *Proc Am Assoc Cancer Res* 13(March):82, 1972.

1145 MEMBRANE-ASSOCIATED DNA POLYMERASE, THYMIDINE KINASE AND RIBONUCLEOTIDE REDUCTASE ACTIVITIES IN REGENERATING RAT LIVER AND NOVIKOFF TUMOR. *(E.)* Baril, E. (Duke U. Med. Ctr., Durham, N.C.), B. Baril and H. Elford. *Proc Am Assoc Cancer Res* 13(March):84, 1972.

1146 STUDIES OF HUMAN MALIGNANT CELL CULTURES. *(E.)* Fjelde, A. (Roswell Park Mem. Inst., Buffalo, N.Y.) and E. A. Mirand. *Proc Am Assoc Cancer Res* 13(March):86, 1972.

1147 EVIDENCE FOR THE SPECIFIC BINDING OF ^{3}H-TRIAMCINOLONE ACETONIDE (^{3}H-TA) TO MACROMOLECULES IN NORMAL AND NEOPLASTIC HUMAN TISSUES. *(E.)* Gailani, S. (Roswell Park Mem. Inst., Buffalo, N.Y.), F. Rosen, A. Nussbaum, P. Silvernail and K. Shimaoka. *Proc Am Assoc Cancer Res* 13(March):86, 1972.

1148 STIMULATION OF HUMAN NEUROBLASTOMAS *IN VITRO* WITH NERVE GROWTH FACTOR. *(E.)* Goldstein, M. N. (Washington U. Sch. Med., St. Louis, Mo.), V. Land and R. Bradshaw. *Proc Am Assoc Cancer Res* 13(March):89, 1972.

1149 KININ FORMING SYSTEM IN MURINE MASTOCYTOMA. *(E.)* Johnston, M. M. (Coll. Physicians, Surg., Columbia U., New York, N.Y.) and L. M. Greenbaum. *Proc Am Assoc Cancer Res* 13(March):103, 1972.

1150 SURFACE EXONUCLEASE OF NOVIKOFF HEPATOMA ASCITES CELLS (NHAC). *(E.)* Crooke, S. T. (Baylor Coll. Med., Houston, Tex.), S. Okada and H. Busch. *Proc Am Assoc Cancer Res* 13(March):106, 1972.

1151 SEQUENCE ANALYSIS OF HIGH MOLECULAR WEIGHT RNA OF NOVIKOFF HEPATOMA CELLS - THE B_3 FRAGMENT. *(E.)* Kanamaru, R. (Baylor Coll. Med., Houston, Tex.), Y. C. Choi and H. Busch. *Proc Am Assoc Cancer Res* 13(March):106, 1972.

1152 REDUCTION OF LEUKEMIC INCIDENCE FOLLOWING SPLENECTOMY IN THE RAT. *(E.)* Moloney, W. C. (Shields Warren Radiation Lab., Boston, Mass.) and V. King. *Proc Am Assoc Cancer Res* 13(March):107, 1972.

1153 EFFECT OF CARBOXYPEPTIDASE G_1 ON LEVELS OF DIHYDROFOLATE REDUCTASE AND N_{10} FORMYLTETRAHYDROFOLATE SYNTHETASE IN HUMAN 4265 LEUKEMIA CELLS IN TISSUE CULTURE. *(E.)* Chello, P. L. (Yale U. Sch. Med., New Haven, Conn.) and J. R. Bertino. *Proc Am Assoc Cancer Res* 13(March):89, 1972.

1154 AMINO ACID ACTIVATION AND PROTEIN SYNTHESIS IN ISOLATED NOVIKOFF TUMOR NUCLEOLI. *(E.)* Hurlbert, R. B. (U. Texas M.D. Anderson Hosp, Tumor Inst., Houston) and A. F. Lamkin. *Proc Am Assoc Cancer Res* 13(March):89, 1972.

1155 HUMAN LYMPHOID CELL LINES DERIVED FROM A PATIENT WITH ACUTE LYMPHOBLASTIC LEUKEMIA: SOME CHARACTERISTICS OF THYMUS-DERIVED LYMPHOCYTES. *(E.)* Minowada, J. (Roswell Park Mem. Inst., Buffalo, N.Y.), T. Ohnuma, S. Watanabe, J. R. Mitchen and G. E. Moore. *Proc Am Assoc Cancer Res* 13(March):91, 1972.

1156 CHRONIC MYELOCYTIC LEUKEMIA OF CHILDHOOD. *(E.)* Smith, K. L. (St. Jude Children's Res. Hosp., Memphis, Tenn.). *Proc Am Assoc Cancer Res* 13(March):99, 1972.

1157 NUCLEOTIDE PHOSPHODIESTERASE ACTIVITIES IN COMPENSATORY HYPERPLASIA OF RAT LIVER. *(E.)* Tsou, K. C. (Sch. Med., U. Pennsylvania, Philadelphia) and K. W. Lo. *Proc Am Assoc Cancer Res* 13(March):102, 1972.

1158 RNA AND DNA DEPENDENT DNA POLYMERASES FROM "NORMAL" AND MALIGNANT HUMAN CELLS. *(E.)* Srivastava, B. I. S. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Proc Am Assoc Cancer Res* 13(March):102, 1972.

1159 CHARACTERIZATION OF POLYADENYLATE CONTAINING RNA OF POLYSOMES FROM RAT LIVER AND MORRIS HEPATOMA 7800. *(E.)* Tweedle, J. W. (McArdle Lab., U. Wisconsin, Madison) and H. C. Pitot. *Proc Am Assoc Cancer Res* 13(March):102, 1972.

1160 EOSINOPHILIC LEUKEMIA WITH FIBROSING ENDOCARDITIS AND SHORT Y CHROMOSOME. *(E.)* Flannery, E. P. (Walter Reed Gen. Hosp., Washington, D.C.), D. E. Dillon, M. V. R. Freeman, J. D. Levy, U. D'Ambrosio and J. L. Bedynek. *Ann Intern Med* 77(2):223-228, 1972.

1161 MALIGNANT DEGENERATION OF OSTEOBLASTOCLASTOMAS AND BONE CHRONDROMAS. *(Rus.)* Fedotov, P. D. (M. F. Bladimirskiy Clin. Res. Inst. Moscow, USSR) and G. B. Fomin. *Vopr Onkol* 18(11):98-100, 1972.

1162 CARBOHYDRATE METABOLISM DISORDERS OCCURRING IN LEUKEMIA. *(Rus.)* Germanov, A. I. (D. I. Ul'yanov Med. Inst. Kubyshev, USSR), V. I. Sorokina, M. N. Stepanova and N. Z. Tarasova. *Kazan Med Zh* (5):34-36, 1972.

1163 SERUM FREE AMINO ACID CONTENT IN PATIENTS WITH CANCER OF THE RECTUM BEFORE AND AFTER SURGERY. *(Rus.)* Aminev, A. M. (D. I. Ul'yanov Med. Inst., Kubyshev, USSR), M. N. Yeliseyev and O. I. Yeliseyeva. *Kazan Med Zh* (5):30-33, 1972.

1164 BRONCHOSCOPIC CRITERIA OF THE SPREAD PROCESS IN LUNG CANCER. *(Rus.)* Kornishin, G. N. (Kubyshev Med. Inst. USSR) and V. N. Shabayev. *Kazan Med Zh* (5):25-26, 1972.

1165 THE FUNCTIONAL CONDITION OF THE LIVER IN PATIENTS WITH BRAIN TUMORS. *(Rus.)* Lakirovich, G. Ya. (Republ. Hosp. Chuvash ASSR Min. Pub. Hlth., USSR). *Kazan Med Zh* (5):23-24, 1972.

1166 THE EARLY SYMPTOMATOLOGY OF PRIMARY CANCER OF THE LIVER. *(Rus.)* Afanas'yeva, O. N. (USSR Min. Pub. Hlth. Hosp. 4th Main Dept.). *Kazan Med Zh* (5):20-22, 1972.

1167 DIFFERENCES IN THE PRIMARY STRUCTURE OF NUCLEOLAR AND RIBOSOMAL HIGH MOLECULAR WEIGHT RNA OF THE NOVIKOFF HEPATOMA AND RAT LIVER, AND POSSIBLE THERAPEUTIC IMPLICATIONS. *(E.)* Seeber, S. (Cancer Res. Dept. U. Clin., Essen, W. Germany) and H. Busch. *Z Krebsforsch* 78(3):265-281, 1972.

1168 BONE MARROW BIOPSY IN CARCINOLOGY: A STUDY OF 65 CASES. *(Fr.)* Diebold, J. (Hotel Dieu Hosp., Paris, France) and C. Devred. *Z Krebsforsch* 78(3):282-289, 1972.

1169 GROWTH OF LARYNX CANCER METASTASES IN WOMEN. *(Rus.)* Lazo, V. V. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR) and E. A. Tsvetkov. *Zh Ushn Nos Gorl Bolezn* 32(3):14-18, 1972.

1170 CONGENITAL LEUKEMIA. *(Sp.)* Fabel, J. (Felicio Rocho Hosp., Belo Horizonte, Brazil). *Pediatr Prat* 43(4):109-112, 1972.

1171 ON REMOTE METASTATIC SPREADING OF LARYNX CANCER. *(Rus.)* Baradulina, M. G. (P. A. Gertsen Sci. Res. Inst. Oncol., Moscow, USSR) and R. I. Slesareva. *Zh Ushn Nos Gorl Bolezn* 32(3):8-14, 1972.

1172 TOTAL ACTIVITY OF LYSOSOMAL ENZYME IN HEPATIC AND MUSCLE TISSUE IN MALIGNANT NEOPLASMS. *(Pol.)* Belowski, H. (Acad. Med. Krakow, Poland), M. Schmidt and Z. Schmidt. *Pol Tyg Lek* 27(35):1347-1350, 1972.

1173 CARCINOMA IN CHILDHOOD. *(Ger.)* Bradic, I. (Surg. Clin. U. Heidelberg, W. Germany), R. Daum, U. Schütze and M. Pieper. *Bruns Beitr Klin Chir* 219(5):393-407, 1972.

1174 THE EFFECT OF FREELY OR ISOLATED GROWING MICE POPULATION ON THE DEVELOPMENT OF TRANSPLANTABLE TUMORS. *(Rus.)* Dechambre, R.-P. (No affiliation) and C. Gosse. *Vopr Onkol* 18(9):51-55, 1972.

1175 ELECTRON MICROSCOPY OF THE CERVIX UNDER CONDITIONS OF DYSPLASIA, CARCINOMA *IN SITU*, AND INVASIVE CARCINOMA. *(Sp.)* Cardoso, J. M. J. ("20th of November" Hosp. Ctr. Mexico City, Mexico), A. Sangines F., E. G. Leuze and A. Sangines M. *Ginecol Obstet Mex* 32(189):53-58, 1972.

1176 TOXIC FACTORS OF THE BLOOD SERUM IN PATIENTS SUFFERING FROM LEUKEMIA AT VARIOUS STAGES OF THE DISEASE. *(Rus.)* Kakhetelidze, M. G. (Central Inst. Hematol. Blood Transf., USSR Min. Pub. Hlth., Moscow), G. V. Osechenskaya and M. G. Sevastyanova. *Probl Gematol Pereliv Krovi* (6):17-20, 1972.

1177 CHANGES IN TOTAL PROTEOLYTIC ACTIVITY OF THE BLOOD AS A RESULT OF TRANSFUSION OF LEUKEMIC LEUKOCYTE CONCENTRATE. *(Rus.)* Petukhov, V. I. (Vladimir Reg. Hosp., Vladimir, USSR). *Probl Gematol Pereliv Krovi* (6):15-17, 1972.

1178 MICROBIAL FACTOR IN COMPLICATIONS OF ACUTE LEUKEMIA. *(Rus.)* Golosova, T. V. (Central Inst. Hematol. Blood Transf., USSR Min. Pub. Hlth., Moscow), V. A. Martynova, G. V. Osechenskaya, E. M. Abakumov, L. G. Kovaleva. *Probl Gematol Pereliv Krovi* (6):7-11, 1972.

1179 HISTOCHEMICAL STUDIES ON PROTEINS, SH AND NH_2 GROUPS IN INFILTRATING MAMMARY CARCINOMA. *(Pol.)* Nowak, H. F. (Inst. Path. Anat., Acad. Med. Bialystok, Poland), J. Jodczyk and B. Cylwik. *Patol Pol* 23(3):317-325, 1972.

1180 OVARIAN CARCINOMAS *IN VITRO* UNDER ENDOXAN INFLUENCE. A CYTOCHEMICAL

NVESTIGATION. *(Ger.)* Teufel, G. (Women's lin. U. Tübingen, W. Germany) and A. Pfleiderer, r. *Arch Gynaekol* 212(2):151-164, 1972.

181 INTRA-OPERATIVE STUDY OF THE LYMPH OUTFLOW FROM THE CARDIAC END OF THE STOMACH N CANCER. *(Rus.)* Pronin, V. I. (N. I. Pirogov ed. Inst., Moscow, USSR). *Eksp Khir Anesteziol* 7(3):27-30, 1972.

182 THE SPREAD OF METASTASES OF MALIGNANT MELANOMA TO REGIONAL LYMPH NODES. *(Ger.)* eidner, F. (Dermatol. Clin. U. Erlangen, W. ermany) and O. P. Hornstein. *Arch Dermatol orsch* 245(1):50-62, 1972.

183 INTERACTION OF TUMOUR NUCLEIC ACIDS WITH DYES. *(Rus.)* Iogannsen, M. G. N. N. Petrov Res. Inst. Oncology, Ministry ubl. Hlth., USSR, Leningrad) and M. A. Dolgopyatova. *iokhimiia* 37(5):1082-1087, 1972.

184 SOME FEATURES OF THE INHIBITION OF DNA SYNTHESIS BY DEOXYADENOSINE IN TUMOUR CELLS. *Rus.)* Vornovitskaya, G. I. (Inst. Exp. Clin. Oncol., cad. Med. Sci. USSR, Moscow), A. D. Alshanetskaya, M. . Novikova, L. P. Karelina, N. S. Morozova and V. S. hapot. *Biokhimiia* 37(5):940-947, 1972.

185 IRON METABOLISM IN HODGKIN'S DISEASE. *(E.)* Beamish, M. R. (Welsh Natl. Sch. Med., Cardiff), P. A. Jones, D. Trevett, I. H. Evans and A. Jacobs. *Br J Cancer* 26:444-452, 1972.

1186 UPTAKE AND METABOLISM OF HISTAMINE BY NEOPLASTIC MAST CELLS. *(E.)* Moroni, F. (Dept. Pharmacology, U. Florence, Italy), E. Buiatti and P. F. Mannaioni. *Pharmacol Res Commun* 4(1):5-15, 1972.

1187 TRANSPLANTABLE LUTEOMA OF THE MOUSE. AN ULTRASTRUCTURAL AND BIOCHEMICAL STUDY. *(E.)* Roth, L. M. (Tulane U. Sch. Med., New Orleans, La.), W. H. Sternberg, R. A. Huseby, A. A. MacPhee, F. E. Cole and B. F. Rice. *Lab Invest* 27(1):115-122, 1972.

1188 CYTOGENETIC STUDIES IN PATIENTS WITH LEUKEMIA. *(Sp.)* Elejalde, R. (Dept. Path., U. Antioquia, Medellin, Columbia) and A. Restrepo. *Antioquia Med* 22(1):5-22, 1972.

1189 INDUCTION AND TREATMENT OF WILMS TUMOR BY TRANSPLANTATION OF RENAL BLASTOMA IN A NEW EXPERIMENTAL MODEL. *(E.)* Javadpour, N. (Chicago Med. Sch., Ill.) and I. M. Bush. *J Urol* 107:931-937, 1972.

1190 SPINDLE CELL SQUAMOUS CARCINOMA OF THE PENIS. *(E.)* Wood, E. W. (Med. U. South Carolina, Charleston), W. A. Gardner, Jr. and F. M. Brown. *J Urol* 107:990-991, 1972.

1191 PLASMA CELL GRANULOMA OF THE RENAL PELVIS. *(E.)* Davides, K. C. (No affiliation), S. H. Johnson, III, M. Marshall, Jr., S. E. Price, Jr. and A. Stavrides. *J Urol* 107:938-939. 1972

1192 PRIMARY CARCINOMA OF THE LIVER (HEPATOMA) IN RHODESIA. A CLINICAL STUDY. *(E.)* Gelfand, M. (Dept. Path., U. Rhodesia, Salisbury), W. M. Castle and W. M. Buchanan. *S Afr Med J* 46:527-532, 1972.

1193 MULTIPLE GASTRIC NEOPLASMS FOLLOWED BY COLOVESICAL FISTULA. *(E.)* Siddiqui, A. (Beekman-Downtown Hosp., New York, N.Y.), H. Steinberg, A. Vasilas, B. A. Payson, B. Moumgis, H. H. Coppersmith, J. T. Flynn, S. A. Fink, B. M. Wagner and G. Schreiber. *NY State J Med* 72(12):1606-1614, 1972.

1194 CONGENITAL MALIGNANT MELANOMA. *(E.)* Mompoint, O. (Jamaica Hosp., N.Y.) and M. L. Blumberg. *NY State J Med* 72(12):1629-1631, 1972.

1195 INTRACRANIAL LEUKAEMIA AND THE CEREBROSPINAL FLUID. *(E.)* Rawbone, R. G. (Fulham Hosp., London, England), M. T. Shaw and K. D. Bagshawe. *Br Med J* 2:444, 1972

1196 ZINC, COPPER, AND IRON CONTENT OF PLEURAL FLUID IN BENIGN AND NEOPLASTIC DISEASE. *(E.)* Dines, D. E. (Mayo Clin., Mayo Fdn., Rochester, Minn.), L. R. Elveback and J. T. McCall. *Thorax* 27:368-372, 1972.

1197 LACK OF INTRACELLULAR CONVERSION OF DIHYDROHOMOFOLATE TO TETRAHYDROHOMOFOLATE IN LEUKEMIA CELLS WITH HIGH LEVELS OF DIHYDROFOLATE REDUCTASE. *(E.)* Nahas, A. (Tufts U. Sch. Med., Boston, Mass.) and M. Friedkin. *Mol Pharmacol* 8(3):353-361, 1972.

1198 SOME PATHOLOGICAL ASPECTS OF THE SPONTANEOUS LYMPHOID TUMOUR IN *XENOPUS LAEVIS*. *(E.)* Hadji-Azimi, I. (Exp. Zoology Station, U. Geneva, Switzerland) and M. Fischberg. *Pathol Microbiol* 38(2):118-132, 1972.

1199 PATTERNS OF CUTANEOUS METASTASIS. *(E.)* Brownstein, M. H. (Armed Forces Inst. Path., Washington, D. C.) and E. B. Helwig. *Arch Dermatol* 105(6):862-868, 1972.

1200 AGE-ASSOCIATED ANEUPLOIDY: LOSS OF Y CHROMOSOME FROM HUMAN BONE MARROW CELLS WITH AGING. *(E.)* Pierre, R. V. (Mayo Clin. Fdn., Rochester, Minn.) and H. Clark Hoagland. *Cancer* 30(4):889-894, 1972.

AARONSON, S.A.
941
AARSSEN, A.M.
845
ABAKUMOV, E.M.
1178*
ABLASHI, D.V.
788, 889*
ABRAMS, R.
820
ABSE, D.W.
753*
ACHEY, P.M.
779*
ADAM, E.
963
ADAMSON, R.H.
889*
ADELMAN, H.
745*
ADELSTEIN, A.M.
1050
AFANAS'YEVA, O.N.
1166*
AGOSTINI, C.
689
AHMED, N.
1020*
AKIMOVA, R.N.
1038*
ALBERT, S.
1118*
ALBRIGHT, N.L.
729*
ALDRICH, C.D.
903*
ALEKSANDROV, S.V.
781*
ALEKSANDROV, V.A.
732*
ALESSANDRI, G.
647*
ALEXANDER, M.
667
ALEXANDER, P.
948
ALLEN, D.W.
637*
ALLEN, P.W.
649*
ALLFREY, V.G.
715
ALSHANETSKAYA, A.D.
1184*
AMES, B.N.
717
AMINEV, A.M.
1163*
ANDERSEN, E.
908*
ANDERSON, J.P.
869
ANDERSON, K.M.
698, 757*
ANDERSON, W.B.
795
AOKI, K.
704
AOKI, T.
941
APFFEL, C.A.
1132*
ARCOS, J.M.
981
ARMSTRONG, G.R.
788, 889*
ARNOLD, W.J.
916*
ARNSTEIN, P.
829
ASAL, N.R.
1044
ASFAW, T.
904*
ASHMAN, R.B.
905*
AUSTIN, G.E.
1135*
AUTIAN, J.
748*
AXELROD, L.R.
897*
AYANABA, A.
667
AZAR, H.A.
1010*
AZARNIA, R.
1094
BABUSIKOVA, O.
938
BACCICHETTI, F.
980
BAGSHAWE, D.
1195*
BAGSHAWE, K.D.
989*
BAKER, M.A.
918
BAKER, M.S.
1109*
BALDI, I.
993*
BALDWIN, R.W.
933, 951
BALL, J.K.
806
BAMBERGER, E.G.
1095
BANG, F.B.
1117*
BANJO, C.
1014*
BANNIKOV, G.A.
655
BANSAL, H.C.
743*
BANSAL, S.C.
965
BARADULINA, M.G.
1171*
BARDOS, T.J.
852
BARESOVA, M.
887
BARIL, B.
1145*
BARIL, E.
1145*
BARKER, L.F.
988*
BARLOW, J.J.
929
BARNHART, F.E.
1042
BARON, S.
846
BARRY, E.J.
701
BATEMAN, J.L.
634*
BATRA, B.K.
755*, 813
BATSAKIS, J.G.
618*, 648*
BATTEMA, W.L.
1111*
BAUER, H.
801, 834, 935
BAUER, L.
657
BAUER, W.C.
617*
BAXT, W.
825
BEAHRS, O.H.
1041
BEAMISH, M.R.
1185*
BEAUDREAU, G.S.
831
BEDFORD, A.J.
666
BEDYNEK, J.L.
1160*
BEKESI, J.G.
983*
BEKTEMIROV, T.A.
792
BELITSKIY, G.A.
734*
BELL, T.M.
1043
BELMONTE, A.A.
671
BELOWSKI, H.
1172*
BEN-ISHAI, R.
782*
BEN-MENACHEM, Y.
754*
BENACERRAF, B.
947
BENDER, R.I.
615*
BENEDICT, W.F.
724*
BENJAMIN, S.A.
770
BENTVELZEN, P.
845
BENVENISTE, R.
812
BENYESH-MELNICK, M.
838

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

BEREZESKY, I.K.
837
BERGOC, P.M.
972
BERKSON, P.
745*
BERNHARD, W.
809
BERTINO, J.R.
1153*
BESA, E.
730*
BHATTACHARYA, M.
929
BIANCO, C.
952
BIANCOFIORI, C.
762*
BIGGS, P.M.
801
BIGNER, D.D.
860, 896*
BINIAMINOV, M.
1013*
BISHOF, J.M.
821
BISWAL, N.
838
BLACK, D.D.
1142*
BLACK, P.H.
803
BLAIR, D.G.R.
1127*
BLINOVA, G.A.
1031*
BLIZNAKOV, E.G.
953
BLOKZIJL, F.J.
739*
BLOMBERG, F.
928, 946, 954
BLUMBERG, M.L.
1194*
BLUMENFELD, C.C.
1003*
BLUNCK, J.M.
692
BOERZECKA, K.
994*
BODEY, G.P.
780*
BOEKER, B.B.
770
BOGDAL, J.
994*
BOGUSH, T.A.
734*
BOHLIG, H.
650*
BOHUON, A.C.
1140*
BOLIS, G.E.
907*
BOLUND, L.
902*
BOONE, C.W.
925, 971
BORDES, M.
937
BOSMANN, H.B.
842
BOURGEAUX, C.
937
BOYSE, E.A.
999*
BRADIC, I.
1173*
BRADSHAW, R.
1148*
BRADY, R.O.
868
BRAILOVSKY, C.
888, 997*
BRALOW, S.P.
754*
BRANCH, W.R.
927
BRANDCHAFT, P.B.
971
BRANDNER, G.
863, 901*
BRAUNSTEIN, G.D.
1009*
BRENNEMAN, A.R.
1077
BRESNICK, E.
691
BRIDGES, K.R.
996*
BRIDSON, W.E.
1009*
BRIGHTWELL, J.
669
BRINKHOF, J.
845
BRISCOE, W.T.
1142*
BRODSKY, I.
1019*
BROSS, I.D.J.
737*
BROUCEK, J.
867
BROWN, C.E.
768
BROWN, F.M.
1190*
BROWN, R.S.
753*
BROWNSTEIN, M.H.
1199*
BRUGAROLAS, A.
1144*
BUBENIK, J.
887
BUCHANAN, W.M.
1192*
BUCKLER, C.E.
846
BUIATTI, E.
1186*
BUL'BULYAN, M.A.
610*
BURBANK, F.
690
BURDICK, J.T.
936
BURGER, D.R.
969
BURK, R.R.
882
BURKE, P.J.
1139*
BURNETT, J.P.
848
BURNETT, W.S.
713
BURTON, A.F.
723*
BUSCH, H.
1150*, 1151*, 1167*
BUSH, I.M.
1189*
BUTLER, L.K.
848
BUTLER, M.
1069
BUXTON, W.D.
753*
BYKOVSKY, A.F.
792
BYRE, G.E.
895*
BYRNE, M.
924
BYVOET, P.
1098*
CABINESS, J.R.
1020*
CAGNOL, C.
647*
CAIN, J.A.
749*
CALABRESI, P.
968
CALAFAT, J.
828
CALALRESI, P.
924
CALOTHY, G.
855
CALVIN, M.
710
CANAANI, E.
884
CAPPS, M.J.
673
CARDOSO, J.M.J.
1175*
CASEY, A.E.
764*
CASTLE, W.M.
1192*
CASTRO, A.E.
815
CASTRO, J.E.
1012*
CAVALIERI, E.
722*
CAWLEY, J.
899*
CEGLOWSKI, W.S.
931

CERNY, J.
849
CHAKLIN, A.V.
610*
CHANCE, B.
844
CHANDRA, P.
852
CHANG, S.S.
915*
CHARKVIANI, L.I.
1061*
CHASE, G.A.
1048
CHELLO, P.L.
1153*
CHEN, Y.M.
1125*
CHERKASSKIY, L.A.
1036*, 1037*
CHEW, P.C.T.
644*, 645*
CHEW, S.C.
644*
CHIFFELLE, T.L.
770
CHO-CHUNG, Y.S.
1133*
CHOI, Y.C.
1151*
CHOPRA, H.C.
850
CHORY, J.
917*
CHOU, S.T.
744*
CHU, C.
711, 766*
CIESIELSKI-TRESKA, J.
1088
CINTI, D.L.
694
CLARKE, G.D.
805
CLAYSON, D.E.
666
CLEMENT, J.
977
CLEVELAND, P.
815
CLOUSE, J.A.
727*
COBO DE GUTIERREZ, A.
1022
COCHRAN, A.J.
1005*
COFFIN, D.E.
660
COHEN, A.M.
930, 936, 942, 979
COHEN, F.B.
619*
COLBURN, N.H.
1134*
COLBY, C.
911*
COLCHER, D.
843
COLE, F.E.
1187*
COLE, F.
637*
COLIGAN, J.E.
985*
COLLOT, M.
1097
COMI, F.
689
COOPER, E.H.
636*
COPPERSMITH, H.H.
1193*
CORBETT, T.H.
668
CORNELIUS, E.A.
816
CORY, J.G.
1138*
COURTNEY, D.G.
779*
COUTINHO, W.G.
1105*
CRAIG, A.W.
673
CRAVIOTO, H.
697
CREAGAN, E.T.
1068*
CRITTENDEN, L.B.
808
CROCE, C.M.
855
CROCKER, T.T.
678
CROOKE, S.T.
1150*
CSATARY, L.K.
623*
CUBIE, H.A.
959
CULP, L.A.
803
CUMMINGS, B.
913*
CUMMINGS, F.J.
924
CUTRONEO, K.R.
1110*
CYLWIK, B.
1179*
D'AMBROSIO, U.
1160*
D'ATH, E.F.
714
DALLDORF, G.
1042
DANILOV, V.S.
1023
DAS, M.R.
822
DAUM, R.
1173*
DAVID, C.B.
772*
DAVIDES, K.C.
1191*
DAVIDSON, J.G.
1051*
DAVIES, D.J.
663
DAVIES, J.N.P.
1042
DAWSON, P.J.
865
DAY, E.D.
860
DAY, N.
1065*
DE BAULT, L.E.
902*
DE HARVEN, E.
912*
DE KERNION, J.E.
857
DE KONING-PIERENS, W.E.
740*
DE PASQUALE, A.
1083
DE VITA, V.T.
1053*
DECHAMBRE, R.-P.
1174*
DEENEY, A.O'C.
831
DEFENDI, V.
855
DELAGARZA, M.
1073
DELLA FERA, M.A.
1134*
DELPECH, A.
977
DELPECH, B.
977
DEN OTTER, W.
948
DENLINGER, R.H.
675
DESAI, L.S.
1124*
DESCHNER, E.
725*
DETHLEFSEN, L.A.
1055*, 1131*
DEVRED, C.
1168*
DHENNIN, L.
917*
DIAMANDOPOULOS, G.T.
870
DICK, F.
1123*
DIEBOLD, J.
1168*
DIEZ, J.
1124*
DIGGELMANN, H.
940
DIGGS, C.H.
1139*
DILETTUSO, B.
1056*
DILLINGHAM, E.O.
748*

DILLON, D.E. 1160*
DIMITROV, N.V. 1019*
DINES, D.E. 1196*
DIPAOLO, J.A. 720
DIPPLE, A. 661, 670
DIWAN, B.A. 755*
DIXON, J.A. 1054*
DOBRYNIN, V.A. 609*
DOCKERTY, M.E. 1041
DOKU, H.C. 1032*
DOLGOPYATOVA, M.A. 1183*
DOLLER, E. 990*
DONNA, A. 606
DONOVAN, J.W. 1059*
DORLING, P.R. 692
DORZH, S. 1066*
DORZHGOTOV, E. 1066*
DRAYCOTT, C. 892*
DUESBERG, P. 884
DUFF, R. 990*
DUNAWAY, G.A., JR. 1006*
DURSTON, W.E. 717
DUSCIO, D. 876
DYMLING, J.F. 1027
EAGLE, H. 855
EASON, R. 789
EBBESEN, P. 926
ECKEL, J. 693
EDWARDS, G.S. 715
EL RIDI, R. 887
ELAD, D. 782*
ELEJALDE, R. 1188*
ELFORD, H. 1145*
ELHILALI, M.M. 997*
ELLEGARD, J. 1019*
ELLIOTT, A.Y. 815
ELS, H.J. 798
ELVEBACK, L.R. 1041, 1196*
EMILIA, G. 1083
EMMELOT, P. 681
ENG, C.P. 992*
ENGEL, W.K. 860
ERNST, P. 1057*
ESSNER, E.S. 984*
ETINGEN, L.YE. 626*
EULDERINK, F. 775*
EVANS, A.E. 1101*
EVANS, I.H. 1185*
EVANS, R. 948
EVATT, B.L. 1048
EWART, D.F. 736*
FABEL, J. 1170*
FABIANI, A. 686
FARMER, A. 998*
FARRON, F. 1119*
FATHALLA, M.F. 605
FAUST, C.H., JR. 940
FAVRE, R. 763*
FEDOTOV, P.D. 1161*
FELSBURG, P.J. 897*
FEOLA, J. 1018*
FERDINAND, F. 878
FERGUSON, S.W. 1044
FERON, V.J. 681
FERREIRA, E. 1083
FETTERMAN, G.H. 1081
FIBACH, E. 960, 1086
FIELDSTEEL, A.H. 865
FILATOV, F.P. 792
FINE, D.L. 850
FINK, S.A. 1193*
FISCHBERG, M. 1198*
FISH, C.A. 1102*
FISHMAN, P.H. 868
FJELDE, A. 1146*
FLAKS, A. 658
FLAKS, B. 658
FLANNERY, E.P. 1160*
FLECKENSTEIN, B. 858
FLESHER, J.W. 662
FLUKE, D.J. 772*
FLYNN, J.T. 1193*
FOARD, M. 1117*
FOGEL, M. 785
FOLEY, G.E. 902*, 1124*
FOLK, W.R. 807
FOMIN, G.B. 1161*
FORBES, P.D. 654
FORREST, A.P.M. 1092
FORTUNY, I.E. 893*, 1123*
FOWLER, A.K. 872, 874
FOX, J.E. 648*
FRAENKEL-CONRAT, H. 638*
FRALEY, E.E. 815, 857
FRANKEL, J.W. 890*
FRAUMENI, J.F., JR. 1040
FREDRICKSON, T.N. 878
FREEDMAN, L.S. 1140*
FREEDMAN, S.O. 1014*
FREEMAN, M.V.R. 1160*
FREIREICH, E.J. 780*
FRIBERG, S., JR. 923

FRIDMAN, I.A.
1030*
FRIEDKIN, M.
1197*
FRIEDMAN, H.
931
FRIEDMAN, M.A.
679
FRIEND, C.
804, 912*
FRUEHLING, J.
1058*
FUDENBERG, H.H.
962
FUJINAGA, K.
847
FUKUDA, S.
700
GAGNON, H.J.
1128*
GAHRTON, G.
902*
GAILANI, S.
1147*
GALLIMORE, P.H.
982
GALLO, R.C.
826, 898*
GANINA, K.P.
1035*
GANTT, R.
873
GARCIN, M.
647*
GARDNER, D.G.
1111*
GARDNER, F.H.
730*
GARDNER, H.A.
698
GARDNER, S.D.
1021*
GARDNER, W.A., JR.
1190*
GARISCAIN, M.J.
981
GARNER, R.C.
672
GARROD, C.
616*
GAY, S.
986*
GAZDAR, A.F.
846, 974
GEDDES, E.W.
927
GELDERBLOM, H.
935
GELFAND, M.
1060*, 1192*
GERBER, A.
900*, 991*
GERBER, P.
971
GERMANOV, A.I.
1162*
GESERICK, G.
987*
GEY, G.O.
1117*
GIBSON, J.E.
744*
GIELEN, J.E.
724*
GILBERT, P.J.
1111*
GILEAD, Z.
794
GILLESPIE, D.
843, 898*
GILLESPIE, S.
843
GIRARD, M.
853
GIRARDI, S.
765*
GITLIN, D.
1122*
GITLIN, G.M.
1122*
GLASER, R.
894*
GLASS, A.
1009*
GLAZER, R.I.
694
GLEBOVA, M.I.
1062*
GLEICHMANN, E.
601
GLEICHMANN, H.
601
GOEBEL, H.H.
697
GOKDOGAN, G.
738*
GOLD, J.
1129*
GOLD, P.
1014*
GOLDSTEIN, A.L.
1003*
GOLDSTEIN, M.
1140*
GOLDSTEIN, M.N.
1148*
GOLDSTONE, A.H.
998*
GOLOSOVA, T.V.
1178*
GOMARD, E.
976
GOODALL, C.M.
714
GOODIN, F.
925
GORDON, A.S.
878
GOREVAYA, A.N.
1038*
GORISHEK, W.M.
1054*
GORNY, M.K.
1008*
GORSHEIN, D.
730*
GOSDEN, J.
787
GOSSE, C.
1174*
GRAF, T.
801
GRAFF, E.
782*
GRATTAROLA, R.
1026
GRAVES, M.
947
GREEN, M.
782*, 832
GREENBAUM, L.M.
1149*
GREENBLATT, M.
703
GREENWALD, P.
713
GRICE, H.C.
719
GRIFFIN, A.C.
1142*
GRIFFITHS, K.
680
GRIMES, W.J.
802
GRIMLEY, P.M.
837
GRIMM, W.
683
GRISWOLD, D.E.
968
GRISWOLD, D.P.
764*
GROFOVA, M.
877
GROSSI-PAOLETTI, E.
686
GROTH, D.P.
1114*
GROUPE, V.
890*
GRUENEISEN, A.
1002*
GUELSTEIN, V.I.
655
GUERINOT, F.
1140*
GUICHERIT, R.
739*
GULLINO, P.M.
1133*
GUR, R.
1066*
GUREVICH, S.M.
733*
GUTMANN, H.R.
701
HABTE, D.
904*
HADJI-AZIMI, I.
1198*
HAFF, R.F.
783
HAGEMAN, P.C.
828

HAHN, F.F.
770
HAIN, E.
650*
HAIT, W.N.
730*
HAKAMA, M.
1046
HAKOMORI, S.
883
HALASA, J.
849
HANNA, M.G., JR.
871
HANSEN, H.H.
1112*
HARAN-GHERA, N.
836, 975
HARDIN, J.M.
1098*
HARDY, W.D., JR.
878
HAREL, E.
794
HAREWOOD, K.
913*
HARINGTON, J.S.
716
HARNDEN, D.G.
1082
HARRINGTON, J.A.
848
HARRIS, J.R.
933
HARTMAN, S.A.
1134*
HARTMANN, N.R.
1057*
HARVEY, D.
806
HATZFELD, J.
774*
HAYAMI, M.
886
HAYASHI, M.
1086
HEATH, C.W., JR.
640*, 1048
HEBERLING, R.L.
872, 897*
HEHLMANN, R.
825, 854
HEINE, U.
788, 889*
HELLMAN, A.
872, 874
HELLSTROEM, I.
886, 934, 944
HELLSTROEM, K.E.
886, 934, 944
HELM, K.V.D.
884
HELMKE, R.J.
872, 897*
HELWIG, E.B.
1199*
HENDERSON, W.J.
680
HENKART, P.
985*
HENLE, G.
783, 919
HENLE, W.
783
HENRY, J.M.
1071
HENSCHKE, U.K.
1049
HEPPLESTON, A.G.
669
HEPPNER, G.H.
924, 968
HERBERMAN, R.B.
955, 966, 974
HERMAN, B.
602
HESHMAT, M.Y.
1047
HEUFER, M.
901*
HILF, R.
1108*
HILFRICH, J.
693
HINRICHS, D.J.
969
HIRAI, K.
688
HIRANO, A.
856
HIRSHAUT, Y.
984*
HOAGLAND, H.C.
1200*
HOBBS, C.H.
770
HOFFMAN, D.H.
1085
HOLLAND, J.F.
983*
HOLLINSHEAD, A.C.
957
HOLTZMAN, J.L.
699
HOGUE, M.Z.
742*
HORNSTEIN, C.P.
1182*
HORST, J.
638*
HOSODA, S.
608
HSU, H.H.T.
1119*
HUANG, A.T.
772*, 1011*
HUBERMAN, E.
721
HUEBNER, R.J.
829, 922
HULL, E.W.
1009*
HUMMELER, K.
1101*
HUMPHREYS, D.M.
1052*
HURLBERT, R.B.
1154*
HURWITZ, J.
833
HUSEBY, R.A.
1187*
IGLESIAS, R.
765*
IL'ICHEVA, I.A.
735*
IL'YINA, K.P.
1063*
INGENITO, G.
763*
INUI, N.
1029
IOGANNSEN, M.G.
1183*
IRETON, H.J.C.
656, 663
IRISH, L.E.
969
IRVINE, W.J.
998*
IRWIG, L.
716
ISHCHENKO, M.P.
1038*
ISHIMARU, T.
769
ITO, M.
704
ITO, Y.
847
IVERSEN, O.H.
687
IVERSEN, U.
687
JABLON, S.
632*
JACOBS, A.
1185*
JACOBSON, E.L.
1078
JAERPLID, B.
767
JAFFE, N.
1040
JANCINSKI, J.J.
1032*
JARCZEWSKA, K.
1008*
JASTRY, V.
823
JAVADPOUR, N.
1189*
JEFFREY, C.
1060*
JEPSON, J.H.
730*
JODCZYK, J.
1179*
JOHNSON, G.S.
795
JOHNSON, M.D.
710
JOHNSON, S.H., III
1191*

JOHNSTON, M.M.
1149*
JONES, G.H.
995*, 996*
JONES, P.A.
1185*
JONES, R.K.
770
JONGSMA, A.
786
JOSLIN, C.A.F.
680
KAGAN, V.YE.
1023
KAGAWA, R.
741*
KAKHETELIDZE, M.G.
1176*
KALCKAR, H.M.
883
KALTER, S.S.
872, 897*
KAMATA, S.
1025
KANAMARU, R.
1151*
KAPLAN, H.S.
814
KARBY, S.
794
KARELINA, L.F.
1184*
KARLE, H.
1057*
KARNOVSKY, M.J.
932
KARPAS, A.
899*
KARRAN, P.
773*
KARSAI, T.
861
KARTAVOVA, N.S.
1038*
KASNIC, G., JR.
892*
KATCHALSKI, E.
1013*
KATO, K.
844
KATZ, D.H.
947
KAUFMAN, D.G.
1109*
KAUFMAN, R.H.
963
KAWAKAMI, T.G.
925
KEANE, W.M.
1033*
KECLIKOVA, M.
887
KEEFER, L.
714
KEITH, J.
638*
KELLEN, J.A.
698
KENSLER, C.J.
1104*
KERR, L.
998*
KETCHAM, A.S.
930, 942
KEYDAR, J.
794
KHALEELI, M.
1033*
KHAYETS'KYY, I.K.
761*
KHUDOLEY, V.V.
760*
KIJOMOTO, S.
883
KILLANDER, D.
902*
KILLMANN, S.-A.
1057*
KING, R.J.E.
677
KING, V.
1152*
KINGSBURY, E.W.
788, 850
KINZEL, V.
747*
KIRDANI, R.Y.
728*
KIRSCHNER, G.
753*
KIT, S.
790, 866
KIZER, D.E.
727*
KLAGSBRUN, M.
1091
KLEIN, E.
956
KLEISBAUER, J.P.
763*
KLIETMANN, W.
844
KLOSTERKOETTER, W.
650*
KNEALE, G.W.
771*
KNOX, J.M.
776*
KNOX, W.E.
1119*
KOCENT, A.
950
KODAMA, M.
1075
KODAMA, T.
1075
KOEHNLEIN, W.
777*
KOESTNER, A.
675
KOIKE, K.
1078
KOMOLOVA, G.S.
778*
KONIECZNY, L.
994*
KONIKOVA, E.
938
KOPELOVICH, L.
1096
KOPROWSKI, H.
855
KORETSKAYA, L.N.
1038*
KORNISHIN, G.N.
1164*
KOSHY, R.
822
KOURI, R.E.
665
KOVALEVA, L.G.
1178*
KOVI, J.
1047
KOZLOV, YU.F.
1023
KREIBICH, G.
747*
KREIDER, J.W.
1004*
KREMER, W.B.
772*, 1011*
KRIEG, L.
683
KRITSKIY, G.A.
781*
KRUPEY, J.
1014*
KRUSH, A.J.
1130*
KUBICEK, M.T.
850
KUCERA, J.
756*
KUEHN, P.G.
1077
KUFE, D.
835, 854
KURIMURA, T.
856
KURLAND, L.T.
1041
KUTINOVA, L.
867
KVEDAR, J.P.
860
LAKIROVICH, G.YA.
1165*
LALLIER, R.
888
LAMKIN, A.F.
1154*
LAMON, E.W.
956
LANCLOS, K.D.
691
LAND, V.
1148*
LANDAU, T.
960
LANDON, J.C.
850
LANGLANDS, A.O.
1082

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

LANIER, A.P.
1041
LANTOS, P.L.
696
LANZEROTTI, R.H.
1133*
LAROYE, G.J.
627*
LARSSON, A.
1074
LASFARGUES, E.Y.
1105*
LASZLO, J.
1011*
LAUFS, R.
858
LAUMONIER, R.
977
LAVAL, P.
763*
LAW, L.W.
915*, 970
LAWSON, T.A.
674
LAZO, V.V.
1169*
LE MEVEL, B.F.
978
LECATSAS, G.
798
LECLERC, J.C.
976
LECLUSE VAN DER BILT, F.
786
LEDERER, E.
1104*
LEE, F.D.
717
LEE, G.R.
1033*
LEE, T.-C.
1099*
LEESTMA, J.E.
1071
LEFFALL, L.D., JR.
1049
LEIGHTON, J.
1081
LENGLE, N.
1004*
LENNETTE, E.H.
829
LEO, M.C.
1134*
LEPAGE, R.N.
692
LEUCHTENBERGER, C.
659, 1029
LEUCHTENBERGER, R.
659, 1029
LEUZE, E.G.
1175*
LEVIN, J.G.
837
LEVY, A.H.
963
LEVY, J.D.
1160*
LEVY, J.P.
976
LI, F.P.
1040
LIEBELT, A.G.
1110*
LIEBERMAN, M.W.
654
LIJINSKY, W.
714
LILLIEHOOK, E.
923
LILLY, F.
939
LINDENMANN, J.
900*, 991*
LIPKIN, M.
725*
LISKER, R.
1022
LIU, P.I.
769
LIU, T.
1124*
LIZONOVA, A.
877
LJUNGBERG, O.
1027
LLOYD, E.
1100*
LO, K.W.
1157*
LOBUE, J.
878
LOEB, L.A.
1024
LOEB, W.F.
788
LOEWENSTEIN, W.R.
604, 1094
LONG, A.
1118*
LONG, C.
879
LOTLIKAR, P.D.
700
LOZZIO, B.B.
1095
LOZZIO, C.B.
1095
LUKES, R.J.
621*
LYNCH, H.T.
1130*
MACGREGOR, G.A.
630*
MACH, E.
940
MACKENZIE, A.M.R.
1080
MACKEVICIUS, F.
1100*
MACKIE, R.M.
1005*
MACPHEE, A.A.
1187*
MAGNUSSON, G.
820
MAGRATH, J.T.
835
MAJUMDAR, S.K.
751*
MAKARCHUK, N.M.
1038*
MALATHI, V.G.
833
MALIR, A.
950
MANASTER, J.
1058*
MANDEL, P.
1088
MANNAIONI, P.F.
1186*
MANSO, C.
927
MANSUKHANI, S.
1081
MANTEUIL, S.
853
MANTIONE, M.-J.
752*
MARCOVE, R.C.
984*
MARGISON, G.P.
673
MARINO, J.V.
1051*
MARKOE, A.M.
910*
MARKS, F.
683
MAROUDAS, N.G.
707
MARSHALL, M., JR.
1191*
MARSOV, G.N.
652*
MARTIN, F.
937, 943
MARTIN, J.-P.
631*
MARTIN, M.S.
937
MARTONE, W.J.
731*
MARTYNENKO, A.G.
664
MARTYNOVA, V.A.
1178*
MARUYAMA, Y.
1018*
MARX, J.L.
629*
MARZLUFF, W.F.
1136*
MASON, C.H.
1049
MASON, M.M.
695
MASUDA, Y.
741*
MASUKO, K.
704
MATIYENKO, N.A.
1064*

MATOLC, N.N.
1054*
MATSUSHIMA, T.
702
MATSUYAMA, M.
704
MAURI, C.
1083
MAY, J.T.
790, 866
MAYNE, N.
848
MAZUROVSKAYA, O.P.
1036*
MCCALL, J.T.
1196*
MCCARTER, J.A.
806
MCCARTY, K.S.
1136*
MCCLELLAN, R.C.
770
MCCONAHEY, P.J.
874
MCCORMICK, J.
869
MCDONALD, D.
1092
MCDONOUGH, E.
924
MCGAVRAN, M.H.
617*
MCGILL, T.
750*
MCGIVEN, A.R.
656, 663
MCGREGOR, D.H.
769
MCGUIRE, W.L.
1073
MCINTIRE, K.R.
1009*
MCLAREN, J.C.
1078
MCMILLAN, R.
1051*
MCNUTT, N.S.
803
MEDRANO, L.
791
MEDZIHRADSKY, J.
938
MEGO, J.L.
749*
MEHRISHI, J.N.
628*
MEIER, H.
922
MEIJER, P.E.
740*
MELAMED, M.R.
746*
MELNICK, H.
931
MELNICK, J.L.
963
MENEZES, J.
824
MERKLE, K.
1063*
MESTECKY, J.
1001*
METZGAR, R.S.
972
MEYER-ARENDT, J.R.
1052*
MICHEEL, B.
887
MIGLIORATO, D.
1056*
MILAS, L.
958
MILES, C.P.
1126*
MILLER, D.M.
1136*
MILLER, G.
607
MILLER, W.R.
1092
MILLS, L.
931
MILNE, B.S.
801
MINOWADA, J.
1155*
MIRAND, E.A.
1146*
MIRVISH, S.S.
711, 766*
MISHRA, N.K.
881
MITCHEN, J.R.
1155*
MITTAL, S.R.
624*
MITTELMAN, A.
1144*
MITUS, W.J.
1040
MOENKEHAUS, F.
777*
MOLLING, K.
834
MOLONEY, W.C.
902*, 1040, 1152*
MOMPOINT, W.
1194*
MOODIE, C.A.
719
MOONEY, T.F., JR.
708
MOORE, D.H.
1105*
MOORE, E.C.
1120*
MOORE, G.E.
1155*
MOORE, G.W.
1090
MOQUIN, R.E.
641*
MORA, P.T.
868
MORENO, M.A.
972
MORGAN, J.F.
992*, 1127*
MORHENN, V.
799
MORONI, F.
1186*
MOROZOVA, N.S.
1184*
MORRIS, H.P.
1006*, 1097, 1107*, 1137*
MORTON, D.L.
930, 942
MOSLANDER, V.
1054*
MOTEYUNENE, N.P.
759*
MOTOI, M.
921
MOUNGIS, B.
1193*
MUELLER, E.
657
MUELLER, G.-M.
987*
MUELLER, M.
864
MUELLER, N.
863, 901*
MUELLER, W.E.G.
839
MUGGIA, F.M.
1053*, 1112*
MUIR, C.S.
1065*
MUJAGIC, H.
958
MUKAI, F.H.
731*
MUNUBE, G.M.R.
1043
MURAHATA, R.I.
694
MURPHEE, S.
1120*
MURPHY, G.P.
728*
MUSAJO, L.
980
NAGAYO, T.
704
NAHAS, A.
1197*
NARUMI, K.
1016*
NASCA, P.C.
713
NAUDE, W.DU T.
800
NAUJOKS, H.
1090
NAYAK, S.K.
997*
NEAVES, W.B.
1093
NEBERT, D.W.
724*
NEFEDOV, V.P.
609*

NELSON, J.S.R.
1056*
NETTESHEIM, P.
668
NEWELL, G.R.
1045
NEWTON, R.S.
751*
NIGAM, V.N.
888
NOJIMA, T.
827
NOLAND, B.J.
1098*
NONOYAMA, M.
810, 894*
NOON, C.
758*
NOVIKOVA, M.A.
1184*
NOVGORODSKY, A.
1013*
NOWAK, H.F.
1179*
NOWINSKI, R.C.
821
NUNES, L.L.
678
NUNN, J.F.
716
NUSSBAUM, A.
1147*
NUSSENZWEIG, V.
952
O'CONNOR, R.J.
673
O'CONNOR, W.E., JR.
1128*
O'CONNELL, T.V.
678
O'NEILL, C.H.
707
O'RIORDAN, M.L.
1082
O'TOOLE, C.
967
OGAWA, K.
921
OHNUMA, T.
1155*
OIE, H.K.
846
OKADA, H.
769
OKADA, S.
1150*
OKEY, A.E.
684
ORDER, S.E.
947
ORES, C.N.
1140*
ORLANDO, R.A.
1108*
ORMEROD, M.G.
773*
ORTIZ DE LANDAZURI, M.
955
OSECHENSKAYA, G.V.
1176*, 1178*
OSHIRO, Y.
720
OSTER, S.K.
1112*
OSYNS'KYY, S.P.
761*
OWENS, A.H., JR.
1139*
PAGANO, J.S.
810
PAGEAUT, G.
712
PAGES, J.
853
PALAYOOR, S.T.
813
PALESTRO, G.
606
PANIGEL, M.
872, 897*
PANOV, M.A.
734*
PAOLETTI, P.
686
PAOLI, J.
763*
PAPADIMITRIOU, J.M.
905*
PAPE, H.D.
1089
PARK, S.A.
841, 1079
PARKER, J.E.
1000*, 1007*
PARKS, W.P.
812, 829
PASTAN, I.
795
PAUL, D.
909*
PAULSON, D.F.
857
PAUW, W.
797
PAYSON, B.A.
1193*
PEARSON, G.
788
PEARSON, L.D.
903*
PECKHAM, J.C.
764*
PELED, A.
975
PENDOLA, R.G.
823, 880
PERLIN, E.
641*
PERLMANN, P.
967
PERRICELLI, A.
1122*
PERRY, E.
824
PETERSON, E.E.
901*
PETIT, P.
786
PETUKHOV, V.I.
1177*
PEZZOTTA, S.
686
PFITZER, P.
1089
PFLEIDERER, A., JR.
1180*
PHAM, T.C.
1010*
PIANTODOSI, C.
1099*
PICKRELL, J.A.
770
PIEFER, W.
1173*
PIERRE, R.V.
1200*
PIGIET, V.
820
PILON, J.C.
660
PINCUS, S.
952
PINKERTON, H.
895*
PINKUS, H.
646*
PINTO, C.A.
783
PITOT, H.C.
1159*
PLESOVSKIKH, L.D.
1037*
POIRIER, R.
763*
POLAN, A.
713
POLANCO, X.
765*
POLOZOV, R.V.
735*
POLTEV, V.I.
735*
POPOVA, O.B.
1030*
POPOVIC, M.
877
PORTER, M.W.
768
PORTUGAL, M.
927
POUND, A.W.
674
PRASAD, K.N.
1070, 1113*
PREISLER, H.D.
804
PREJEAN, J.D.
764*
PRICE, M.R.
933, 951
PRICE, S.E., JR.
1191*
PRIMACK, A.
988*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

PRONIN, V.I.
1181*
PROZESKY, C.W.
798
PURVES, L.R.
927
QUAGLINO, D.
1083
QUEENER, S.F.
1137*
RAAFF, C.I.
800
RABIN, H.
788
RABINOWITZ, Z.
799
RABSON, A.S.
857, 1076
RADSAK, K.
862
RAFTELL, M.
928, 946, 954
RAICK, A.N.
718
RAJEWSKY, M.F.
622*, 1002*
RAMOT, B.
1013*
RAND, K.H.
879
RANDOLPH, W.
1056*
RANSOHOFF, J.
697
RAPP, F.
642*, 990*
RAPP, G.W.
685
RATNAM, S.S.
644*, 645*
RAWBONE, R.G.
1195*
RAWLS, W.E.
963
RAYMAN, M.P.
661, 670
REBOUD, S.
712
RECTOR, W.D.
1108*
REDMAN, H.C.
770
REEVES, A.L.
708
REEVES, W.J.
1020*
REICHARD, P.
820
REIF, A.E.
1102*
REINHOLD, A.W.
1049
REINISCH, C.L.
875
REIS, H.E.
613*
REITZ, M.S.
826
REJTHAR, A.
1015*
RESTREPO, A.
1188*
REZNIK, G.
693
RIBACCHI, R.
1017*
RICCERI, G.
876
RICE, B.F.
1187*
RIEDLER, G.F.
1028
RIGLER, R.
902*
RILEY, R.M.
1055*
RITTER, U.
1029
ROBBINS, P.W.
1087
ROBERT, M.
898*
ROBERTSON, J.S.
634*
ROBINS, R.A.
951
ROCCHI, G.
859
RODRIGUEZ, V.
780*
ROELLINGHOFF, M.
920
ROGERS, E.
787
ROGERS, G.T.
989*
ROMANENKO, A.M.
664
ROMANO, A.H.
911*
ROSEN, F.
1147*
ROSEN, S.W.
1076
ROSENFELD, C.
1103*
ROSENTHAL, L.J.
817
ROSS, D.D.
1114*
ROTH, L.M.
1187*
ROWLATT, C.
809
ROY-CHOWDHURY, A.K.
708
RUBIN, H.
830
RUBINO, G.F.
606
RUFFNER, B.W., JR.
1141*
RUKOVENA, E.F.
1106*
RUSSELL, E.K.
974
RUSSO, G.
876
RYAN, W.L.
881
RYBARSKA, J.
994*
SAADI, A.A.
1121*
SABESIN, S.
754*
SACHS, L.
721, 851, 960, 1086
SACKLER, J.F.
745*
SADASIVAN, E.
822
SAFFIOTTI, U.
1109*
SAKIYAMA, H.
1087
SAKOUHI, M.
917*
SALERNO, R.A.
665
SALINAS, S.
765*
SALM, R.
1039*
SALOMON, J.
782*
SALZBERG, S.
832
SAMPSON, D.
728*
SAMUNDZHAN, YE.M.
1038*
SANBORN-REDMOND, S.
870
SANDBERG, A.A.
728*
SANDERS, L.A.
1136*
SANDRITTER, W.
1090
SANGINES F., A.
1175*
SANGINES M., A.
1175*
SAPRIN, A.N.
733*
SARIN, P.S.
826
SARKAR, N.H.
821
SARNGADHARAN, M.G.
826
SARTORELLI, A.C.
694
SATO, K.
1107*
SATO, N.
844
SATO, S.
702
SATO, T.
912*
SATOH, T.
914*

SAUTER, C.
900*, 991*
SAXBY, M.
639*
SCANSETTI, G.
606
SCHENKMAN, J.B.
694
SCHER, W.
804
SCHEUFENS, E.E.
1057*
SCHIDLOVSKY, G.
913*
SCHIFFER, D.
686, 1072
SCHIFFER, L.M.
1056*
SCHLESINGER, R.M.
1102*
SCHLOM, J.
843
SCHLUETER, G.
1090
SCHMIDT, M.
1172*
SCHMIDT, Z.
1172*
SCHNEIDER, A.
659
SCHNEIDER, R.L.
1049
SCHREIBER, G.
1193*
SCHROEDER, J.L.
802
SCHROHENLOHER, R.E.
1001*
SCHUELLER, H.
693
SCHUETZ, A.
650*
SCHUETZE, U.
1173*
SCHULMAN, L.H.
833
SCHULTZ, R.M.
784
SCHUMACHER, H.R.
841, 1079
SCOLNICK, E.M.
812
SCOTT, J.L.
1091*
SEARLE, F.
989*
SEEBER, S.
1167*
SEIGLER, H.F.
972
SEKIKAWA, K.
847
SELA, B.-A.
851
SELIGMAN, A.M.
699
SERAFINO, X.
763*
SETLOW, R.B.
1011*
SETTNES, O.P.
964
SEVASTYANOVA, M.G.
1176*
SHABAYEV, V.N.
1164*
SHAFFER, C.
860
SHAMBERGER, R.J.
1106*
SHAPOT, V.S.
1184*
SHAPOVAL, V.V.
1034*
SHAW, M.T.
1195*
SHEPHERD, G.R.
1098*
SHEVCHENKO, V.S.
653*
SHIMADA, K.
847
SHIMAOKA, K.
1147*
SHIVAS, A.A.
1092
SHOFF, W.H.
1004*
SHORE, F.J.
634*
SIDDIQUI, A.
1193*
SILBER, R.
833
SILVERNAIL, F.
1147*
SIMARD, A.
1115*
SINDELAROVA, J.
818
SINGH, D.
743*
SINKOVICS, J.G.
1020*
SIPE, J.C.
896*
SIRSAT, S.M.
822
SITKOVSKIY, M.V.
1023
SIVAK, A.
726*
SIZARET, P.
943
SJOEGREN, H.O.
965
SJOGREN, H.O.
934
SKARIN, A.
902*
SKOOG, K.L.
1074
SKURZAK, H.M.
956
SLESAREVA, R.I.
1171*
SLYNCHAK, S.M.
1034*
SMITH, A.
1050
SMITH, B.
1069
SMITH, C.
805
SMITH, D.C.
719
SMITH, E.E.
1143*
SMITH, E.J.
808
SMITH, G.H.
793
SMITH, H.S.
891*
SMITH, J.C.
776*
SMITH, K.L.
1156*
SMITH, P.G.
1043
SMULEVICH, V.B.
610*
SNODGRASS, M.J.
871
SNYDER, F.
1099*
SNYDER, S.P.
903*
SO, B.T.
706
SOEDERHAELL, S.S.
1074
SOEGTROP, H.H.
839
SOLOVIEV, V.D.
792
SOMERS, K.D.
790, 866
SONIS, S.
1032*
SOROKINA, V.I.
1162*
SOULE, H.
1118*
SOVOVA, V.
887
SPIEGELMAN, S.
825, 835, 843, 854
SPILG, W.G.S.
1005*
SPORN, M.B.
1109*
SPRENGER, E.
1090
SPRINGGATE, C.F.
1024
SPROUL, E.E.
1144*
SQUARTINI, F.
907*
SRIVASTAVA, E.I.S.
1158*
STANCLIFF, E.K.
1045

STANTON, M.F.
707
STANTON, T.H.
1001*
STAVRIDES, A.
1191*
STEER, A.
769
STEGGLES, A.W.
677
STEHELIN, D.
853
STEIN, N.
815
STEINBERG, H.
1193*
STEINMAUS, H.
782*
STELL, F.M.
750*
STENBACK, R.
709
STENBACK, W.A.
869
STEPANOVA, M.N.
1162*
STEPHENSON, J.R.
941
STERNBERG, W.H.
1187*
STEWARD, A.M.
973
STEWART, A.
771*, 1042
STEWART, S.E.
892*
STEWART, W.W.
676
STOLBACH, L.
924
STRAUB, P.W.
1028
STRICKLAND, J.E.
872
STROMBERG, K.
873
STRYCKMANS, P.
1058*
SUDARSANAM, T.
904*
SUESS, R.
747*
SUGIMURA, T.
702
SUKHORUKOY, B.I.
735*
SUN, C.N.
895*
SUZUKI, H.
704
SVOTELIS, M.
1117*
SWARBRICK, J.
671
SWEENEY, M.J.
1085
SWENBERG, J.A.
675
SYDNOR, K.L.
662
SZEKELY, I.E.
841, 1079
TABUCHI, K.
906*
TAKASHIMA, F.
702
TAKAYAMA, S.
1025
TAKEMOTO, K.
898*
TAKEMOTO, K.K.
970
TAN, D.S.K.
919
TAN, P.M.
644*, 645*
TANAKA, N.
702
TANINO, T.
1016*
TARASOVA, N.Z.
1162*
TARRO, G.
945, 957
TASHJIAN, A.H., JR.
1076
TAUB, R.N.
918
TAYLOR, E.A.
922
TAYLOR, W.
1142*
TCHIPYSHEVA, T.A.
655
TEMPLETON, J.W.
969
TENNANT, R.
1077
TERRY, W.D.
985*
TEUFEL, G.
1180*
TEWARI, K.K.
1116*
THEOLOGIDES, A. .
1123*
THOLEY, G.
1088
THOMAS, B.A.
860
THOMAS, C.E.
1005*
THOMAS, D.E.
961
THOMAS, J.W.
1007*
THORNE, H.V.
796, 811
THURNHERR, N.
725*
THURZO, V.
877
TIDINGS, J.
737*
TODARO, G.J.
819, 829
TODARO, M.
876
TODD, C.W.
985*
TOMKINS, G.M.
799, 1131*
TOMPKINS, W.A.F.
784
TONEGAWA, S.
993*
TOTH, B.
682
TOTH, F.D.
861
TRENTIN, J.J.
869
TREVETT, D.
1185*
TRIFONOV, E.N.
778*
TROWBRIDGE, S.T.
838
TRUDEL, M.
888
TSIEN, D.M.
984*
TSOU, K.C.
1157*
TSUMITA, T.
1016*
TSVETKOV, E.A.
1169*
TUKEI, P.M.
1043
TULINIUS, H.
1065*
TULUSAN, A.H.
657
TURKEVICH, N.M.
1038*
TURNBULL, A.C.
680
TURNELL, R.W.
723*
TURNER, R.
666
TWEEDLE, J.W.
1159*
UHR, J.
949
UHR, J.W.
999*
ULLREY, D.
883
UNANUE, E.M.
932
UNSGAARD, B.
967
URASINSKI, I.
994*
URBANIAK, S.J.
998*
VACZI, L.
861
VAIDYA, A.B.
822
VALAZQUEZ-FERRARI, M.
1022

VALERIO, M.C.
788, 850
VAN DER NOORDAA, J.
797
VAN DUUREN, B.L.
726*
VAN RIJSSEL, TH.G.
775*
VAN WYK, J.
798
VARKARAKIS, M.J.
728*
VARMUS, H.E.
821
VASILAS, A.
1193*
VAZQUEZ, J.
1118*
VENUTA, S.
830
VERHEST, A.
786
VERSTRAETE, W.
667
VESELY, P.
818
VICK, N.A.
860, 896*
VIDEBAEK, A.
908*
VIGIER, P.
885
VIKSHRAYTIS, CH.YU.
759*
VITETTA, E.S.
949, 999*
VIZCAINO-GAMEZ, C.
893*
VOGEL, C.L.
988*
VORNOVITSKAYA, G.I.
1184*
VOSKA, V.
867
VOSSENAAR, T.
681
VUKUSIC, P.
765*
WAGNER, E.M.
1193*
WAGNER, G.
620*
WAGNER, H.
920
WAHREN, B.
902*
WAKEFIELD, J.
1050
WALDENSTROEM, J.G.
633*
WALKER, I.G.
736*
WALTER, L.
983*
WARNER, G.A.
934
WARREN, S.
768
WASTIAUX, J.P.
1103*
WATANABE, S.
1155*
WATSON, K.F.
834
WATTIAUX-DE CONINCK, S.
1097
WATTIAUX, R.
1097
WEBER, G.
1006*, 1137*
WEBER, G.H.
831
WECHSLER, W.
675
WEIDNER, F.
1182*
WEINHOLD, D.
986*
WEINHOUSE, S.
1107*
WEINTRAUB, B.D.
1076
WEISBURGER, E.K.
764*
WEISBURGER, J.H.
764*
WEISS, D.W.
836
WEISS, E.DE C.
697
WEISS, J.F.
697
WEISS, L.
603
WEISS, R.A.
818
WEISSMAN, I.L.
814
WEITZ-HAMBURGER, A.
878
WELLS, S.A., JR.
936, 978
WERNER, J.
783
WESTON, D.L.
753*
WHITE, A.
1003*
WHITE, J.E.
1049
WHITE, M.
789
WHITE, M.T.
1116*
WHITFORD, T.W., JR.
1138*
WHITMIRE, C.E.
665
WICKER, R.
809
WILKINS, M.M.
753*
WILLIS, C.E.
1106*
WILSON, H.
989*
WILSON, S.H.
873
WINNACKER, E.L.
820
WITTE, O.N.
814
WITTLIFF, J.L.
1108*
WITZ, G.
726*
WIVEL, N.A.
793
WOITOWITZ, H.-J.
650*
WOLFF-TERROINE, M.
620*
WOLKE, R.E.
823
WOLSTENHOLME, D.R.
1078
WOOD, E.W.
1190*
WOOD, H.B., JR.
764*
WOTKE, J.
1015*
WRIGHT, P.W.
891*
WURTZ, B.
1088
WYBRAN, J.
962
WYCHULIS, A.R.
1041
WYNDER, E.L.
705
YAMAMOTO, N.
700
YAMANGUCHI, K.
886
YAMAZAKI, Z.-I.
839
YANAGIDA, K.
921
YANNOPOULOS, K.
1010*
YASUHIRA, K.
688
YATES, V.J.
823, 880
YEGOROV, I.A.
778*
YELISEYEV, M.N.
1163*
YELISEYEVA, O.I.
1163*
YERGANIAN, G.
1124*, 1128*
YESAIR, D.W.
1104*
YOSHIKAWA-FUKADA, M.
827
YOUNG, D.
787
YUHAS, J.M.
871
YULE, R.
1050

- NTHOMA, LARGE-CELL
  - EPIDERMAL MOSAIC
    - HUMAN, REVIEW, 646*

- CETOXY-2-ACETYLAMINOFLUORENE
  - DNA SYNTHESIS
    - EPIDERMIS, MOUSE, RAT, PIG, 654

- CETOXY-2-FLUORENYLACETAMIDE
  - RIBONUCLEASE ACETYLATION, 701

- CETYLAMINOFLUORENE
  - CELL VIABILITY
    - BACTERIA, 700
  - HEPATOMA
    - FISH, 702
  - PHAGE VIABILITY
    - BACTERIOPHAGE, 700

- NOMYCIN D
  - ALTERATIONS IN DMBA-INDUCED MAMMARY TUMORS
    - RAT, 698
  - HISTONE ACETYLATION
    - LIVER, RAT, 715
  - HISTONE DEACETYLATION
    - LIVER, RAT, 715

- INE PHOSPHORIBOSYLTRANSFERASE
  - TUMORIGENIC AND NONTUMORIGENIC ACTIVITY
    - MOUSE CELLS, 1127*

- OCARCINOMA
  - MAMMARY GLAND
    - CITRATE CONDENSING ENZYME, MOUSE, 1096
    - PYRUVATE DEHYDROGENASE, MOUSE, 1096
  - 20-METHYLCHOLANTHRENE
    - ULTRASTRUCTURE, LUNG, MOUSE, 658

- OMA
  - 20-METHYLCHOLANTHRENE
    - ULTRASTRUCTURE, LUNG, MOUSE, 658

- OSINE NUCLEOTIDE POOL
  - MAMMALIAN CELL CYCLE
    - ENERGY REQUIREMENTS, 1120*

- YLATE CYCLASE
  - ROUS SARCOMA VIRUS-TRANSFORMED FIBROBLASTS
    - CHICK-EMBRYO, 795

- TOXIN
  - PREGNANCY
    - RAT, 719

- TOXIN B1
  - DNA BINDING
    - MICROSOMAL EXTRACT, HAMSTER, 672
  - EPITHELIAL CELLS
    - ULTRASTRUCTURE, MOLLUSK, 657
  - HEPATIC PARENCHYMA
    - ULTRASTRUCTURE, FISH, 657
  - HEPATOMA
    - FISH, 702
  - HISTONE ACETYLATION
    - LIVER, RAT, 715
  - HISTONE DEACETYLATION
    - LIVER, RAT, 715
  - POLYRIBONUCLEOTIDE BINDING
    - MICROSOMAL EXTRACT, HAMSTER, 672
  - PROTEIN BINDING
    - MICROSOMAL EXTRACT, HAMSTER, 672
  - RNA BINDING
    - MICROSOMAL EXTRACT, HAMSTER, 672

- AGGLUTINATION
  - MYELOBLAST
    - NEURAMINIDASE, LEUKEMIA PATIENTS, 991*
  - RNA TUMOR VIRUS-TRANSFORMED CELLS
    - MOUSE, CAT, RAT, 832

- ALBUMIN
  - PROTEIN LEUCINE UPTAKE
    - 4-DIMETHYLAMINOAZOBENZENE, RAT LIVER, 689

- ALKALINE PHOSPHATASE
  - DIMETHYLNITROSAMINE
    - TRANSFORMED CELL, HAMSTER, 851
  - VIRUS, POLYOMA
    - TRANSFORMED CELL, HAMSTER, 851

- ALKYL CARBAMATE
  - DNA
    - INTERACTIONS, MOUSE, 674

- AMINO ACID
  - SERUM FREE CONTENT
    - RECTAL CARCINOMA PATIENTS, 1163*

- AMINO ACID ACTIVATION
  - PROTEIN SYNTHESIS
    - ISOLATED NOVIKOFF TUMOR NUCLEOLI, 1154*

- AMINO ACID NAPHTHYLAMIDASE
  - HUMAN LIVER METASTASES
    - FETAL SERUM, 1143*

- ANDROGEN
  - MAMMARY CARCINOMA
    - HUMAN, 1026
  - METABOLISM
    - HUMAN BREAST TISSUE, 1092

- ANEMIA
  - PRELEUKEMIC SIDEROBLASTIC
    - DIMORPHIC RED CELL, CASE REPORT, 1028

- ANEMIA, SIDEROBLASTIC
  - MULTIPLE MYELOMA
    - CLINICAL STUDY, 1033*

- ANTIBODY
  - ANTINUCLEAR
    - MALIGNANCY, CLINICAL STUDY, 1008*
  - CYTOTOXIC RESPONSE
    - FIBROMA VIRUS-INDUCED TUMORS, RABBIT, 784
  - CYTOTOXIC TUMOR-SPECIFIC
    - 125I-IODODEOXYURIDINE-LABELED SARCOMA CELLS, MOUSE, 978

  - ENZYME INACTIVATION
    - CANCER PATIENTS, REVIEW, 653*
  - HELA CELL
    - TRYPSIN, NORMAL CELL REACTIVITY, HUMAN, 945
  - IGM DEFICIENCY
    - CRYOGLOBULINEMIA, PLASMACYTOMA, CASE REPORT, 986*
  - MELANOMA
    - MONKEY, 972
  - METHYLCHOLANTHRENE-INDUCED SARCOMA
    - GUINEA PIG, 729*
  - VIRUS, EPSTEIN-BARR
    - CHILDREN, WEST MALAYSIAN, 919
  - VIRUS, MAMMARY TUMOR
    - MOUSE, 864
  - VIRUS, PAPILLOMA
    - HUMAN, 959
  - VIRUS, POLYOMA
    - CLINICAL STUDY, ENGLAND, 1021*

- ANTIBODY, NEUTRALIZING
  - VIRUS, EPSTEIN-BARR
    - PATIENTS, 859

- ANTIGEN
  - CARCINO-FETAL
    - DIGESTIVE TRACT TUMORS, HUMAN, 937
  - CARCINOEMBRYONIC
    - ALPHA-FETOGLOBULIN, REVIEW, 619*
    - HETEROSACCHARIDES, HUMAN DIGESTIVE SYSTEM, 1014*
    - LUNG CANCER, HUMAN, 943
    - PRIMARY MEDIASTINAL CHORIOCARCINOMA PATIENT, 989*
  - CELL-SURFACE
    - VIRUS MURINE SARCOMA, 941
  - GROUP-SPECIFIC
    - ASCITIC TUMORS, MOUSE, 1017*
  - H-2
    - IMMUNORESISTANT TUMOR, MOUSE, 923
  - HEPATOMA
    - DIMETHYLNITROSAMINE, RAT, 981
  - LEUKEMIA-ASSOCIATED, HUMAN
    - ANTISERUM PRODUCTION, MOUSE, 918
  - PLASMA MEMBRANE
    - ISOLATION, LYMPHOID CELLS, 971
  - SARCOMA
    - DISTRIBUTION, HUMAN CELLS, 984*
  - SOLUBLE MEMBRANE
    - LIP AND CERVICAL CARCINOMAS, 957
  - TRANSPLANTATION
    - BO-IV CARCINOMA, SYNGENEIC SYSTEM, MOUSE, 1015*
    - SARCOMAS, MOUSE, 970
  - TUMOR-ASSOCIATED
    - CELLULAR IMMUNITY, HUMAN, 966
    - HODGKIN'S DISEASE, HUMAN, 947
  - TUMOR-CELL
    - ISOTOPIC ANTIGLOBULIN ASSAY, 936
  - TUMOR-SPECIFIC
    - CYSTADENOCARCINOMA OF OVARY, HUMAN, 929
    - HEPATOMA, RAT, 933
    - LYMPHOCYTE CYTOTOXICITY, RAT HEPATOMA, 951
    - VIRUS, ROUS SARCOMA, 935
  - VIRAL ENVELOPE
    - MURINE LEUKEMIA VIRUS, 976
    - MURINE SARCOMA VIRUS, 976
  - VIRUS, C-TYPE RNA TUMOR
    - UTERINE TISSUE, MOUSE, 874
  - VIRUS, FELINE LEUKEMIA
    - LEUKEMIC CELLS, CAT, 925
  - VIRUS, HERPES SIMPLEX
    - INFECTION, HAMSTER CELLS, 867
  - VIRUS, MAREK'S DISEASE
    - CELL CULTURE, CHICK, 964
  - VIRUS, MOLONEY LEUKEMIA
    - LYMPHOCYTE CYTOTOXICITY, MOUSE, 956
  - VIRUS, MURINE LEUKEMIA
    - GENETIC CONTROL, MOUSE, 922

- ANTIGEN, THETA
  - IMMUNOGLOBULIN SECRETION
    - THYMUS CELLS, MOUSE, 999*
  - IMMUNOGLOBULIN SYNTHESIS
    - THYMUS CELLS, MOUSE, 999*

- ANTISERUM
  - ANTIGEN, HUMAN LEUKEMIA-ASSOCIATED
    - MOUSE, 918
  - VIRUS, FRIEND LEUKEMIA
    - MOUSE, 912*

- APPENDIX
  - CARCINOID TUMOR
    - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608

- 1-BETA-D-ARABINOFURANOSYLCYTOSINETRIPHOSPHATE
  - DNA POLYMERASE INHIBITION
    - LYMPHOMA CELLS, MOUSE, 839

- ARSENIC
  - BRONCHIAL CARCINOMA
    - CASE REPORT, 763*

- ARYL HYDROCARBON HYDROXYLASE
  - INDUCIBILITY
    - SARCOMA SENSITIVITY, MOUSE, 665
  - SKIN TUMOR
    - MOUSE, 724*

- ASBESTOS
  - CARCINOGENESIS
    - TRACE METALS, 708
  - CARCINOMA OF HEAD AND NECK
    - HUMAN, 750*
  - CHRONIC EXPOSURE
    - PERITONEAL MESOTHELIOMA, CASE REPORT, 745*
  - MESOTHELIOMA
    - RAT, 707
  - OCCUPATIONAL HAZARD
    - REVIEW, 650*
  - POLLUTION, AIR
    - DETECTION AND MEASUREMENT, 740*

- ASCITES TUMOR
  - SEE ALSO EHRLICH
  - ANTIGENS, GROUP-SPECIFIC
    - C-TYPE PARTICLES, MOUSE, 1017*
  - CYTOKINETICS
    - MOUSE, 1056*
  - DEACTIVATION

    SULFHYDRYL BLOCKING, 1132*
  DEVELOPMENT
    ISOLATED MOUSE POPULATION, 1174*
  IMMUNOPROTECTION INDUCTION
    TISSUE CULTURE CELLS, MOUSE, 992*

CORBIC ACID
  AMINOPYRINE NITROSATION BLOCKING
    MOUSE, 703

TROCYTOMA
  FIBROSARCOMA, MENINGEAL
    CASE REPORT, 1071
  IMMUNOLOGY
    BRAIN, HUMAN, 977
  MUCOPOLYSACCHARIDE
    HUMAN, 1069

PASE
  ACTIVITY
    LYMPHOCYTES, SOLID MALIGNANT TUMOR PATIENTS, 1019*

ATHIOPRINE
  CARCINOGENICITY
    MOUSE, RAT, 764*

CILLUS CALMETTE-GUERIN
  VIRUS, RADIATION LEUKEMIA
    LEUKEMOGENESIS, MOUSE, 836

CTERIA
  VIABILITY
    2-ACETYLAMINOFLUORENE, 700

CTERIOPHAGE
  VIABILITY
    2-ACETYLAMINOFLUORENE, 700

CTERIOPHAGE T4
  INDICATOR ORGANISM
    HOST-MEDIATED ASSAY SYSTEM, 731*

NZO(A)PYRENE
  BRONCHIAL MUCOSA
    TOXICITY, HUMAN CULTURE, 678
  INDUCED NUCLEOPHILIC SUBSTITUTION, 710
  METABOLISM
    EPITHELIAL CELLS, HUMAN, 721
    FIBROBLASTS, HUMAN, 721
    LIVER, RAT, 662

NZO(A)PYRENE HYDROXYLASE
  INFLUENZA VIRUS INFECTION
    LUNG TISSUE, MOUSE, 668

NZOYLHYDRAZINE
  LUNG TUMOR
    MOUSE, 682
  LYMPHOMA, MALIGNANT
    MOUSE, 682

BENZYLTHIO CYCLIC AMP
  NEUROBLASTOMA
    TUMORIGENICITY, MOUSE, 1070

N'-BIS(4-AMINOPHENYL)-N,N'-
DIMETHYLENEDIAMINE
  OXIDATION
    RAT LIVER, 699

1,3-BIS(2-CHLOROETHYL)-1-NITROSOUREA
  CARCINOGENICITY
    MOUSE, RAT, 764*

BLOOD
  DIMORPHIC RED CELL
    PRELEUKEMIC SIDEROBLASTIC ANEMIA, CASE REPORT, 1028
  LYMPHOCYTE, COMPLEMENT-RECEPTOR
    LEUKEMIA PATIENTS, 952
  NUCLEIC ACIDS
    RADIATION-INDUCED DAMAGE, RAT, 781*
  PROTEOLYTIC ACTIVITY
    LEUKEMIA LEUKOCYTE TRANSFUSION, HUMAN, 1177*

BLOOD SERUM
  TOXICITY
    LEUKEMIA PATIENTS, 1176*

BONE
  CHONDROMA
    MALIGNANT DEGENERATION, HUMAN, 1161*
  OSTEOBLASTOCLASTOMA
    MALIGNANT DEGENERATION, HUMAN, 1161*
  OSTEOSARCOMA
    PARTICLES, HUMAN, 1100*

BONE MARROW
  CARCINOGENESIS
    BIOPSY STUDY, 1168*
  CHROMOSOME
    LEUKEMIA, HUMAN, 1022
  CHROMOSOME, Y
    AGE-ASSOCIATED ANEUPLOIDY, 1200*
  LEUKEMIA, LYMPHATIC
    MOUSE, 975

BRAIN
  ASTROCYTOMA
    IMMUNOLOGY, HUMAN, 977
  GREY MATTER EDEMA
    VIRALLY-INDUCED GLIOMA, DOG, 896*

BRAIN TUMOR
  N-ETHYL-N-NITROSOUREA
    ULTRASTRUCTURE, RAT, 696
  ETHYLNITROSOUREA
    RAT, 686
  LIVER FUNCTION
    HUMAN, 1165*
  NUCLEOSIDE DIPHOSPHATASE
    HUMAN, 1072
  THIAMINE PYROPHOSPHATASE
    HUMAN, 1072
  VIRUS, ADENO 12
    INTRACRANIAL TRANSPLANTATION, HAMSTER, 906*
  VIRUS, ROUS SARCOMA
    DOG, 860

BREAST
  SEE MAMMARY

- 5-BROMO-2-DEOXYURIDINE
  - HEMOGLOBIN SYNTHESIS
    - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 804

- 5-BROMODEOXYURIDINE
  - INCORPORATION INTO HEPATOMA CELLS
    - INHIBITION OF TUMOR FORMATION, 1131*
  - POLYOMA VIRUS INDUCTION
    - FLUORESCENT LIGHT, RAT, 785

- 7-BROMOETHYLBENZ(A)ANTHRACENE
  - DNA SYNTHESIS
    - EPIDERMIS, MOUSE, RAT, PIG, 654

- 7-BROMOMETHYL-12-METHYLBENZ(A)ANTHRACENE
  - DNA
    - IN VITRO REACTIONS, 670
    - SKIN, MOUSE, 661

- 7-BROMOMETHYLBENZ(A)ANTHRACENE
  - DNA
    - IN VITRO REACTIONS, 670
    - SKIN, MOUSE, 661

- BURKITT'S LYMPHOMA
  - CHROMOSOME ABERRATIONS
    - QUINACRINE FLUORESCENCE, 786
  - ETIOLOGY
    - REVIEW, 630*
  - INCIDENCE
    - AFRICAN CHILDREN, 1042
  - NASOPHARYNGEAL CARCINOMA
    - HOMOLOGOUS RNA, HUMAN, 854
  - REOVIRUS 3 INFECTION
    - INCIDENCE, CORRELATION, UGANDA, 1043
  - RNA-INSTRUCTED DNA POLYMERASE ACTIVITY
    - TUMOR CELLS, CLINICAL STUDY, 835
  - VIRUS, EPSTEIN-BARR
    - HOMOLOGOUS DNA, HUMAN, 810
    - INCIDENCE, ETHIOPIA, 904*

- CANCER
  - LITERATURE INFORMATION RETRIEVAL SYSTEM
    - REVIEW, 620*
  - RESEARCH PROGRAMS
    - REVIEW, 625*

- CARBARYL
  - MUTAGEN, CHEMICAL
    - DROSOPHILA MELANOGASTER, 742*

- CARBOHYDRATE
  - CATABOLISM
    - POLYOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 883
  - DIETARY EFFECT
    - TUMOR GROWTH RATE, RATS, 1129*
  - METABOLISM
    - LEUKEMIA, HUMAN, 1162*

- CARBON TETRACHLORIDE
  - LYSOSOME FUNCTION
    - KIDNEY, MOUSE, 749*
    - LIVER, MOUSE, 749*

- CARBOXYPEPTIDASE G1
  - EFFECT ON FOLATE COENZYMES
    - HUMAN LEUKEMIA CELLS IN TISSUE CULTURE, 1153*

- CARCINOGEN
  - CLASSIFICATION
    - BACTERIAL TEST SYSTEM, 717

- CARCINOID TUMOR
  - APPENDIX
    - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608
  - LUNG
    - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608
  - STOMACH
    - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608

- CELL
  - CHICK EMBRYO, 1117*
  - DEATH
    - TUMORS, REVIEW, 636*
  - GENERATION RATE
    - EHRLICH ASCITES TUMOR, MATHEMATICAL MODEL, 1063*
  - ROSETTE-FORMING
    - THYMUS-DERIVED LYMPHOCYTES, HUMAN, 962
  - SOMATIC HYBRIDS
    - MAMMALIAN GENETICS, REVIEW, 629*
  - SV40-TRANSFORMED KIDNEY CELLS
    - SURFACE CHARACTERISTICS, MOUSE, 856
  - TUMOR CELL POPULATION CHARACTERIZATION
    - DNA MEASUREMENT, HUMAN, 1089

- CELL CYCLE
  - MACROPHAGE MIGRATION INHIBITION
    - LEUKEMIC CELL, MOUSE, 961
  - POLYOMA VIRUS INFECTION SUSCEPTIBILITY
    - MOUSE, 796
  - RNA METABOLISM
    - ACUTE LEUKEMIA, HUMAN, 1083

- CELL DIFFERENTIATION
  - LEUKEMIC CELL
    - PROTEIN MGI, MOUSE, 960
  - MYELOID LEUKEMIC CELLS
    - MOUSE, 1086

- CELL, MAST
  - HISTAMINE METABOLISM
    - MOUSE, 1186*
  - INCIDENCE
    - ORAL LESIONS, HUMAN, 1032*

- CELL MEMBRANE
  - PERMEABILITY CHANGES
    - LEUKEMIC AND NON-LEUKEMIC HUMAN BLOOD CELL LINES, 1103*

- CELL MIGRATION
  - ADHESIVENESS
    - REVIEW, 616*
  - SV40-TRANSFORMED CELL
    - MOUSE, HAMSTER, 882

- L PROLIFERATION
  - DENSITY-DEPENDENT REGULATION
    - TRANSFORMED CELLS, MOUSE, 720
  - DNA REPAIR SYNTHESIS
    - X-RAY-PRODUCED STRAND BREAKS, RAT, 773*
  - N,N'-2,7-FLUORENYLENEBISACETAMIDE
    - BILE DUCTULAR CELLS, RAT, 704
  - GROWTH, TUMOR
    - REVIEW, 622*
  - TRANSPLANTED MAMMARY TUMOR
    - IMMUNE RESPONSE, RAT, 1002*

- L, TUMOR
  - KINETIC STUDIES
    - THYMIDINE INJECTION FOLLOWED BY FINE-NEEDLE ASPIRATION, 1053*
  - QUANTITATIVE MORPHOLOGY, 1052*

- EBROSPINAL FLUID
  - LEUKEMIA, INTRACRANIAL
    - CASE REPORTS, 1195*

- IUM 144
  - INHALATION CARCINOGENESIS
    - DOG, 770

- VICAL CARCINOMA
  - DNA
    - CHROMATIN PATTERN ANALYSIS, 1090
  - EPIDEMIOLOGY
    - U.S.S.R., 1061*, 1062*
  - ETIOLOGY
    - REVIEW, 624*
  - 20-METHYLCHOLANTHRENE
    - PROGESTERONE, MOUSE, 712
  - MORTALITY
    - LOUISIANA, 1045
    - RELATION TO HUSBAND'S OCCUPATION, 1050
    - U.S.S.R., 1061*, 1062*
  - SOLUBLE MEMBRANE ANTIGENS
    - HUMAN, 957
  - TALC
    - HUMAN, 680
  - ULTRASTRUCTURE
    - CLINICAL STUDY, 1175*
  - VIRUS, HERPES
    - REVIEW, 614*
  - VIRUS, HERPES 2
    - SEROEPIDEMIOLOGIC STUDIES, 963

- IX
  - DIMETHYLNITROSAMINE
    - HUMAN, 716

- LDREN
  - CANCER
    - OBSTETRIC RADIOGRAPHY, 771*
    - OBSTETRIC RADIOGRAPHY, REVIEW, 634*
  - CARCINOMA
    - CLINICAL STUDY, 1173*
  - DIGITAL FIBROUS TUMORS
    - REVIEW, 649*

- RAMBUCIL
  - CARCINOGENICITY
    - MOUSE, RAT, 764*

- CHLORAMPHENICOL
  - 3H-DIMETHYLBENZ(A)ANTHRACENE
    - EMBRYO CELLS, MOUSE, 734*
  - HEPATOMA INHIBITION
    - ENZYME ACTIVITY, RAT, 692

- 1-(2-CHLOROETHYL)-3-CYCLOHEXYL-1-NITROSOUREA
  - CARCINOGENICITY
    - MOUSE, RAT, 764*

- CHLOROMA
  - LEUKEMIA, MYELOGENOUS
    - AUTOPSY STUDY, HIROSHIMA-NAGASAKI, 769

- CHLORPROMAZINE
  - CANCER
    - REVIEW, 623*

- CHOLESTEROL
  - DIBENZANTHRACENE
    - AIR-WATER INTERFACE INTERACTION, 671

- CHONDROMA
  - MALIGNANT DEGENERATION
    - HUMAN, 1161*

- CHORIOALLANTOIC MEMBRANE
  - POLYOMA VIRUS-TRANSFORMED BHK CELLS
    - INVASIVENESS, CHICK, 813

- CHORIOCARCINOMA
  - PRIMARY MEDIASTINAL
    - CARCINOEMBRYONIC ANTIGEN, 989*
    - HUMAN CHORIONIC GONADOTROPHIN, 989*
    - REGAN ISOENZYME, 989*

- CHROMATIN
  - PATTERN ANALYSIS
    - CERVICAL CARCINOMA, 1090

- CHROMATIN, SEX
  - HORMONE-DEPENDENT NEOPLASTIC CONDITIONS
    - HUMAN, 1035*
  - HORMONE-DEPENDENT PRENEOPLASTIC CONDITIONS
    - HUMAN, 1035*

- CHROMOSOME
  - BONE MARROW
    - LEUKEMIA, HUMAN, 1022
  - CHROMATID LESIONS
    - N-NITROSO-N-ETHYLUREA, VICIA FABA, 756*
    - N-NITROSO-N-METHYLUREA, VICIA FABA, 756*
  - TRANSLOCATION HETEROZYGOTE
    - CAPSICUM ANNUUM L., 743*

- CHROMOSOME ABERRATIONS
  - BURKITT'S LYMPHOMA CELL LINE
    - QUINACRINE FLUORESCENCE, 786
  - LYMPHOCYTE
    - CANCER PATIENTS, 1082
  - STATISTICAL ANALYSIS

    HUMAN CANCER CELLS, 1126*

CHROMOSOME, Y
  BONE MARROW
    AGE-ASSOCIATED ANEUPLOIDY, 1200*

COAL TAR
  CARCINOGENICITY
    SKIN, MOUSE, 687

COLON CARCINOMA
  CARCINO-FETAL ANTIGEN
    HUMAN, 937
  1,2-DIMETHYLHYDRAZINE
    MOUSE, 725*
  INCIDENCE
    MINNESOTA, 1041
  N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
    RAT, 706

COLONY STIMULATING FACTOR
  EFFECTS UPON MACROMOLECULAR SYNTHESIS
    MOUSE BONE MARROW CULTURES, 1114*

CONCANAVALIN A
  LYMPHOCYTE BINDING
    CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1013*
    RAT, HUMAN, 1013*
  RECEPTORS
    ULTRASTRUCTURE, HAMSTER CELL LINES, 809

COPPER
  PLEURAL FLUID
    NEOPLASTIC DISEASE PATIENTS, 1196*

CORPUS UTERI
  CANCER
    MORPHOGENESIS, HUMAN, 1037*

CORTICOSTEROID
  RESISTANCE
    MOUSE, 723*

CORTISOL
  POLYOMA VIRUS REPLICATION
    MOUSE FIBROBLASTS, 799

CRYOGLOBULINEMIA
  PLASMACYTOMA
    IGM-ANTIBODY DEFICIENCY, CASE REPORT, 986*

CYCLIC AMP
  GROWTH REGULATION
    MOUSE CELLS, 909*
  NEUROBLASTOMA
    TUMORIGENICITY, MOUSE, 1070

CYCLOHEXIMIDE
  POLYRIBOSOME DISAGGREGATION
    LIVER, RAT, 676

CYSTADENOCARCINOMA
  OVARY
    TUMOR-SPECIFIC ANTIGENS, HUMAN, 929

CYTOCHROME C
  LIVER MITOCHONDRIAL OXIDATION
    3,3'-DIAMINOBENZIDINE, RAT, 688

CYTOCHROME OXIDASE
  HEPATOMA
    MITOCHONDRIA, DENSITY-GRADIENT CENTRIFUGATION, 1097

CYTOCHROME SYNTHESIS
  ETHIDIUM BROMIDE
    SV40 TRANSFORMED CELLS, HAMSTER, 844

CYTOTOXICITY
  LYMPHOCYTE
    IMMUNE RESPONSE, URINARY BLADDER TUMOR, HUMAN, 967
    MOLONEY LEUKEMIA VIRUS ANTIGEN, MOUSE, 956
    ROUS SARCOMA CELLS, QUAIL, 886
    SERUM FACTORS, NEOPLASTIC DISEASE PATIENTS, 1020*
    TUMOR ANTIGEN, RAT HEPATOMA, 951
  LYMPHOCYTE-MEDIATED
    MELANOMA CELLS, DIFFUSION CHAMBER DETECTION METHOD, MOUSE, 1004*
  MACROPHAGE
    TUMOR ALLOGRAFT IMMUNITY, MOUSE, 948
  TUMOR-SPECIFIC CELLULAR
    SARCOMAS, HUMAN, 942

CYTOXAN
  CARCINOGENICITY
    MOUSE, RAT, 764*

DDT
  7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMOR
    INHIBITION, RAT, 684

DEOXYADENOSINE
  DNA SYNTHESIS INHIBITION
    HEPATOMA CELLS, RAT, 1184*

2-DEOXYGLUCOSE
  TEMPERATURE DEPENDENT UPTAKE ALTERATIONS
    MOLONEY SARCOMA VIRUS-TRANSFORMED RAT CELLS, 866

DEOXYRIBONUCLEOTIDES
  LIVER REGENERATION
    RAT, 1074

DEXAMETHASONE
  POLYOMA VIRUS REPLICATION
    MOUSE FIBROBLASTS, 799

2,7-DIACETYLAMINOFLUORENE
  LEUKEMIA
    RAT, 1025

3,3'-DIAMINOBENZIDINE
  MITOCHONDRIAL OXIDATION
    LIVER, RAT, 688

DIBENZANTHRACENE

- CHOLESTEROL
  - AIR-WATER INTERFACE INTERACTION, 671
- ENZO(A,L)PYRENE
  - SYNTHESIS
    - CARCINOGENECITY, MOUSE, 741*
- ENZPYRENE
  - VIRUS, FRIEND LEUKEMIA
    - COENZYME Q10, IMMUNITY, MOUSE, 953
- UTYRL CYCLIC AMP
  - 2-DEOXYGLUCOSE TRANSPORT
    - POLYOMA VIRUS-TRANSFORMED CELLS, MOUSE, 802
- UTYRYL CYCLIC AMP
  - NEUROBLASTOMA
    - TUMORIGENICITY, MOUSE, 1070
- T
  - LOW CARBOHYDRATE
    - TUMOR GROWTH RATE, RATS, 1129*
- THYLNITROSAMINE
  - HEPATOCARCINOGENESIS
    - MICROSOMAL DETOXIFICATION SYSTEM, RAT, 733*
  - HEPATOMA
    - ANTIGENICITY, RAT, 981
  - MORPHOLOGICAL ALTERATIONS
    - LIVER, FISH, 760*
  - NASAL CAVITY
    - GLANDULAR TUMORS, HAMSTER, 709
  - RESPIRATORY TRACT TUMORS
    - FERRIC OXIDE, HAMSTER, 681
- THYLSTILBESTROL
  - GAS CHROMATOGRAPHIC RESIDUE DETERMINATION
    - ANIMAL TISSUES, 660
  - KIDNEY TUMOR
    - HAMSTER, 857
  - PREGNANCY
    - CLINICAL STUDY, 705
  - PROSTATE
    - HAMSTER, 857
- ESTIVE SYSTEM
  - CARCINOEMBRYONIC ANTIGEN
    - IMMUNOLOGICALLY ACTIVE HETEROSACCHARIDES, HUMAN, 1014*
- ESTIVE TRACT TUMORS
  - CARCINO-FETAL ANTIGEN
    - HUMAN, 937
- YDROFOLATE REDUCTASE
  - LEUKEMIA
    - CELLS, MOUSE, 1197*
- YDROHOMOFOLATE
  - INTRACELLULAR CONVERSION
    - LEUKEMIA CELLS, MOUSE, 1197*
- YDROOROTATE DEHYDROGENASE
  - HEPATOMA, MORRIS
    - LIVER, RAT, 1085
- 4-DIMETHYLAMINOANTIPYRINE
  - NITROSATION BLOCKING
    - ASCORBIC ACID, MOUSE, 703
- 4-DIMETHYLAMINOAZOBENZENE
  - HEPATOMA
    - TUMOR-SPECIFIC ANTIGEN, RAT, 933
  - PROTEIN LEUCINE UPTAKE
    - ALBUMIN, RAT LIVER, 689
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - CYTOGENETIC EFFECTS
    - HAWORTHIA CALLUS, 751*
  - LEUKEMIA, LYMPHATIC
    - BONE MARROW, MOUSE, 975
    - THYMUS, MOUSE, 975
  - MAMMARY TUMOR
    - ACTINOMYCIN D PROTECTION, RAT, 698
    - DDT, RAT, 684
    - METABOLISM, RAT, 761*
    - POLY I:C, RAT, 757*
  - MORPHOGENETIC EFFECTS
    - HAWORTHIA CALLUS, 751*
  - PAPILLOMA
    - SKIN GRAFTS, MOUSE, 926
  - SKIN TUMOR
    - MOUSE, 724*
  - TRANSFORMATION
    - CELL PROLIFERATION, MOUSE, 720
- 3H-DIMETHYLBENZ(A)ANTHRACENE
  - CHLORAMPHENICOL
    - EMBRYO CELLS, MOUSE, 734*
- DIMETHYLFORMAMIDE
  - HEMOGLOBIN SYNTHESIS
    - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 804
- 1,2-DIMETHYLHYDRAZINE
  - COLON CARCINOMA
    - MOUSE, 725*
- DIMETHYLNITROSAMINE
  - ALKALINE PHOSPHATASE
    - TRANSFORMED CELL, HAMSTER, 851
  - ANCHORAGE DEPENDENCE
    - FIBROBLASTS, RAT EMBRYO, 881
  - CERVIX
    - HUMAN, 716
  - DNA METHYLATION
    - 7-METHYLGUANINE LOSS, RAT LIVER, 673
  - FORMATION
    - MICROBIAL METABOLISM, SEWAGE, SOIL, 667
  - HEPATOMA
    - FISH, 702
  - KIDNEY TUMOR
    - ARTERIOGRAPHY, RAT, 693
    - PYELOGRAPHY, RAT, 693
    - RAT, 656
  - MORPHOLOGICAL ALTERATIONS
    - LIVER, FISH, 760*
  - NUCLEOTIDE PHOSPHODIESTERASE
    - TRANSFORMED CELL, HAMSTER, 851
  - RENAL MESENCHYMAL TUMOR
    - ULTRASTRUCTURE, RAT, 663
  - VAGINA

- HUMAN, 716

- N,N'-DIMETHYLNITROSOUREA
  - THYMIC LYMPHOMA DEVELOPMENT
    - IMMUNE RESPONSE, MOUSE, 921

- DIMETHYLSULFOXIDE
  - HEMOGLOBIN SYNTHESIS
    - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 804

- DNA
  - ALKYL CARBAMATE
    - INTERACTIONS, MOUSE, 674
  - 7-BROMOMETHYL-12-METHYLBENZ(A) ANTHRACENE
    - IN VITRO REACTIONS, 670
    - SKIN, MOUSE, 661
  - 7-BROMOMETHYLBENZ(A)ANTHRACENE
    - IN VITRO REACTIONS, 670
    - SKIN, MOUSE, 661
  - CERVICAL CARCINOMA
    - CHROMATIN PATTERN ANALYSIS, 1090
  - CIGARETTE SMOKE
    - HUMAN LUNG EXPLANTS, 1029
  - CIRCULAR MOLECULES
    - MITOCHONDRIA, RAT, MOUSE, 1078
  - DEGRADATION
    - UV RADIATION, YEAST, 774*
  - INTRAMOLECULAR INTERACTION ENERGY
    - POTENTIAL MUTAGEN DETECTION, 735*
  - ISOLATION
    - AVIAN MYELOBLASTOSIS VIRUS, 831
    - MC29 TUMOR VIRUS, 831
  - MARIJUANA SMOKE
    - HUMAN LUNG EXPLANTS, 1029
  - METHYLATION
    - 7-METHYLGUANINE LOSS, RAT LIVER, 673
  - MILK PARTICLE
    - MAMMARY TUMOR RNA HOMOLOGY, HUMAN, 822
  - PHOTOALKYLATION OF PURINES
    - RADIATION, UV, 782*
  - RADIATION, GAMMA
    - RAT, 778*
  - RADIATION, IONIZING
    - SENSITIVITY TO BREAKAGE, 779*
  - RADIATION, UV
    - B. SUBTILIS, 777*
  - RNA LINKAGE
    - POLYOMA VIRUS, MOUSE CELL, 820
  - SERUM LEVELS
    - MALIGNANT DISEASE PATIENTS, REVIEW, 641*
  - TUMOR CELL POPULATION CHARACTERIZATION
    - HUMAN, 1089
  - VIRUS, EPSTEIN-BARR
    - BURKITT'S LYMPHOMA, 810
    - NASOPHARYNGEAL CARCINOMA, HUMAN, 810
  - VIRUS, HERPES SIMPLEX
    - PLAQUE FORMING ABILITY, HELA CELLS, 914*
  - VIRUS, MAMMARY TUMOR
    - NUCLEOTIDE SEQUENCE, MOUSE, 821
  - VIRUS, POLYOMA
    - ASSAY, MOUSE CELLS, 800
  - VIRUS, SIMIAN ADENO
    - CHARACTERIZATION, 848
  - VIRUS, SV40
    - ENCAPSIDATION, 853

- DNA POLYMERASE
  - ACTIVITY
    - EHRLICH ASCITES TUMOR CELLS, MOUSE, 1138*
    - REGENERATING LIVER, NOVIKOFF HEPATOMA, RAT, 1145*
  - BURKITT'S LYMPHOMA
    - TUMOR CELLS, CLINICAL STUDY, 835
  - DEFECTIVE MOUSE SARCOMA VIRUS, 790
  - INHIBITION
    - LYMPHOMA CELLS, MOUSE, 839
    - RNA TUMOR VIRUSES, POLYCYTIDYLIC ACID, 852
  - ISOLATION
    - AVIAN MYELOBLASTOSIS VIRUS, 831
    - MC29 TUMOR VIRUS, 831
  - MUTAGENESIS
    - LEUKEMIC CELLS, HUMAN, 1024
  - RNA AND DNA DEPENDENT
    - NORMAL AND MALIGNANT HUMAN CELLS, 1158*
  - RNA DEPENDENT
    - PURIFICATION, HUMAN LEUKEMIC CELLS, 826
  - VIRUS, AVIAN MYELOBLASTOSIS
    - RIBONUCLEASE H ACTIVITY, 834
  - VIRUS, C-TYPE RNA TUMOR
    - UTERINE TISSUE, MOUSE, 874
  - VIRUS, POLYOMA
    - EMBRYO CELLS, RAT, 876
  - VIRUS, SV40
    - MONKEY CELL CULTURE, 791

- DNA REPLICATION
  - LEUKEMIA
    - MUTAGENESIS, HUMAN, 1024

- DNA SYNTHESIS
  - N-ACETOXY-2-ACETYLAMINOFLUORENE
    - EPIDERMIS, MOUSE, RAT, PIG, 654
  - 7-BROMOETHYLBENZ(A)ANTHRACENE
    - EPIDERMIS, MOUSE, RAT, PIG, 654
  - CELL PROLIFERATION
    - X-RAY-PRODUCED STRAND BREAKS, RAT, 773*
  - 4-ETHYLSULPHONYLNAPHTHALENE-1-SULPHONAMIDE
    - BLADDER, MOUSE, 666
  - INHIBITION BY DEOXYADENOSINE
    - HEPATOMA CELLS, RAT, 1184*
  - LEUKEMIA, CHRONIC LYMPHOCYTIC
    - LYMPHOCYTES, HUMAN, 1011*
  - METHYLNITROSOUREA
    - HELA AND L-CELLS, 736*
  - BETA-PROPIOLACTONE
    - EPIDERMIS, MOUSE, RAT, PIG, 654
  - RADIATION, UV
    - EPIDERMIS, MOUSE, RAT, PIG, 654
    - HELA AND L-CELLS, 736*
  - RADIATION, X-RAY
    - E.COLI, 772*
  - REGULATION BY SERUM FACTOR
    - NORMAL AND LEUKEMIC BONE MARROW CELLS, 1139*
  - RNA, MESSENGER

    - PLASMACYTOMA, MOUSE, 940
  - VIRUS, APHTHOUS FEVER
    - EPITHELIOMA CELLS, RAT, 917*
  - VIRUS, HERPES SIMPLEX
    - HELA CELLS, 862

- SE
  - SV40-INFECTED CELLS
    - MONKEY, 791

- AMINE-BETA-HYDROXYLASE
  - NOREPINEPHRINE BIOSYNTHESIS
    - NEUROBLASTOMA, 1140*

- G
  - TESTING METHODS
    - CARCINOGENICITY, REVIEW, 651*

- LICH ASCITES TUMOR
  - CELL GENERATION RATE
    - MATHEMATICAL MODEL, 1063*
  - INVASIVENESS
    - ULTRASTRUCTURE, MOUSE, 1075
  - RIBONUCLEOTIDE REDUCTASE ACTIVITY
    - CELLS, MOUSE, 1138*

- OMETRIUM
  - CARCINOMA
    - CLINICAL STUDY, REVIEW, 644*

- OXAN
  - OVARIAN CARCINOMA
    - CYTOCHEMICAL INVESTIGATION, 1180*

- VIRONMENTAL HAZARD
  - RADIATION, LOW LEVEL IONIZING
    - REVIEW, 635*

- ZYME
  - SEE ALSO NAMES OF
  - ACTIVITY
    - EHRLICH ASCITES TUMOR CELLS, MOUSE, 1138*
  - AMINO ACID NAPHTHYLAMIDASE
    - HUMAN LIVER METASTASES, FETAL SERUM, 1143*
  - CARBOXYPEPTIDASE G1
    - EFFECT ON FOLATE COENZYMES, HUMAN LEUKEMIA CELLS, 1153*
  - CITRATE CONDENSING
    - MAMMARY ADENOCARCINOMA, MOUSE, 1096
  - COENZYME-A LEVELS
    - MALIGNANT TUMORS, RAT, MOUSE, HUMAN, 685
  - DIGESTION PATTERNS
    - VIRAL RNAS, REVIEW, 638*
  - DNA POLYMERASE
    - ACTIVITY IN REGENERATING RAT LIVER AND NOVIKOFF TUMOR, 1145*
  - DOPAMIME-BETA-HYDROXYLASE
    - NEUROBLASTOMA, 1140*
  - GLYCOGEN METABOLISM ENZYMES
    - HEPATOMA RAT, 1107*
  - INACTIVATION BY AUTOANTIBODIES
    - CANCER PATIENTS, REVIEW, 653*
  - LEUKOKININ
    - MURINE MASTOCYTOMA, 1149*
  - LYSOSOMAL ENZYME ACTIVITY
    - MALIGNANT NEOPLASMS, CLINICAL STUDY, 1172*
  - MEMBRANE MARKER ACTIVITY
    - DNA VIRUS-TRANSFORMED CELLS, MOUSE, 842
    - RNA VIRUS-TRANSFORMED CELLS, MOUSE, 842
  - NUCLEOTIDE PHOSPHODIESTERASE
    - ACTIVITY IN COMPENSATORY HYPERPLASIA OF RAT LIVER, 1157*
  - PATTERN
    - TRANSPLANTED RAT NEOPLASMS, FETAL RAT TISSUE, 1119*
  - REGAN ISOENZYME
    - PRIMARY MEDIASTINAL CHORIOCARCINOMA PATIENT, 989*
  - RIBONUCLEOTIDE REDUCTASE
    - ACTIVITY IN REGENERATING RAT LIVER AND NOVIKOFF TUMOR, 1145*
  - THYMIDINE KINASE
    - ACTIVITY IN REGENERATING RAT LIVER AND NOVIKOFF TUMOR, 1145*

- EPICORTISOL
  - POLYOMA VIRUS REPLICATION
    - MOUSE FIBROBLASTS, 799

- EPIDEMIOLOGY
  - CANCER
    - PROPHYLAXIS, REVIEW, 610*
  - CANCER INCIDENCE
    - MIGRANT POPULATIONS, REVIEW, 602
    - NEGROES, UNITED STATES AND AFRICA, 1047
  - CANCER MORTALITY
    - NEGROES, UNITED STATES, 1049
  - CANCER STATISTICS
    - CANADA, REVIEW, 643*
  - CERVICAL CARCINOMA
    - U.S.S.R., 1061*, 1062*
  - HODGKIN'S DISEASE
    - REVIEW, 640*
  - LUNG CANCER
    - TOBACCO SMOKING, 737*
  - OVARIAN CARCINOMA
    - REVIEW, 605

- EPIDERMIS
  - EPIDERMAL MOSAIC
    - HUMAN NEOPLASMS, REVIEW, 646*
  - 12-O-TETRADECANOYL-PHORBOL-13-ACETATE
    - SKIN TUMOR, ULTRASTRUCTURE, MOUSE, 718

- ERYTHROBLASTOSIS
  - VIRUS, RAUSCHER LEUKEMIA
    - DEFECTIVITY, MOUSE, 845

- ERYTHROLEUKEMIA
  - INHIBITION
    - HEMOLYTIC ANEMIA INDUCTION, MOUSE, 878
    - RAUSCHER LEUKEMIA VIRUS INFECTION, MOUSE, 878

- ERYTHROPOIETIN
  - VIRUS, LACTIC DEHYDROGENASE
    - ERYTHROID RESPONSE, MOUSE, 871

- ESTERASE
  - IMMUNOCHEMISTRY
    - HEPATOMA, RAT, 928

- ESTRADIOL
  - BINDING PROTEIN
    - MAMMARY TUMOR, RAT, HUMAN, 1073
  - POLYOMA VIRUS REPLICATION
    - MOUSE FIBROBLASTS, 799

- ESTRIOL
  - PANCREAS
    - DOG, 728*

- ESTROGEN
  - KIDNEY TUMOR DEPENDENCY
    - RECEPTOR, HAMSTER, 677
  - MAMMARY TUMOR
    - METABOLISM, RAT, 761*

- ETHIDIUM BROMIDE
  - CYTOCHROME SYNTHESIS
    - SV40 TRANSFORMED CELLS, HAMSTER, 844

- DL-ETHIONINE
  - ALPHA-NAPHTHYL-ISO-THIOCYANATE
    - COMPARATIVE HISTOCHEMISTRY, RAT LIVER, 744*

- N-ETHYL-N-NITROSOUREA
  - BRAIN TUMOR
    - ULTRASTRUCTURE, RAT, 696

- ETHYLNITROSOUREA
  - BRAIN TUMOR
    - RAT, 686
  - NERVOUS SYSTEM TUMORS
    - RAT, 686, 697
  - STOMACH
    - RAT, 711

- 4-ETHYLSULPHONYLNAPHTHALENE-1-SULPHONAMIDE
  - DNA SYNTHESIS
    - BLADDER, MOUSE, 666

- EXONUCLEASE
  - NOVIKOFF HEPATOMA ASCITES CELLS
    - GENETIC ENGINEERING OF CANCER CELLS, 1150*

- FALLOPIAN TUBE
  - CARCINOMA
    - REVIEW, 615*

- FERRIC OXIDE
  - RESPIRATORY TRACT TUMORS
    - DIETHYLNITROSAMINE, HAMSTER, 681

- ALPHA-FETOGLOBULIN
  - CARCINOEMBRYONIC ANTIGEN
    - REVIEW, 619*

- ALPHA-FETOPROTEIN
  - CANCER
    - REVIEW, 631*
  - HEPATOCELLULAR CARCINOMA
    - CLINICAL STUDY, 987*
  - HUMAN CHORIONIC GONADOTROPHIN PRODUCTION
    - HEPATOBLASTOMA, CASE REPORT, 1009*
  - SERUM LEVELS
    - CLINICAL STUDY, 927
  - SYNTHESIS
    - HUMAN CONCEPTUS, 1122*

- FIBER CARCINOGENESIS
  - MESOTHELIOMA
    - RAT, 707

- FIBROBLAST
  - ANCHORAGE DEPENDENCE
    - DIMETHYLNITROSAMINE, RAT EMBRYO, 881
    - 3-METHYLCHOLANTHRENE, RAT EMBRYO, 881
  - NIL 2 LINE
    - GLYCOLIPID SYNTHESIS, HAMSTER EMBRYO, 1087
    - TUMORIGENICITY, HAMSTER EMBRYO, 1087
  - PHORBOL MYRISTATE ACETATE
    - MOUSE, 726*
  - ROUS SARCOMA VIRUS-TRANSFORMED
    - ADENYLATE CYCLASE ACTIVITY, CHICK-EMBRYO, 795

- FIBROSARCOMA
  - METHYLCHOLANTHRENE
    - METASTASIS, SPLENECTOMY, MOUSE, 958
  - NICKEL SULFIDE
    - RAT, 695

- FIBROSARCOMA, MENINGEAL
  - ASTROCYTOMA
    - CASE REPORT, 1071

- N,N'-2,7-FLUORENYLENEBISACETAMIDE
  - CELL PROLIFERATION
    - BILE DUCTULAR CELLS, RAT, 704

- FREE RADICALS
  - MICROSOMAL DETOXIFICATION SYSTEM
    - DIETHYLNITROSAMINE CARCINOGENESIS, RAT, 733*

- FRUCTOSE DIPHOSPHATE ALDOLASE
  - NEUROBLASTOMA
    - MOUSE CELLS, 1088

- GALACTOSE
  - MEMBRANE TRANSPORT
    - POLYOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 883

- GASTROINTESTINAL CARCINOMA
  - DETECTION
    - BLOOD SELENIUM, 1106*
  - N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
    - RADIOGRAPHY, RAT, 754*

- GASTROINTESTINAL TRACT
  - ALPHA-FETOPROTEIN SYNTHESIS
    - HUMAN CONCEPTUS, 1122*

- GENETICS
  - CANCER FAMILY SYNDROME

- PROSPECTIVE STUDY, 1130*
- GROWTH, TUMOR
  - CELL-TO-CELL COMMUNICATION, RAT, 1094
- SOMATIC CELL HYBRIDS
  - REVIEW, 629*

MA
- PERIVENTRICULAR PLEOMORPHIC
  - N-ETHYL-N-NITROSOUREA, ULTRASTRUCTURE, RAT, 696
- VIRUS, ROUS SARCOMA
  - GREY MATTER EDEMA, DOG, 896*

COSE-6-PHOSPHATE DEHYDROGENASE
- NEUROBLASTOMA
  - MOUSE CELLS, 1088

-GLUCURONIDASE
- MAMMARY CARCINOMA
  - GROWTH AND REGRESSION, 1133*

TAMATE DEHYDROGENASE
- HEPATOMA
  - MITOCHONDRIA, DENSITY-GRADIENT CENTRIFUGATION, 1097
- NEUROBLASTOMA
  - MOUSE CELLS, 1088

COGEN PHOSPHORYLASE
- HEPATOMA
  - RAT, 1107*

COGEN SYNTHETASE
- HEPATOMA
  - RAT, 1107*

COLIPID SYNTHESIS
- NIL 2 LINE
  - TUMORIGENICITY, HAMSTER EMBRYO FIBROBLASTS, 1087

COPROTEIN
- AMINO ACID SEQUENCES
  - COLON CARCINOMA PATIENT, 985*
- PLASMA MEMBRANE
  - VIRUS SUSCEPTIBILITY, CHICK, 808

COSIDASE
- IMMUNOCHEMISTRY
  - HEPATOMA, RAT, 928

ADOTROPHIN
- HUMAN CHORIONIC
  - HEPATOBLASTOMA, CASE REPORT, 1009*
  - PRIMARY MEDIASTINAL CHORIOCARCINOMA PATIENT, 989*
- LEYDIG-CELL TUMOR TRANSFORMATION
  - ULTRASTRUCTURE, MOUSE, 1093
- PRODUCTION
  - HUMAN LUNG CARCINOMA CELL LINE, 1076

FT-VS-HOST REACTION
- VIRAL LYMPHOMA INDUCTION
  - MOUSE, 816

NULOCYTE
- DIFFERENTIATION
  - LEUKEMIC CELL, MOUSE, 1086

GRANULOCYTIC SARCOMA
- LEUKEMIA, MYELOGENOUS
  - AUTOPSY STUDY, HIROSHIMA-NAGASAKI, 769

GROWTH
- CHALONE MECHANISM DISTURBANCE
  - PHORBOL ESTER, MOUSE, 683
- INHIBITION BY LIPOPOLYSACCHARIDES
  - SV40-TRANSFORMED CELLS, RAT, 888*
- LARYNGEAL CARCINOMA METASTASES
  - WOMEN, 1169*
- LONG TERM
  - CHICK EMBRYO CELL, 1117*
- MAMMARY TUMOR
  - KINETICS, MOUSE, 1064*
- MEMBRANE JUNCTIONS
  - CANCER CELL, REVIEW, 604
- REGULATION
  - CYCLIC AMP, MOUSE CELLS, 909*
- SPLENIC INHIBITOR
  - CHRONIC MYELOCYTIC LEUKEMIC CELLS, HUMAN, 1095
- TUMOR CELL
  - MYO-INOSITOL, RESISTANCE, MOUSE, 1016*
- TUMOR FORMATION
  - NEOPLASTIC CELLS, RAT, 818

GROWTH, TUMOR
- CELL PROLIFERATION
  - REVIEW, 622*
- GENETICS
  - CELL-TO-CELL COMMUNICATION, RAT, 1094
- HYDROPEROXIDE ACCUMULATION
  - LIVER CELL MEMBRANE PHOSPHOLIPIDS, MOUSE, RAT, 1023
- IMMUNE RESPONSE
  - RAT, 965
- IMMUNOLOGY
  - ALLERGENIC FACTORS, REVIEW, 609*
- IMMUNOSUPPRESSION
  - REVIEW, 613*
- LOW CARBOHYDRATE DIET
  - RAT, 1129*

HEAD
- CARCINOMA
  - ASBESTOS EXPOSURE, HUMAN, 750*
- RHABDOMYOSARCOMA
  - HUMAN, REVIEW, 648*

HEART DISEASE
- TOBACCO SMOKING
  - CANCER, REVIEW, 628*

HEMOGLOBIN
- SYNTHESIS
  - MURINE VIRUS-INDUCED LEUKEMIC CELLS, 804

HEPATOBLASTOMA
- ALPHA-FETOPROTEIN PRODUCTION
  - HUMAN CHORIONIC GONADOTROPHIN PRODUCTION, CASE REPORT, 1009*

- HEPATOCARCINOGENESIS
    - DIETHYLNITROSAMINE
        - MICROSOMAL DETOXIFICATION SYSTEM, RAT, 733*
    - TRANSLATION MECHANISMS
        - LIVER, RAT, 727*

- HEPATOCELLULAR CARCINOMA
    - ALPHA-FETOPROTEIN
        - CLINICAL STUDY, 987*
    - IMMUNOLOGY
        - UGANDAN PATIENTS, 988*

- HEPATOMA
    - SEE ALSO LIVER
    - 2-ACETYLAMINOFLUORENE
        - FISH, 702
    - AFLATOXIN B1
        - FISH, 702
    - CIRCULAR DNA MOLECULES
        - MITOCHONDRIA, RAT, MOUSE, 1078
    - DIETHYLNITROSAMINE
        - ANTIGENICITY, RAT, 981
    - 4-DIMETHYLAMINOAZOBENZENE
        - TUMOR-SPECIFIC ANTIGEN, RAT, 933
    - DIMETHYLNITROSAMINE
        - FISH, 702
    - DNA SYNTHESIS INHIBITION
        - DEOXYADENOSINE, RAT, 1184*
    - GLYCOGEN METABOLISM ENZYMES
        - RAT, 1107*
    - MEMBRANE FRACTIONS
        - ENZYME ACTIVITY, RAT, 946
        - RAT, 928, 954
    - 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
        - INHIBITION BY CHLORAMPHENICOL, RAT, 692
    - MITOCHONDRIA
        - DENSITY-GRADIENT CENTRIFUGATION, 1097
    - PHOSPHOFRUCTOKINASE
        - LIVER, RAT, 1006*
    - PRIMARY LIVER CARCINOMA
        - RHODESIA, CLINICAL STUDY, 1192*

- HEPATOMA, MORRIS
    - DIHYDROOROTATE DEHYDROGENASE
        - LIVER, RAT, 1085

- HEPATOMA, NOVIKOFF
    - ASCITES CELLS
        - SURFACE ENDONUCLEASE, 1150*
    - RNA
        - LIVER, RAT, 1167*
    - RNA SEQUENCE ANALYSIS
        - B3 FRAGMENT, 1151*

- HETEROSACCHARIDE
    - CARCINOEMBRYONIC ANTIGEN
        - DIGESTIVE SYSTEM, HUMAN, 1014*

- HETEROTRANSPLANTATION
    - HUMAN BLADDER CANCER
        - HAMSTER, 997*
    - HUMAN TUMORS
        - MOUSE, 1012*
    - SARCOMA INDUCTION
        - RAT TUMOR CELLS, MOUSE, 887

- HISTAMINE
    - METABOLISM
        - NEOPLASTIC MAST CELLS, MOUSE, 1186*

- HISTONE
    - ACETYLATION
        - ACTINOMYCIN D, RAT LIVER, 715
        - AFLATOXIN B1, RAT LIVER, 715
    - DEACETYLATION
        - ACTINOMYCIN D, RAT LIVER, 715
        - AFLATOXIN B1, RAT LIVER, 715
    - TURNOVER OF LABELED METHYL GROUPS
        - CELL CULTURES, WHOLE ANIMALS, 1098*

- HISTONE ACETYLATION
    - NORMAL AND LEUKEMIC LYMPHOCYTES
        - DIFFERENCES, 1136*

- HODGKIN'S DISEASE
    - ANTIGENS, TUMOR-ASSOCIATED
        - HUMAN, 947
    - DETECTION
        - BLOOD SELENIUM, 1106*
    - EPIDEMIOLOGY
        - REVIEW, 640*
    - HISTOLOGY
        - REVIEW, 621*
    - IRON METABOLISM
        - CLINICAL STUDY, 1185*

- HORMONE
    - SEE ALSO NAMES OF
    - DYSFUNCTION
        - MAMMARY GLAND HYPERPLASIA, HUMAN, 1030*, 1038*

- HORMONE, STEROID
    - EXCRETION
        - STOMACH CANCER PATIENTS, 1034*

- HYDRAZINE
    - EMBRYONAL CELL TISSUE CULTURE
        - RAT, 738*

- HYDRAZINE SULFATE
    - LUNG TUMOR
        - MOUSE, 762*
    - MAMMARY TUMOR
        - MOUSE, 762*

- HYDROCARBON, POLYCYCLIC AROMATIC
    - NUCLEIC ACID STACKING
        - SPECTRAL MODIFICATIONS, 752*

- HYDROCARBON, POLYNUCLEAR AROMATIC
    - AIR POLLUTION
        - NETHERLANDS, 739*

- HYDROPEROXIDE
    - TUMOR GROWTH
        - LIVER CELL MEMBRANE PHOSPHOLIPIDS, MOUSE, RAT, 1023

- 4-HYDROXYAMINOQUINOLINE 1-OXIDE
    - VIRUS, HERPES SIMPLEX
        - PLAQUE FORMING ABILITY, HELA CELLS 914*

- YDROXYMETHYLBENZO(A)PYRENE
  - METABOLISM
    - LIVER, RAT, 662

- DROXYUREA
  - MAMMARY TUMOR
    - CELL KINETICS, MOUSE, 1055*

- PERFIBRINOGENEMIA
  - THROMBOSIS
    - PATIENTS WITH NEOPLASIA, 1144*

- UNE RESPONSE
  - CANCER ETIOLOGY
    - REVIEW, 627*
  - GROSS VIRUS-INDUCED LYMPHOMA
    - RAT, 955
  - GROWTH, TUMOR
    - RAT, 965
  - THYMIC LYMPHOMA DEVELOPMENT
    - N,N'-DIMETHYLNITROSOUREA, MOUSE, 921
  - TRANSPLANTED MAMMARY TUMOR
    - CELL PROLIFERATION, RAT, 1002*
  - URINARY BLADDER TUMOR
    - LYMPHOCYTE CYTOTOXICITY, HUMAN, 967
  - VIRUS, FRIEND LEUKEMIA
    - ROSETTE FORMING CELLS, MOUSE, 849
  - VIRUS, MURINE SARCOMA
    - MOUSE, 974
  - VIRUS, RAUSCHER LEUKEMIA
    - MOUSE, 861

- UNITY
  - CELL-MEDIATED
    - MALIGNANT MELANOMA PATIENTS, 1005*
    - PLASMA TUMOR CELLS, MOUSE, 920
  - CELL MEDIATED TUMOR
    - MALIGNANT MELANOMA, CLINICAL STUDY, 934
  - COENZYME Q10
    - DIBENZPYRENE, FRIEND LEUKEMIA VIRUS, MOUSE, 953
  - HUMORAL IMMUNE DEFICIENCY
    - LEUKEMIA, REGIONAL ILEITIS, FAMILIAL OCCURRENCE, 1007*
  - LEUKEMIA, ACUTE
    - HUMAN, 1000*
  - LEUKEMIA, GRAFFI
    - PSORALEN-TREATED LEUKEMIC CELLS, MOUSE, 980
  - SARCOMA GROWTH
    - GUINEA PIG, 979
    - HUMAN, 979
  - TUMOR ALLOGRAFT
    - MACROPHAGE CYTOTOXICITY, MOUSE, 948
  - VIRUS, FRIEND LEUKEMIA
    - MOUSE, 931

- UNITY, CELLULAR
  - INHIBITION
    - SERA, SARCOMA PATIENTS, 930
  - MELANOMA
    - HUMAN, 944
  - TUMOR-ASSOCIATED ANTIGENS
    - HUMAN, 966
  - TUMOR-SPECIFIC
    - SARCOMA, GUINEA PIG, 979
    - SARCOMA, HUMAN, 979

- IMMUNOGLOBULIN
  - CELL SURFACE
    - LYMPHOCYTES, SYNTHESIS, BIOCHEMISTRY, 949
  - SECRETION
    - THYMUS CELLS, MOUSE, 999*
  - SYNTHESIS
    - THYMUS CELLS, MOUSE, 999*

- IMMUNOGLOBULIN IGA
  - MYELOMA PROTEIN
    - J CHAIN, MOLECULAR WEIGHT, HUMAN, 1001*

- IMMUNOGLOBULIN IGG
  - MYELOMA
    - MOLECULAR WEIGHT, 994*

- IMMUNOLOGY
  - ASTROCYTOMA
    - BRAIN, HUMAN, 977
  - CYSTADENOCARCINOMA OF OVARY
    - TUMOR-SPECIFIC ANTIGENS, HUMAN, 929
  - GROWTH, TUMOR
    - ALLERGENIC FACTORS, REVIEW, 609*
  - HEPATOCELLULAR CARCINOMA
    - UGANDAN PATIENTS, 988*

- IMMUNOSUPPRESSION
  - GROWTH, TUMOR
    - REVIEW, 613*
  - NEOPLASIA
    - REVIEW, 601
  - NERVOUS SYSTEM TUMORS
    - N-METHYL-N-NITROSOUREA, RAT, 675
  - TUMOR PRODUCTION
    - ADENO VIRUS 2-TRANSFORMED CELLS, RAT, 982
  - URINARY BLADDER TUMOR
    - N-METHYL-N-NITROSOUREA, RAT, 675
  - VIRUS, MAMMARY TUMOR
    - MOUSE, 968
  - VIRUS, MURINE SARCOMA
    - ONCOGENESIS, MOUSE, 939

- INFECTIOUS MONONUCLEOSIS
  - ACRIDINE ORANGE BINDING
    - LEUCOCYTE NUCLEOPROTEINS, 902*
  - RETICULUM CELL SARCOMA
    - CASE REPORT, 908*

- INHALATION CARCINOGENESIS
  - CERIUM 144
    - DOG, 770

- MYO-INOSITOL
  - GROWTH, TUMOR CELL
    - RESISTANCE, MOUSE, 1016*

- INTERFERON
  - PRODUCTION
    - CHRONIC LYMPHATIC LEUKEMIA, HUMAN, 1080
    - TRANSFORMED CELLS, MOUSE, 846

IODINE
    NORMAL DIET EFFECT ON THYROID GLAND
        IODINE DEFICIENT RATS, 1121*

125I-IODODEOXYURIDINE
    LABELED SARCOMA CELLS
        CYTOTOXIC TUMOR-SPECIFIC ANTIBODY QUANTITATION, MOUSE, 978

IRON
    METABOLISM
        HODGKIN'S DISEASE PATIENTS, 1185*
    PLEURAL FLUID
        NEOPLASTIC DISEASE PATIENTS, 1196*

JAW
    RADIATION-INDUCED MESENCHYMAL TUMOR
        MOUSE, 775*

KERATOSIS
    LARYNGEAL CARCINOMA
        REVIEW, 617*

KIDNEY
    ESTROGEN-DEPENDENT TUMOR
        ESTROGEN RECEPTOR, HAMSTER, 677
    LUCKE RENAL TUMOR CELLS
        CULTIVATION, FROG, 1084
    LYSOSOME FUNCTION
        CARBON TETRACHLORIDE, MOUSE, 749*
    MESENCHYMAL TUMOR
        DIMETHYLNITROSAMINE, ULTRASTRUCTURE, RAT, 663
    PELVIC PAPILLARY TUMOR
        C-TYPE VIRUS PARTICLES, ULTRASTRUCTURE, HUMAN, 815
    PLASMA CELL GRANULOMA
        CASE REPORT, 1191*
    WILM'S TUMOR
        CULTIVATION, CHICK EMBRYO, 1081

KIDNEY TUMOR
    DIETHYLSTILBESTROL
        HAMSTER, 857
    DIMETHYLNITROSAMINE
        ARTERIOGRAPHY, RAT, 693
        PYELOGRAPHY, RAT, 693
        RAT, 656
    RADIATION, X-RAY
        PARABIOSIS, RAT, 768
    VIRUS, SV40
        HAMSTER, 857

LACTATE DEHYDROGENASE
    NEUROBLASTOMA
        MOUSE CELLS, 1088

LARYNX CARCINOMA
    ASBESTOS EXPOSURE
        HUMAN, 750*
    KERATOSIS
        REVIEW, 617*
    METASTASIS
        CLINICAL STUDY, 1171*
        GROWTH, WOMEN, 1169*

LESIONS, PATHOLOGICAL
    FIXATION
        ADVANCED INBRED CHINESE HAMSTERS, 1128*

LEUCYL-BETA-NAPHTHYLAMIDASE
    IMMUNOCHEMISTRY
        HEPATOMA, RAT, 928

LEUKEMIA
    BONE MARROW CHROMOSOME ABNORMALITIES
        HUMAN, 1022
    CARBOHYDRATE METABOLISM
        HUMAN, 1162*
    CELL DIFFERENTIATION
        PROTEIN MGI, MOUSE, 960
    CYTOGENETIC STUDIES, 1188*
    2,7-DIACETYLAMINOFLUORENE
        RAT, 1025
    DIHYDROFOLATE REDUCTASE
        CELLS, MOUSE, 1197*
    DNA REPLICATION
        MUTAGENESIS, HUMAN, 1024
    INCIDENCE
        RHODESIAN AFRICAN, 1060*
    INCIDENCE REDUCTION IN RAT
        SPLENECTOMY, RAT, 1152*
    MACROPHAGE MIGRATION INHIBITION
        CELL CYCLE, MOUSE, 961
    PRELEUKEMIC SIDEROBLASTIC ANEMIA
        DIMORPHIC RED CELL, CASE REPORT, 1028
    RADIATION
        CLINICAL STUDY, 780*
    TOXICITY
        BLOOD SERUM, CLINICAL STUDY, 1176*

LEUKEMIA, ACUTE
    ACRIDINE ORANGE BINDING
        LEUCOCYTE NUCLEOPROTEINS, 902*
    IMMUNITY
        HUMAN, 1000*
    MICROBIAL FACTOR IN COMPLICATIONS
        CLINICAL STUDY, 1178*
    REGIONAL ILEITIS
        HUMORAL IMMUNE DEFICIENCY, FAMILIAL OCCURRENCE, 1007*
    RNA METABOLISM
        CELL CYCLE, HUMAN, 1083
    TIME-SPACE CLUSTERING
        STATISTICAL ANALYSES, GEORGIA, 1048

LEUKEMIA, ACUTE LYMPHATIC
    HL-A2, HL-A12 PHENOTYPE ASSOCIATION
        GEOGRAPHIC FACTOR, 1123*

LEUKEMIA, ACUTE LYMPHOBLASTIC
    CYTOKINETICS
        LYMPHOBLASTS, HUMAN, 1057*
    HUMAN LYMPHOID CELL LINES
        THYMUS-DERIVED LYMPHOCYTE CHARACTERISTICS, 1155*
    VIRUS-LIKE PARTICLES
        CASE REPORT, 895*

LEUKEMIA, ACUTE MONOBLASTIC
    MONOBLAST
        ULTRASTRUCTURE, HUMAN, 1079

LEUKEMIA, ACUTE MYELOBLASTIC
    MYELOBLAST AGGLUTINATION

    - NEURAMINIDASE, 991*
  - VIRUS-LIKE PARTICLES
    - MITOCHONDRIA, PATIENTS, 841

UKEMIA, ACUTE MYELOMONOCYTIC
  - INCIDENCE
    - CHILDREN, 1040

EUKEMIA, CHRONIC LYMPHATIC
  - INTERFERON PRODUCTION
    - HUMAN, 1080

EUKEMIA, CHRONIC LYMPHOCYTIC
  - DNA SYNTHESIS
    - LYMPHOCYTES, HUMAN, 1011*
  - LEUKOCYTE LABELING WITH 51CHROMIUM
    - LYMPHOCYTE KINETICS, 1051*
  - LYMPHOCYTE, COMPLEMENT-RECEPTOR
    - BLOOD, HUMAN, 952
  - LYMPHOCYTE KINETICS
    - CLINICAL STUDY, 1058*
  - RESIDUAL T CELLS
    - CASE REPORT, 998*

EUKEMIA, CHRONIC MYELOCYTIC
  - SPLENECTOMY
    - CHILDREN, 1156*
  - SPLENIC INHIBITOR OF CELL GROWTH
    - HUMAN, 1095

EUKEMIA, CONGENITAL
  - CASE REPORT, 1170*

EUKEMIA, EOSINOPHILIC
  - FIBROSING ENDOCARDITIS
    - SHORT Y CHROMOSOME, 1160*

EUKEMIA, GRAFFI
  - IMMUNITY
    - PSORALEN-TREATED LEUKEMIC CELLS, MOUSE, 980

EUKEMIA, INTRACRANIAL
  - CEREBROSPINAL FLUID
    - CASE REPORTS, 1195*

EUKEMIA, LYMPHATIC
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - BONE MARROW, MOUSE, 975
    - THYMUS, MOUSE, 975

EUKEMIA, LYMPHOBLASTIC
  - INCIDENCE
    - AFRICAN CHILDREN, 1042

EUKEMIA, MYELOGENOUS
  - GRANULOCYTIC SARCOMA
    - AUTOPSY STUDY, HIROSHIMA-NAGASAKI, 769

EUKEMOGENESIS
  - VIRUS, RADIATION LEUKEMIA
    - BCG, MOUSE, 836

EUKOCYTE
  - LABELING WITH 51CHROMIUM
    - CHRONIC LYMPHOCYTIC LEUKEMIA KINETICS, 1051*
  - LYMPHOBLAST CULTURE ESTABLISHMENT
    - GIBBON, 783

LEUKOKININ
  - MASTOCYTOMA, MURINE
    - ENZYME CHARACTERIZATION, 1149*

LEYDIG-CELL TUMOR
  - TRANSFORMATION
    - GONADOTROPHIN, MOUSE, 1093

LIP
  - CARCINOMA
    - SOLUBLE MEMBRANE ANTIGENS, 957

LIPID, ETHER-LINKED
  - SUBCELLULAR DISTRIBUTION
    - TUMOR, MOUSE PREPUTIAL GLAND, 1099*

LIPID, MACROMOLECULAR
  - LEUKEMIC CELLS, L1210
    - MOUSE, 1104*

LIPOPOLYSACCHARIDE
  - GROWTH INHIBITION
    - SV40-TRANSFORMED CELLS, RAT, 888*

LIVER
  - DIETHYLNITROSAMINE
    - MORPHOLOGICAL ALTERATIONS, FISH, 760*
  - DIHYDROOROTATE DEHYDROGENASE
    - HEPATOMA, RAT, 1085
  - DIMETHYLNITROSAMINE
    - MORPHOLOGICAL ALTERATIONS, FISH, 760*
  - DRUG OXIDATION
    - URETHANE, RAT, 694
  - DL-ETHIONINE
    - HISTOCHEMISTRY, RAT, 744*
  - ALPHA-FETOPROTEIN SYNTHESIS
    - HUMAN CONCEPTUS, 1122*
  - FUNCTIONAL CONDITION
    - BRAIN TUMOR PATIENTS, 1165*
  - HEPATOCARCINOGENESIS
    - TRANSLATION MECHANISMS, RAT, 727*
  - HEPATOCELLULAR CARCINOMA
    - ALPHA-FETOPROTEIN, CLINICAL STUDY, 987*
    - IMMUNOLOGY, UGANDAN PATIENTS, 988*
  - HEPATOMA
    - 2-ACETYLAMINOFLUORENE, FISH, 702
    - AFLATOXIN B1, FISH, 702
    - DIMETHYLNITROSAMINE, FISH, 702
    - MEMBRANE FRACTIONS, RAT, 928, 954
    - PHOSPHOFRUCTOKINASE CONCENTRATION, RAT, 1006*
    - RHODESIA, CLINICAL STUDY, 1192*
  - HEPATOMA INHIBITION
    - CHLORAMPHENICOL, RAT, 692
  - HEPATOMA, NOVIKOFF
    - RNA, RAT, 1167*
  - LYSOSOMAL ENZYME ACTIVITY
    - MALIGNANT NEOPLASMS, CLINICAL STUDY, 1172*
  - LYSOSOME FUNCTION
    - CARBON TETRACHLORIDE, MOUSE, 749*
  - MICROSOMAL DETOXIFICATION SYSTEM
    - DIETHYLNITROSAMINE CARCINOGENESIS,

- RAT, 733*
- MITOCHONDRIAL OXIDATION
  - 3,3'-DIAMINOBENZIDINE, RAT, 688
- ALPHA-NAPHTHYL-150-THIOCYANATE
  - HISTOCHEMISTRY, RAT, 744*
- N-NITROSODODECAMETHYLENEIMINE
  - ONCOGENESIS, MOUSE, 714
- PRIMARY CARCINOMA
  - EARLY SYMPTOMS, HUMAN, 1166*
- PROTEIN LEUCINE UPTAKE
  - 4-DIMETHYLAMINOAZOBENZENE, RAT, 689
- PROTEIN SYNTHESIS
  - URETHANE, RAT, 694
- REGENERATION
  - DEOXYRIBONUCLEOTIDES, RAT, 1074
- TUMOR INDUCTION
  - CELO VIRUS, HAMSTER, 869

LIVER TUMOR
- 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - AZO-DYE-BINDING PROTEIN
    - DISTRIBUTION, RAT, 655

LUCKE RENAL TUMOR
- CULTIVATION, IN VITRO
  - FROG, 1084

LUNG
- ADENOCARCINOMA
  - 20-METHYLCHOLANTHRENE, MOUSE, 658
- ADENOMA
  - 20-METHYLCHOLANTHRENE, MOUSE, 658
- BRONCHIAL CARCINOMA
  - ARSENIC, CASE REPORT, 763*
- BRONCHIAL MUCOSA
  - AIR POLLUTION TOXICITY, HUMAN CULTURE, 678
  - BENZO(A)PYRENE TOXICITY, HUMAN CULTURE, 678
- CARCINOID TUMOR
  - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608
- HYDRAZINE SULFATE-INDUCED TUMOR
  - MOUSE, 762*
- MARIJUANA SMOKE
  - HUMAN, 659
- MESOTHELIOMA
  - INCIDENCE, ITALY, REVIEW, 606
- SMALL CELL CARCINOMA
  - CELL KINETIC STUDIES, 1112*
- TUMOR DEVELOPMENT
  - BENZOYLHYDRAZINE, MOUSE, 682
- URETHANE-INDUCED ADENOMA INHIBITION
  - PLUTONIUM DIOXIDE INHALATION, MOUSE, 669

LUNG CANCER
- CARCINOEMBRYONIC ANTIGEN
  - HUMAN, 943
- ENDOCRINCUS FACTORS
  - REVIEW, 612*
- GONADOTROPHIN PRODUCTION
  - HUMAN CELL LINE, 1076
- METASTASIS
  - BRONCHOSCOPIC CRITERIA, 1164*
- MORTALITY
  - UNITED STATES, 690
- TOBACCO SMOKING
  - EPIDEMIOLOGY, 737*
  - SELF-FRUSTRATION, CLINICAL STUDY, 753*

LUTEOMA
- TRANSPLANTATION
  - ULTRASTRUCTURE, MOUSE, 1187*

LYMPH NODE
- VIRUS, ONCORNA
  - MONKEY, 850

LYMPH NODE CELL
- SELECTIVE SENSITIVITY
  - PHYTOHEMAGGLUTININ, 938

LYMPH NODE, REGIONAL
- LYMPH OUTFLOW
  - STOMACH, INTRA-OPERATIVE STUDY, 1181*
- MALIGNANT MELANOMA METASTASES
  - CLINICAL STUDY, 1182*

LYMPHOBLAST
- CULTURE ESTABLISHMENT
  - LEUKOCYTES, GIBBON, 783
- CYTOKINETICS
  - ACUTE LYMPHOBLASTIC LEUKMEIA PATIENT, 1057*

LYMPHOCYTE
- ATPASE ACTIVITY
  - SOLID MALIGNANT TUMOR PATIENTS, 1019*
- CHROMOSOME ABERRATIONS
  - CANCER PATIENTS, 1082
- COMPLEMENT-RECEPTOR
  - BLOOD, LEUKEMIA PATIENTS, 952
- CONCANAVALIN A BINDING
  - CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1013*
  - RAT, HUMAN, 1013*
- CYTOTOXICITY
  - IMMUNE RESPONSE, URINARY BLADDER TUMOR, HUMAN, 967
  - MOLONEY LEUKEMIA VIRUS ANTIGEN, MOUSE, 956
  - ROUS SARCOMA CELLS, QUAIL, 886
  - SERUM FACTORS, NEOPLASTIC DISEASE PATIENTS, 1020*
  - TUMOR ANTIGEN, RAT HEPATOMA, 951
- DNA REPAIR SYNTHESIS
  - LEUKEMIA PATIENTS, 1011*
- IMMUNOGLOBULIN, CELL SURFACE
  - SYNTHESIS, BIOCHEMISTRY, 949
- INTERFERON PRODUCTION
  - CHRONIC LYMPHATIC LEUKEMIA, HUMAN, 1080
- KINETICS
  - CHRONIC LYMPHOCYTIC LEUKEMIA PATIENTS, 1058*
- MEMBRANE RECEPTOR SITES
  - SURFACE MACROMOLECULE MIGRATION, 932
- REACTIVITY
  - MELANOMA TUMOR CELLS, HUMAN, 924
- ROSETTE-FORMING CELLS
  - THYMUS-DERIVED, HUMAN, 962
- TRANSFORMATION

    - SODIUM BOROHYDRIDE REDUCTION, 1003*
    - SODIUM PERIODATE OXIDATION, 1003*

- MPHOID TUMOR
  - INCIDENCE
    - XENOPUS LAEVIS, 1198*
  - MORPHOLOGY
    - XENOPUS LAEVIS, 1198*

- MPHOMA
  - DNA POLYMERASE INHIBITION
    - 1-BETA-D-ARABINOFURANOSYLCYTOSINETRIPHOSPHATE, MOUSE, 839
  - HERPESVIRUS SAIMIRI
    - MONKEY, 788
  - MAXILLOFACIAL
    - INCIDENCE, GUATEMALA, 893*
  - NEURAMINIDASE
    - IMMUNOGENICITY, MOUSE, 983*
  - THYMUS
    - IMMUNE RESPONSE, MOUSE, 921
  - TRANSPLANTATION
    - TUMOR-SPECIFIC RESISTANCE, MOUSE, 1018*
  - VIRAL INDUCTION
    - GRAFT-VS-HOST REACTION, MOUSE, 816
  - VIRUS, GROSS
    - IMMUNE RESPONSE, RAT, 955

- YMPHOMA, MALIGNANT
  - BENZOYLHYDRAZINE
    - MOUSE, 682
  - INCIDENCE
    - AFRICAN CHILDREN, 1042
  - VIRUS, HERPES SAIMIRI
    - CELL CULTURE ESTABLISHMENT, MONKEY, 889*
    - LYMPH NODE, MONKEY, 858

- ACROPHAGE
  - CYTOTOXICITY
    - TUMOR ALLOGRAFT IMMUNITY, MOUSE, 948
  - DIFFERENTIATION
    - LEUKEMIC CELL, MOUSE, 1086
  - MIGRATION INHIBITION
    - LEUKEMIC CELL, MOUSE, 961

- ALATE DEHYDROGENASE
  - NEUROBLASTOMA
    - MOUSE CELLS, 1088

- AMMARY CARCINOMA
  - ANDROGEN
    - HUMAN, 1026
  - ESTROGEN-BINDING PROTEINS
    - RELATION TO HORMONAL RESPONSIVENESS, RAT, 1111*
  - BETA-GLUCURONIDASE
    - GROWTH AND REGRESSION, 1133*
  - PLEURAL EFFUSION
    - HUMAN, 1118*
  - PROTEINS, SH AND NH2 GROUPS
    - HISTOCHEMICAL STUDY, HUMAN, 1179*

- AMMARY GLAND
  - ADENOCARCINOMA
    - CITRATE CONDENSING ENZYME, MOUSE, 1096
    - PYRUVATE DEHYDROGENASE, MOUSE, 1096
  - ANDROGEN METABOLISM
    - HUMAN, 1092
  - HYPERPLASIA
    - HORMONE DEFICIENCY, HUMAN, 1030*, 1038*

- MAMMARY TUMOR
  - BIOCHEMICAL PROPERTIES
    - HUMAN, 1108*
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - ACTINOMYCIN D PROTECTION, RAT, 998
    - DDT, RAT, 684
    - METABOLISM, RAT, 761*
    - POLY I:C, RAT, 757*
  - ESTRADIOL BINDING PROTEIN
    - RAT, HUMAN, 1073
  - GROWTH KINETICS
    - MOUSE, 1064*
  - HYDRAZINE SULFATE
    - MOUSE, 762*
  - HYDROXYUREA
    - CELL KINETICS, MOUSE, 1055*
  - PRIMARY EXPLANTATION AND CONTINUOUS CULTIVATION
    - HUMAN, 1105*
  - PROLINE HYDROXYLASE ACTIVITY
    - MOUSE, 1110*
  - RNA
    - MILK PARTICLE DNA HOMOLOGY, HUMAN, 822
  - TRANSPLANTATION
    - CELL PROLIFERATION, RAT, 1002*
    - IMMUNE RESPONSE, RAT, 1002*
  - VIRUS-LIKE PARTICLES
    - HUMAN, 828
  - VIRUS, VESICULAR STOMATITIS
    - PSEUDOTYPE FORMATION, HUMAN CELLS, 950

- MANTOUX REACTION
  - LYMPHOSUPPRESSIVE-STIMULATORY FACTORS
    - CANCER PATIENTS, 973

- MAREK'S DISEASE
  - HERPESVIRUS-ASSOCIATED ONCOGENESIS
    - CHICKEN, 890*

- MASTOCYTOMA, MURINE
  - LEUKOKININ
    - ENZYME CHARACTERIZATION, 1149*

- MELANOMA
  - ANTIBODY
    - MONKEY, 972
  - DEVELOPMENT
    - ISOLATED MOUSE POPULATION, 1174*
  - IMMUNITY, CELLULAR
    - HUMAN, 944
  - LYMPHOCYTE-MEDIATED CYTOTOXICITY DETECTION
    - DIFFUSION CHAMBER METHOD, MOUSE, 1004*
  - SERUM PROTEIN CHANGES
    - ELECTROPHORESIS, HAMSTER, 969
  - TYROSINASE ACTIVITY AND ENDOGENOUS

- INHIBITION
  - HUMAN, 1125*

- MELANOMA, MALIGNANT
  - BLOCKING SERUM ACTIVITY
    - CLINICAL STUDY, 934
  - CELL-MEDIATED IMMUNITY
    - CLINICAL STUDY, 1005*
  - COMPARATIVE MORPHOGENESIS
    - SKIN, HUMAN, 1031*
  - CONGENITAL
    - CASE REPORT, 1194*
  - IMMUNITY, CELL-MEDIATED TUMOR
    - CLINICAL STUDY, 934
  - METASTASIS
    - REGIONAL LYMPH NODES, CLINICAL STUDY, 1182*
  - TUMOR CELLS
    - LYMPHOCYTE REACTIVITY, HUMAN, 924

- 6-MERCAPTOPURINE
  - CARCINOGENICITY
    - MOUSE, RAT, 764*

- MESOTHELIOMA
  - ASBESTOS
    - RAT, 707
  - CHRONIC ASBESTOS EXPOSURE
    - PARTIAL INTESTINAL OBSTRUCTION, CASE REPORT, 745*
  - FIBER CARCINOGENESIS
    - RAT, 707

- MESOTHELIOMA, PLEURAL
  - INCIDENCE
    - ITALY, REVIEW, 606

- METABOLISM
  - ANDROGEN
    - HUMAN BREAST TISSUE, 1092
  - BENZO(A)PYRENE
    - EPITHELIAL CELLS, HUMAN, 721
    - FIBROBLASTS, HUMAN, 721
    - LIVER, RAT, 662
  - CARBOHYDRATE
    - LEUKEMIA, HUMAN, 1162*
    - POLYOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 883
  - HISTAMINE
    - NEOPLASTIC MAST CELLS, MOUSE, 1186*
  - 6-HYDROXYMETHYLBENZO(A)PYRENE
    - LIVER, RAT, 662
  - IRON
    - HODGKIN'S DISEASE PATIENTS, 1185*
  - MAMMARY TUMOR
    - ESTROGEN, THERAPY, RAT, 761*
  - 6-METHYLBENZO(A)PYRENE
    - LIVER, RAT, 662

- METALS
  - COPPER
    - PLEURAL FLUID, NEOPLASTIC DISEASE PATIENTS, 1196*
  - IRON
    - PLEURAL FLUID, NEOPLASTIC DISEASE PATIENTS, 1196*
  - IRON METABOLISM
    - HODGKIN'S DISEASE PATIENTS, 1185*
  - ZINC
    - PLEURAL FLUID, NEOPLASTIC DISEASE PATIENTS, 1196*

- METALS, TRACE
  - ASBESTOS CARCINOGENESIS, 708

- METASTASIS
  - IMMUNOLOGICAL MANIPULATION
    - HERPESVIRUS TRANSFORMED CELLS, HAMSTER, 990*
  - LARYNX CARCINOMA
    - CLINICAL STUDY, 1171*
    - GROWTH, WOMEN, 1169*
  - LIVER
    - AMINO ACID NAPHTHYLAMIDASE, HUMAN, 1143*
  - LUNG CANCER
    - BRONCHOSCOPIC CRITERIA, 1164*
  - MELANOMA, MALIGNANT
    - REGIONAL LYMPH NODES, CLINICAL STUDY, 1182*
  - METHYLCHOLANTHRENE-INDUCED FIBROSARCOMA
    - SPLENECTOMY, MOUSE, 958
  - SKIN
    - CLINICAL STUDY, 1199*

- 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - HEPATOMA
    - INHIBITION BY CHLORAMPHENICOL, RAT, 692
  - LIVER TUMOR
    - AZO-DYE-BINDING PROTEIN DISTRIBUTION, RAT, 655

- N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
  - COLON CARCINOMA
    - RAT, 706
  - GASTROINTESTINAL CARCINOMA
    - RADIOGRAPHY, RAT, 754*
  - RECTAL CARCINOMA
    - RAT, 706

- N-METHYL-N-NITROSOUREA
  - NERVOUS SYSTEM TUMORS
    - IMMUNOSUPPRESSION, RAT, 675
  - URINARY BLADDER TUMORS
    - IMMUNOSUPPRESSION, RAT, 675

- METHYLATION
  - DNA
    - 7-METHYLGUANINE LOSS, RAT LIVER, 673
  - RNA, TRANSFER
    - MITOCHONDRIA, HELA CELLS, 1091

- METHYLBENZANTHRACENE
  - CARBONIUM ION CHARGE LOCALIZATION, 722*

- 6-METHYLBENZO(A)PYRENE
  - METABOLISM
    - LIVER, RAT, 662

- METHYLCHOLANTHRENE
  - FIBROSARCOMA
    - METASTASIS, SPLENECTOMY, MOUSE, 958

SARCOMA
TUMOR-SPECIFIC ANTIBODY, GUINEA PIG, 729*

METHYLCHOLANTHRENE
ANCHORAGE DEPENDENCE
FIBROBLASTS, RAT EMBRYO, 881
INITIATION FACTOR ACTIVITY
LIVER RIBOSOME, RAT, 691
SARCOMA SENSITIVITY
ARYL HYDROCARBON HYDROXYLASE INDUCIBILITY, MOUSE, 665

-METHYLCHOLANTHRENE
ADENOCARCINOMA
ULTRASTRUCTURE, LUNG, MOUSE, 658
ADENOMA
ULTRASTRUCTURE, LUNG, MOUSE, 658
CERVICAL CARCINOMA
PROGESTERONE, MOUSE, 712

O-METHYLGLUCOSE
TRANSPORT
ROUS SARCOMA VIRUS-TRANSFORMED CHICK FIBROBLASTS, 830

METHYLGUANINE
DNA METHYLATION
RAT LIVER, 673

THYLMETHANESULPHONATE
DNA METHYLATION
7-METHYLGUANINE LOSS, RAT LIVER, 673

THYLNITROSOUREA
DNA REPAIR SYNTHESIS
HELA AND L-CELLS, 736*
FORMATION IN STOMACH
RAT, 766*
STOMACH
RAT, 711

TOCHONDRIA
CIRCULAR DNA MOLECULES
HEPATOMA, RAT, MOUSE, 1078
HEPATOMA
DENSITY-GRADIENT CENTRIFUGATION, 1097
HEPATOMA, NOVIKOFF
BIOCHEMICAL ORGANIZATION, ENZYME ACTIVITY, 1116*
TRANSFER RNA METHYLATION
HELA CELLS, 1091
VIRUS-LIKE PARTICLES
ACUTE MYELOBLASTIC LEUKEMIA PATIENTS, 841

NOAMINE OXIDASE
HEPATOMA
MITOCHONDRIA, DENSITY-GRADIENT CENTRIFUGATION, 1097

NOBLAST
LEUKEMIA, ACUTE MONOBLASTIC
ULTRASTRUCTURE, HUMAN, 1079

RTALITY
CANCER
AMERICAN INDIANS, 1068*
MEXICO, 1067*
NEGROES, UNITED STATES, 1049
NEW ZEALAND, 1059*
CERVICAL CARCINOMA
RELATION TO HUSBAND'S OCCUPATION, 1050
U.S.S.R., 1061*, 1062*
LUNG CANCER
UNITED STATES, 690
MALIGNANT TUMORS
MONGOLIAN PEOPLE'S REPUBLIC, 1066*

MOUTH
ORAL LESIONS
MAST CELL INCIDENCE, HUMAN, 1032*

MUCOPOLYSACCHARIDE
ASTROCYTOMA
HUMAN, 1069
NEURILEMMOMA
HUMAN, 1069
OLIGODENDROGLIOMA
HUMAN, 1069

MUSCLE
LYSOSOMAL ENZYME ACTIVITY
MALIGNANT NEOPLASMS, CLINICAL STUDY, 1172*

MUTAGEN
CLASSIFICATION
BACTERIAL TEST SYSTEM, 717
DETECTION
DNA INTRAMOLECULAR INTERACTION ENERGY, 735*

MUTAGEN, CHEMICAL
CARBARYL
DROSOPHILA MELANOGASTER, 742*

MUTAGENESIS
DNA POLYMERASE
LEUKEMIC CELLS, HUMAN, 1024
PHTHALATE_ESTER
CELLULAR TOXICITY, RAT, 748*

MYELOMA
ALLIED CONDITIONS
REVIEW, 633*
IMMUNOGLOBULIN IGA
J CHAIN, MOLECULAR WEIGHT, HUMAN, 1001*
IMMUNOGLOBULIN IGG
MOLECULAR WEIGHT, 994*
NONSECRETORY PLASMA CELL
ULTRASTRUCTURE, CLINICAL STUDY, 1010*
ONCOGENESIS
SV40 PROTECTION, MOUSE, 875

MYELOMA CELL
IMMUNOGLOBULIN LIGHT CHAIN PROTEIN
MOUSE, 995*

MYELOMA, MULTIPLE
SIDEROBLASTIC ANEMIA
CLINICAL STUDY, 1033*

NANDROLONE DECANOATE
RAPID STEM CELL DIFFERENTIATION
MOUSE, 730*

BETA-NAPHTHOFLAVONE
INITIATION FACTOR ACTIVITY
LIVER RIBOSOME, RAT, 691

ALPHA-NAPHTHYL-ISO-THIOCYANATE
DL-ETHIONINE
COMPARATIVE HISTOCHEMISTRY, RAT LIVER, 744*

NAPHTHYLAMINE
URINARY BLADDER CARCINOMA
RUBBER WORKERS, 758*

BETA-NAPHTHYLAMINE
URINARY BLADDER TUMOR
MORPHOLOGY, HISTOCHEMISTRY, DOG, 664

NASAL CAVITY
DIETHYLNITROSAMINE
GLANDULAR TUMORS, HAMSTER, 709

NASAL SEPTUM
MALIGNANT TUMORS
REVIEW, 647*

NASOPHARYNGEAL CARCINOMA
BURKITT'S LYMPHOMA
HOMOLOGOUS RNA, HUMAN, 854
VIRUS, EPSTEIN-BARR
HOMOLOGOUS DNA, HUMAN, 810
VIRUS, RAUSCHER LEUKEMIA
HOMOLOGOUS RNA, HUMAN, 854

NECK
CARCINOMA
ASBESTOS EXPOSURE, HUMAN, 750*
RHABDOMYOSARCOMA
HUMAN, REVIEW, 648*

NEOPLASIA
IMMUNOSUPPRESSION
REVIEW, 601

NEOPLASMS, MALIGNANT
LYSOSOMAL ENZYME ACTIVITY
LIVER AND MUSCLE TISSUE, CLINICAL STUDY, 1172*

NEOPLASTIC DISEASE
COPPER CONTENT OF PLEURAL FLUID
CLINICAL STUDY, 1196*
IRON CONTENT OF PLEURAL FLUID
CLINICAL STUDY, 1196*
LYMPHOCYTE CYTOTOXICITY
SERUM FACTORS, HUMAN, 1020*
ZINC CONTENT OF PLEURAL FLUID
CLINICAL STUDY, 1196*

NERVE GROWTH FACTOR
NEUROBLASTOMA
STIMULATION IN VITRO, HUMAN, 1148*

NERVOUS SYSTEM TUMORS
ETHYLNITROSOUREA
RAT, 686, 697
IMMUNOSUPPRESSION
N-METHYL-N-NITROSOUREA, RAT, 675

NEURAMINIDASE
LYMPHOMA
IMMUNOGENICITY, MOUSE, 983*
MALIGNANCY
REVIEW, 603
MYELOBLAST AGGLUTINATION
LEUKEMIA PATIENTS, 991*

NEURILEMMOMA
MUCOPOLYSACCHARIDE
HUMAN, 1069

NEUROBLASTOMA
8-BENZYLTHIO CYCLIC AMP
TUMORIGENICITY, MOUSE, 1070
CYCLE AMP
MOUSE CELL CULTURE, 1113*
DIBUTYRYL CYCLIC AMP
TUMORIGENICITY, MOUSE, 1070
DIFFERENTIATION
IN VIVO, IN VITRO, MOUSE, 1141*
DOPAMINE-BETA-HYDROXYLASE
NOREPINEPHRINE BIOSYNTHESIS, 1140*
FRUCTOSE DIPHOSPHATE ALDOLASE
MOUSE CELLS, 1088
GLUCOSE-6-PHOSPHATE DEHYDROGENASE
MOUSE CELLS, 1088
GLUTAMATE DEHYDROGENASE
MOUSE CELLS, 1088
LACTATE DEHYDROGENASE
MOUSE CELLS, 1088
MALATE DEHYDROGENASE
MOUSE CELLS, 1088
NERVE GROWTH FACTOR
STIMULATION IN VITRO, HUMAN, 1148*
PRIMITIVE CELL SIGNIFICANCE IN MARROW ASPIRATES
CHILDREN, 1101*

NICKEL SULFIDE
FIBROSARCOMA
RAT, 695
RHABDOMYOSARCOMA
RAT, 695

NITROSAMINES
CARCINOGENICITY
REVIEW, 611*
FORMATION
SARCOSINE, MOUSE, 679

N-NITROSO-N-ETHYLUREA
CHROMATID LESIONS
VICIA FABA, 756*

N-NITROSO-N-METHYLUREA
CHROMATID LESIONS
VICIA FABA, 756*

N-NITROSOALKYLUREA
TERATOGENESIS
EMBRYOTOXICITY, RAT, 732*

N-NITROSODODECAMETHYLENEIMINE
LIVER

    - ONCOGENESIS, MOUSE, 714
  - STOMACH
    - ONCOGENESIS, MOUSE, 714

- REPINEPHRINE BIOSYNTHESIS
  - DOPAMINE-BETA-HYDROXYLASE
    - NEUROBLASTOMA, 1140*

- CLEIC ACID
  - DYE INTERACTION
    - TUMOR CELLS, 1183*
  - RADIATION-INDUCED DAMAGE
    - BLOOD, RAT, 781*

- CLEOSIDE DIPHOSPHATASE
  - BRAIN TUMOR
    - HUMAN, 1072

- CLEOTIDE PHOSPHODIESTERASE
  - ACTIVITY IN COMPENSATORY HYPERPLASIA
    - RAT LIVER, 1157*
  - DIMETHYLNITROSAMINE
    - TRANSFORMED CELL, HAMSTER, 851
  - VIRUS, POLYOMA
    - TRANSFORMED CELL, HAMSTER, 851

- CUPATIONAL HAZARD
  - ASBESTOS
    - REVIEW, 650*
  - INDUSTRIAL POISONS
    - PRECANCEROUS CONDITIONS OF UTERINE CERVIX, 759*
  - STOMACH CANCER
    - COAL MINING, 1054*
  - URINARY BLADDER CARCINOMA
    - NAPHTHYLAMINES, RUBBER WORKERS, 758*

- IGODENDROGLIOMA
  - MUCOPOLYSACCHARIDE
    - HUMAN, 1069

- NITHINE CARBAMOYLTRANSFERASE
  - ACTIVITY
    - NORMAL, NEOPLASTIC LIVER, 1137*

- TEOBLASTOCLASTOMA
  - MALIGNANT DEGENERATION
    - HUMAN, 1161*

- TEOSARCOMA
  - ELECTRON MICROSCOPE INVESTIGATION
    - PARTICLES, HUMAN, 1100*

- ARIAN CARCINOMA
  - ENDOXAN
    - CYTOCHEMICAL INVESTIGATION, 1180*
  - EPIDEMIOLOGY
    - REVIEW, 605
  - TALC
    - HUMAN, 680

- RY
  - CYSTADENOCARCINOMA
    - TUMOR-SPECIFIC ANTIGENS, HUMAN, 929
  - MALIGNANT TUMORS
    - DIAGNOSTICS, REVIEW, 652*
  - TRANSPLANTABLE TUMOR DEVELOPMENT
    - GONADOTROPIC PITUITARY TUMOR-GRAFTED RATS, 765*

- PAGET'S DISEASE
  - EXTRAMAMMARY
    - FAMILIAL OCCURRENCE, CASE REPORTS, 1077

- PANCREAS
  - ESTRIOL
    - DOG, 728*

- PAPILLOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - SKIN GRAFTS, MOUSE, 926
  - POXVIRUS
    - MARSUPIAL, 905*

- PARABIOSIS
  - KIDNEY TUMOR
    - X-IRRADIATION, RAT, 768

- PENIS
  - SQUAMOUS CELL CARCINOMA
    - CASE REPORT, 1190*

- PHORBOL ESTER
  - CARCINOGENESIS
    - HELA CELLS, 747*
  - CHALONE MECHANISM DISTURBANCE
    - MOUSE, 683
  - EPIDERMAL GROWTH CONTROL DISTURBANCE
    - MOUSE, 683

- PHORBOL MYRISTATE ACETATE
  - FIBROBLAST
    - MOUSE, 726*

- PHOSPHOFRUCTOKINASE
  - HEPATOMA
    - LIVER, RAT, 1006*

- PHOSPHORODIAMIDIC ACID
  - CARCINOGENICITY
    - MOUSE, RAT, 764*

- PHTHALATE ESTER
  - MUTAGENESIS
    - CELLULAR TOXICITY, RAT, 748*
  - TERATOGENESIS
    - CELLULAR TOXICITY, RAT, 748*

- PHYTOHEMAGGLUTININ
  - SELECTIVE SENSITIVITY
    - LYMPHOID CELLS, 938

- PLACENTA
  - VIRUS, C-TYPE PARTICLE
    - BABOON, 897*
    - HUMAN, 872

- PLANT TUMORS
  - TOBACCO CROWN GALL
    - RIBONUCLEASE ACTIVITY, 1115*

- PLASMA CELL GRANULOMA
  - KIDNEY
    - CASE REPORT, 1191*

PLASMA MEMBRANE
  ANTIGEN
    ISOLATION, LYMPHOID CELLS, 971
  GLYCOPROTEIN
    VIRUS SUSCEPTIBILITY, CHICK, 808

PLASMACYTOMA
  CRYOGLOBULINEMIA
    IGM-ANTIBODY DEFICIENCY, CASE REPORT, 986*
  DNA SYNTHESIS
    MESSENGER RNA, MOUSE, 940
  RNA, TRANSFER
    CHROMATOGRAPHIC PATTERNS, MOUSE, 996*

PLUTONIUM DIOXIDE
  URETHANE-INDUCED ADENOMA INHIBITION
    LUNG, MOUSE, 669

POLLUTION, AIR
  ASBESTOS
    DETECTION AND MEASUREMENT, 740*
  BRONCHIAL MUCOSA
    TOXICITY, HUMAN CULTURE, 678
  POLYNUCLEAR AROMATIC HYDROCARBONS
    NETHERLANDS, 739*

POLYADENOSINE
  LEUKEMIC CELLS
    HUMAN, 1124*

POLYADENYLIC ACID
  CHARACTERIZATION, RNA
    RAT LIVER, MORRIS HEPATOMA, 1159*
  VIRUS-LIKE PARTICLES
    MILK, HUMAN, 843
  VIRUS, RNA TUMOR
    MILK, HUMAN, 843
  VISNA VIRUS RNA, 898*

POLYCYTIDYLIC ACID
  DNA POLYMERASE INHIBITION
    RNA TUMOR VIRUSES, 852

POLYINOSINIC:POLYCYTIDYLIC ACID
  DMBA-INDUCED MAMMARY TUMOR
    RAT, 757*

POLYPS, ENDOMETRIAL
  CARCINOMA
    CASE REPORTS, 1039*

POLYRIBOSOME
  DIMETHYLNITROSAMINE-INDUCED DISAGGREGATION
    CYCLOHEXIMIDE, RAT LIVER, 676

POLYSOMES
  RNA CONTAINING POLYADENYLIC ACID SEQUENCES
    RAT LIVER, MORRIS HEPATOMA, 1159*

POXVIRUS
  PAPILLOMA
    MARSUPIAL, 905*

PREGNANCY
  AFLATOXIN
    RAT, 719
  DIETHYLSTILBESTROL
    CLINICAL STUDY, 705

PROGESTERONE
  CERVICAL CARCINOMA
    20-METHYLCHOLANTHRENE, MOUSE, 712
  POLYOMA VIRUS REPLICATION
    MOUSE FIBROBLASTS, 799

BETA-PROPIOLACTONE
  DNA SYNTHESIS
    EPIDERMIS, MOUSE, RAT, PIG, 654

PROSTATE
  DIETHYLSTILBESTROL
    HAMSTER, 857
  PRIMARY AND METASTATIC CANCER
    ACID PHOSPHATASE ISOZYME CHANGES, 1102*
  VIRUS, SV40
    HAMSTER, 857

PROTEIN
  ESTRADIOL BINDING
    MAMMARY TUMOR, RAT, HUMAN, 1073
  IMMUNOGLOBULIN A MYELOMA
    J CHAIN, MOLECULAR WEIGHT, HUMAN, 1001*
  IMMUNOGLOBULIN LIGHT CHAIN
    MYELOMA CELLS, MOUSE, 995*
  MGI
    LEUKEMIC CELL DIFFERENTIATION, MOUSE, 960
  MYELOMA LIGHT CHAIN
    MRNA TRANSLATION, 993*
  SERUM GLOBULIN CHANGES
    MELANOMA-BEARING HAMSTERS, 969
  SH AND NH2 GROUPS
    MAMMARY CARCINOMA, HUMAN, 1179*

PROTEIN SYNTHESIS
  AMINO ACID ACTIVATION
    ISOLATED NOVIKOFF TUMOR NUCLEOLI, 1154*
  URETHANE
    LIVER, RAT, 694

PSORALEN
  IMMUNITY
    GRAFFI LEUKEMIA, MOUSE, 980

PURINE
  DIFFERENTIAL EFFECTS
    POLYOMA VIRUS-TRANSFORMED CELLS, MOUSE, 805

PYRUVATE DEHYDROGENASE
  MAMMARY ADENOCARCINOMA
    MOUSE, 1096

RADIATION
  BLOOD NUCLEIC ACIDS
    RAT, 781*
  CANCER INDUCTION
    REVIEW, 639*
  LEUKEMIA
    CLINICAL STUDY, 780*
  MESENCHYMAL JAW TUMOR INDUCTION

- MOUSE, 775*
- OBSTETRIC RADIOGRAPHY
  - CHILDHOOD CANCER, 771*
  - CHILDHOOD CANCER, REVIEW, 634*

IATION, GAMMA
- DNA
  - RAT, 778*

IATION, IONIZING
- DNA
  - SENSITIVITY TO BREAKAGE, 779*

IATION, LOW LEVEL
- CARCINOGENESIS
  - REVIEW, 632*

IATION, LOW LEVEL IONIZING
- ENVIRONMENTAL HAZARD
  - REVIEW, 635*

IATION, UV
- DNA
  - B. SUBTILIS, 777*
  - PHOTOALKYLATION OF PURINES, 782*
- DNA DEGRADATION
  - YEAST, 774*
- DNA REPAIR SYNTHESIS
  - HELA AND L-CELLS, 736*
- DNA SYNTHESIS
  - EPIDERMIS, MOUSE, RAT, PIG, 654

IATION, X-RAY
- DNA REPAIR SYNTHESIS
  - E.COLI, 772*
- KIDNEY TUMOR
  - PARABIOSIS, RAT, 768
- THYMUS
  - VIRUS PARTICLES, MOUSE, 767

TAL CARCINOMA
- INCIDENCE
  - MINNESOTA, 1041
- N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
  - RAT, 706
- SERUM AMINO ACIDS
  - HUMAN, 1163*

ISTANCE
- CORTICOSTEROID
  - MOUSE, 723*
- GROWTH, TUMOR CELL
  - MYO-INOSITOL, MOUSE, 1016*
- TRANSPLANTATION ANTIGEN
  - BO-IV CARCINOMA, SYNGENEIC SYSTEM, MOUSE, 1015*
- TUMOR-SPECIFIC
  - TRANSPLANT LYMPHOMAS, MOUSE, 1018*

PIRATORY TRACT TUMORS
- DIETHYLNITROSAMINE
  - FERRIC OXIDE, HAMSTER, 681

ICULUM CELL SARCOMA
- INFECTIOUS MONONUCLEOSIS
  - CASE REPORT, 908*

ERSE TRANSCRIPTASE
- PURIFICATION
  - LEUKEMIC CELLS, HUMAN, 826
- RAUSCHER LEUKEMIA VIRUS RNA
  - LEUKEMIC CELLS, HUMAN, 825

RHABDOMYOSARCOMA
- HEAD AND NECK
  - HUMAN, REVIEW, 648*
- NICKEL SULFIDE
  - RAT, 695
- VIRUS, C-TYPE
  - HUMAN, 829

RIBONUCLEASE
- ACETYLATION
  - N-ACETOXY-2-FLUORENYLACETAMIDE, 701
- ACTIVITY
  - TOBACCO CROWN GALL, 1115*

RIBONUCLEASE H
- DNA POLYMERASE
  - AVIAN MYELOBLASTOSIS VIRUS, 834

RIBONUCLEOTIDE REDUCTASE
- ACTIVITY
  - EHRLICH ASCITES TUMOR CELLS, MOUSE, 1138*
  - REGENERATING LIVER, NOVIKOFF HEPATOMA, RAT, 1145*

RIBOSOME
- INITIATION FACTOR ACTIVITY
  - 3-METHYLCHOLANTHRENE, RAT LIVER, 691
  - BETA-NAPHTHOFLAVONE, RAT LIVER, 691

RNA
- DNA LINKAGE
  - POLYOMA VIRUS, MOUSE CELL, 820
- HEPATOMA, NOVIKOFF
  - LIVER, RAT, 1167*
- KIRSTEN SARCOMA VIRUS-TRANSFORMED CELLS
  - OLIGO(DT)CELLULOSE CHROMATOGRAPHY, RAT, 812
- MAMMARY TUMOR
  - MILK PARTICLE DNA HOMOLOGY, HUMAN, 822
- NUCLEOTIDE SEQUENCE ANALYSIS
  - B3 FRAGMENT, NOVIKOFF HEPATOMA CELLS, 1151*
- "70S-ASSOCIATED" 4S
  - BASE COMPOSITION, AVIAN MYELOBLASTOSIS VIRUS, 817
- 30-40S PRECURSOR
  - ROUS SARCOMA VIRUS, 884
- SIZE DISTRIBUTION
  - ADENOVIRUS-TRANSFORMED RAT CELL, 847
- VIRAL
  - ENZYMATIC DIGESTION PATTERNS, REVIEW, 638*
- VIRUS, RAUSCHER LEUKEMIA
  - BURKITT'S LYMPHOMA, HUMAN, 854
  - NASOPHARYNGEAL CARCINOMA, HUMAN, 854
  - REVERSE TRANSCRIPTASE, HUMAN LEUKEMIC CELLS, 825

- VIRUS, ROUS SARCOMA
  - 5'TERMINUS CHARACTERIZATION, 833
- VIRUS, SV40
  - RNA POLYMERASE, E.COLI, 901*
- VIRUS, VISNA
  - POLYADENYLIC ACID, 898*

RNA, MESSENGER
- DNA SYNTHESIS
  - PLASMACYTOMA, MOUSE, 940
- MYELOMA LIGHT CHAIN PROTEIN
  - TRANSLATION, 993*

RNA METABOLISM
- CELL CYCLE
  - ACUTE LEUKEMIA, HUMAN, 1083
- VITAMIN A DEFICIENCY
  - HAMSTER TRACHEAL EPITHELIUM, 1109*

RNA POLYMERASE
- SV40 RNA
  - E.COLI, 901*
- TUMOR CELLS
  - HUMAN, 1135*

RNA, RIBOSOMAL
- METHYLATION
  - MOUSE LIVER, SKIN, AND SKIN TUMOR, 1134*

RNA SYNTHESIS
- VIRUS, SV40
  - TRANSFORMED HUMAN CELLS, 787

RNA, TRANSFER
- METHYLATION
  - MITOCHONDRIA, HELA CELLS, 1091
- PLASMACYTOMA
  - CHROMATOGRAPHIC PATTERNS, MOUSE, 996*
- SERYL AND PHENYLALANYL PATTERNS
  - TUMOR CELLS, 1142*

SALIVARY GLAND
- BENIGN AND MALIGNANT MIXED TUMORS
  - REVIEW, 618*

SARCOMA
- ANTIGEN
  - DISTRIBUTION, HUMAN CELLS, 984*
- CELLULAR IMMUNITY INHIBITION
  - SERA, SARCOMA PATIENTS, 930
- CYTOTOXICITY, TUMOR-SPECIFIC CELLULAR
  - HUMAN, 942
- INDUCTION
  - HETEROTRANSPLANTATION, RAT TUMOR CELLS, MOUSE, 887
- METHYLCHOLANTHRENE
  - TUMOR-SPECIFIC ANTIBODY, GUINEA PIG, 729*
- VIRUS, PROGRESSIVE PNEUMONIA
  - TRANSPLANTATION ANTIGENS, MOUSE, 970
- VIRUS, VISNA
  - TRANSPLANTATION ANTIGENS, MOUSE, 970

SARCOSINE
- NITROSATION
  - MOUSE, 679

SELENIUM, BLOOD
- NORMAL AND CANCER PATIENTS
  - DETECTION OF GASTROINTESTINAL CANCER AND HODGKIN'S DISEASE, 1106*

SIALIC ACID
- MALIGNANCY
  - REVIEW, 603

SKIN
- COAL TAR
  - MOUSE, 687
- MELANOMA, MALIGNANT
  - COMPARATIVE MORPHOGENESIS, HUMAN, 1031*
- METASTASIS
  - CLINICAL STUDY, 1199*

SKIN CANCER
- PREMALIGNANT CONDITIONS
  - HUMAN, 776*

SKIN TUMOR
- ARYL HYDROCARBON HYDROXYLASE
  - MOUSE, 724*
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - MOUSE, 724*
- 12-O-TETRADECANOYL-PHORBOL-13-ACETATE
  - EPIDERMIS, ULTRASTRUCTURE, MOUSE, 718

SPERM
- MAMMARY NODULIGENIC ACTIVITY
  - MAMMARY TUMOR STRAIN MICE, 907*

SPLEEN
- LEUKEMIA, CHRONIC MYELOCYTIC
  - INHIBITOR, HUMAN, 1095

SPLENECTOMY
- LEUKEMIA, CHRONIC MYELOCYTIC
  - CHILDREN, 1156*
- LEUKEMIC INCIDENCE REDUCTION
  - RAT, 1152*
- METHYLCHOLANTHRENE-INDUCED FIBROSARCOMA
  - METASTASIS, MOUSE, 958

SQUAMOUS CELL CARCINOMA
- PENIS
  - CASE REPORT, 1190*

STILBESTROL
- MATERNAL ADMINISTRATION
  - CANCER PATIENTS, 713

STOMACH
- CARCINOID TUMOR
  - ENDOCRINE FUNCTION, HUMAN, REVIEW, 608
- ETHYLNITROSOUREA
  - RAT, 711
- LYMPH OUTFLOW
  - INTRA-OPERATIVE STUDY, 1181*
- METHYLNITROSOUREA
  - RAT, 711

- METHYLNITROSOUREA FORMATION
  - RAT, 766*
- MULTIPLE GASTRIC NEOPLASMS
  - COLOVESICAL FISTULA, CASE REPORT, 1193*
- N-NITROSODODECAMETHYLENEIMINE
  - ONCOGENESIS, MOUSE, 714

MACH CANCER
- INCIDENCE
  - FINLAND, 1046
- OCCUPATIONAL HAZARD
  - COAL MINING, 1054*
- STEROID HORMONE EXCRETION
  - HUMAN, 1034*

CYTIA
- FORMATION
  - C-TYPE VIRUS, HUMAN CELLS, 879
  - ROUS SARCOMA VIRUS, HUMAN CELLS, 879

C
- CERVICAL CARCINOMA
  - HUMAN, 680
- OVARIAN CARCINOMA
  - HUMAN, 680

ATOGENESIS
- N-NITROSOALKYLUREA
  - EMBRYOTOXICITY, RAT, 732*
- PHTHALATE ESTER
  - CELLULAR TOXICITY, RAT, 748*

TIS
- CANCER INCIDENCE
  - NEGROES, 1065*

O-TETRADECANOYL-PHORBOL-13-ACETATE
- SKIN TUMOR
  - EPIDERMIS, ULTRASTRUCTURE, MOUSE, 718

MINE PYROPHOSPHATASE
- BRAIN TUMOR
  - HUMAN, 1072

MIDINE KINASE
- ACTIVITY
  - EHRLICH ASCITES TUMOR CELLS, MOUSE, 1138*
  - REGENERATING LIVER, NOVIKOFF HEPATOMA, RAT, 1145*

US
- LEUKEMIA, LYMPHATIC
  - MOUSE, 975
- LYMPHOMA
  - IMMUNE RESPONSE, MOUSE, 921
- RADIATION, X-RAY
  - VIRUS PARTICLES, MOUSE, 767

OID
- C-CELL NEOPLASIA
  - CASE REPORT, 1027

CCO
- CIGARETTE SMOKE
  - DNA, HUMAN LUNG EXPLANTS, 1029
- CROWN GALL
  - RIBONUCLEASE ACTIVITY, 1115*
- LUNG CANCER MORTALITY
  - UNITED STATES, 690
- MARIJUANA SMOKE
  - DNA, HUMAN LUNG EXPLANTS, 1029
  - LUNG, HUMAN, 659

TOBACCO SMOKING
- CANCER
  - REVIEW, 628*
- HEART DISEASE
  - REVIEW, 628*
- LUNG CANCER
  - EPIDEMIOLOGY, 737*
  - SELF-FRUSTRATION, CLINICAL STUDY, 753*

TRANSFORMATION
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - CELL PROLIFERATION, MOUSE, 720
- HUMAN MALIGNANT CELL CULTURES
  - HELPER ACTIVITY, 1146*
- LEYDIG-CELL TUMOR
  - GONADOTROPHIN, MOUSE, 1093
- LYMPHOCYTE
  - SODIUM BOROHYDRIDE REDUCTION, 1003*
  - SODIUM PERIODATE OXIDATION, 1003*
- VIRUS, CELO
  - EMBRYONIC SKIN, HAMSTER, 880
  - SUBCUTANEOUS TISSUE CELLS, HAMSTER, 880
- VIRUS, MOLONEY SARCOMA
  - 2-DEOXYGLUCOSE UPTAKE ALTERATIONS, RAT CELLS, 866
- VIRUS, POLYOMA
  - CARBOHYDRATE CATABOLISM, HAMSTER CELLS, 883
  - ENZYME ACTIVITIES, MOUSE CELLS, 868
  - GALACTOSE UPTAKE, HAMSTER CELLS, 883
- VIRUS, SV40
  - BIOCHEMICAL CHARACTERIZATION, MOUSE, 827
  - ENZYME ACTIVITIES, MOUSE CELLS, 868
  - HAMSTER EMBRYO CELLS, 870
  - SUGAR TRANSPORT, MOUSE CELLS, 911*

TRANSPLANTATION
- ANTIGEN
  - BO-IV CARCINOMA, SYNGENEIC SYSTEM, MOUSE, 1015*
  - SARCOMAS, MOUSE, 970
- LUTEOMA
  - ULTRASTRUCTURE, MOUSE, 1187*
- LYMPHOMA
  - TUMOR-SPECIFIC RESISTANCE, MOUSE, 1018*
- MAMMARY TUMOR
  - CELL PROLIFERATION, RAT, 1002*
  - IMMUNE RESPONSE, RAT, 1002*

TRIAMCINOLONE ACETONIDE
- BINDING TO MACROMOLECULES
  - NORMAL AND NEOPLASTIC HUMAN TISSUES, 1147*

- TRYPSIN
  - HELA CELL ANTIBODY
    - NORMAL CELL REACTIVITY, HUMAN, 945

- TUBERCULIN REACTION
  - LYMPHOSUPPRESSIVE-STIMULATORY FACTORS
    - CANCER PATIENTS, 973

- TUMOR
  - CELL DEATH
    - REVIEW, 636*
  - VASCULAR ANATOMY
    - HUMAN, REVIEW, 626*

- TUMOR VIRUS
  - SEE VIRUS

- TYROSINASE
  - ACTIVITY AND ENDOGENOUS INHIBITION
    - MELANOMA, HUMAN, 1125*

- URETHANE
  - DRUG OXIDATION
    - LIVER, RAT, 694
  - EMBRYONIC DEVELOPMENT
    - MOUSE, 755*
  - PROTEIN SYNTHESIS
    - LIVER, RAT, 694

- URINARY BLADDER
  - 4-ETHYLSULPHONYLNAPHTHALENE-1-SULPHONAMIDE
    - DNA SYNTHESIS, MOUSE, 666
  - HETEROTRANSPLANTATION OF HUMAN CANCER TISSUE
    - HAMSTER, 997*

- URINARY BLADDER CARCINOMA
  - INCIDENCE
    - OKLAHOMA, 1044
  - MORTALITY
    - OKLAHOMA, 1044
  - OCCUPATIONAL HAZARD
    - NAPHTHYLAMINES, RUBBER WORKERS, 758*

- URINARY BLADDER TUMOR
  - IMMUNE RESPONSE
    - LYMPHOCYTE CYTOTOXICITY, HUMAN, 967
  - IMMUNOSUPPRESSION
    - N-METHYL-N-NITROSOUREA, RAT, 675
  - BETA-NAPHTHYLAMINE
    - MORPHOLOGY, HISTOCHEMISTRY, DOG, 664

- URINARY TRACT CARCINOMA
  - DIAGNOSTIC CYTOLOGY
    - HUMAN, 746*

- UTERINE CERVIX
  - PATHOLOGY
    - HUMAN, 1036*
  - PRECANCEROUS CONDITIONS
    - OCCUPATIONAL HAZARD, 759*

- UTERUS
  - CANCER
    - MORPHOGENESIS, HUMAN, 1037*

- VAGINA
  - DIMETHYLNITROSAMINE
    - HUMAN, 716

- VIRUS
  - HUMAN CANCER
    - REVIEW, 637*, 642*
  - -LIKE PARTICLES
    - ACUTE LYMPHOBLASTIC LEUKEMIA, CASE REPORT, 895*
    - MAMMARY TUMOR, HUMAN, 828
    - MITOCHONDRIA, LEUKEMIA PATIENTS, 841
    - POLYADENYLIC ACID, HUMAN MILK, 843

- VIRUS, A-TYPE PARTICLE
  - MORPHOGENESIS
    - ARBOVIRUS INFECTION, MOUSE CELL LINES, 837
  - RADIATION, X-RAY
    - THYMUS, MOUSE, 767

- VIRUS, ADENO 2
  - RNA SIZE DISTRIBUTION
    - RAT CELL, 847
  - TUMOR PRODUCTION
    - IMMUNOSUPPRESSED RATS, 982

- VIRUS, ADENO 12
  - BRAIN TUMOR
    - INTRACRANIAL TRANSPLANTATION, HAMSTER, 906*
  - CONCANAVALIN A RECEPTORS
    - ULTRASTRUCTURE, HAMSTER CELL LINES, 809

- VIRUS, APHTHOUS FEVER
  - DNA SYNTHESIS
    - EPITHELIOMA CELLS, RAT, 917*

- VIRUS, AVIAN INFLUENZA
  - SERIAL PASSAGES
    - HUMAN EPITHELIAL TUMOR CELLS, 900*
    - HUMAN MYELOBLASTS, 900*

- VIRUS, AVIAN LEUKOSIS
  - CELL MEMBRANE GLYCOPROTEIN
    - VIRUS SUSCEPTIBILITY, CHICK, 808
  - DNA POLYMERASE ISOLATION
    - DNA ISOLATION, 831

- VIRUS, AVIAN MYELOBLASTOSIS
  - CORE COMPONENT
    - ULTRASTRUCTURE, 873
  - DNA POLYMERASE
    - RIBONUCLEASE H ACTIVITY, 834
  - DNA POLYMERASE ISOLATION
    - DNA ISOLATION, 831
  - "70S-ASSOCIATED" 4S RNA
    - MINOR BASE COMPOSITION, 817
  - VESICULAR STOMATITIS
    - PSEUDOTYPE FORMATION, CHICK CELL, 840

- VIRUS, AVIAN SARCOMA
  - GENOME EXPRESSION
    - TUMOR CELLS, RAT, 877

- VIRUS, C-TYPE

  - ISOLATION
    - MYELOMA CELLS, MOUSE, 899*
  - RHABDOMYOSARCOMA
    - HUMAN, 829
  - SPONTANEOUS RELEASE
    - TRANSFORMED CELLS, MOUSE, 819

- US, C-TYPE PARTICLE
  - ACTIVATION
    - HUMAN TUMOR CELL LINES, 892*
  - ANTIGENS, GROUP-SPECIFIC
    - ASCITIC TUMORS, MOUSE, 1017*
  - ISOLATION
    - HUMAN LEUKEMIC CELL LINE, 792
  - MORPHOGENESIS
    - ARBOVIRUS INFECTION, MOUSE CELL LINES, 837
  - PAPILLARY TUMOR OF RENAL PELVIS
    - ULTRASTRUCTURE, HUMAN, 815
  - PLACENTA
    - BABOON, 897*
    - HUMAN, 872
  - RADIATION, X-RAY
    - THYMUS, MOUSE, 767

- JS, C-TYPE RNA
  - ISOLATION
    - MOUSE, 915*
  - SYNCYTIA FORMATION
    - ROUS SARCOMA VIRUS, HUMAN CELLS, 879

- JS, C-TYPE RNA TUMOR
  - ANTIGEN
    - UTERINE TISSUE, MOUSE, 874
  - DNA POLYMERASE
    - UTERINE TISSUE, MOUSE, 874

- JS, CELO
  - PLAQUE VARIANTS
    - ONCOGENICITY, HAMSTER, 823
  - TRANSFORMATION
    - EMBRYONIC SKIN, HAMSTER, 880
    - SUBCUTANEOUS TISSUE CELLS, HAMSTER, 880
  - TUMOR INDUCTION
    - LIVER, HAMSTER, 869

- S, EPSTEIN-BARR
  - ANTIBODY
    - CHILDREN, WEST MALAYSIAN, 919
  - ANTIBODY, NEUTRALIZING
    - PATIENTS, 859
  - BURKITT'S LYMPHOMA
    - HOMOLOGOUS DNA, HUMAN, 810
    - INCIDENCE, ETHIOPIA, 904*
  - GENOME DETECTION
    - BURKITT'S LYMPHOBLASTOID CELLS, 894*
  - LEUKOPROLIFERATIVE STATES
    - HUMAN, REVIEW, 607
  - LYMPHOID CELLS
    - PLASMA MEMBRANE ANTIGEN ISOLATION, 971
  - NASOPHARYNGEAL CARCINOMA
    - HOMOLOGOUS DNA, HUMAN, 810

- S, FELINE FIBROSARCOMA
  - ONCOGENIC ACTIVITY
    - PIG, 903*

- VIRUS, FELINE LEUKEMIA
  - ANTIGEN
    - LEUKEMIC CELLS, CAT, 925

- VIRUS, FELINE SARCOMA
  - TRANSFORMED CELLS
    - AGGLUTINATION, MOUSE, CAT, RAT, 832

- VIRUS, FRIEND LEUKEMIA
  - ANTISERUM
    - MOUSE, 912*
  - DIBENZPYRENE
    - COENZYME Q10, IMMUNITY, MOUSE, 953
  - DNA POLYMERASE INHIBITION
    - POLYCYTIDYLIC ACID, 852
  - IMMUNE RESPONSE
    - ROSETTE FORMING CELLS, MOUSE, 849
  - IMMUNITY
    - MOUSE, 931
  - PSEUDOTYPES
    - BIOLOGICAL PROPERTIES, MOUSE, 865

- VIRUS, GROSS
  - LYMPHOMA
    - IMMUNE RESPONSE, RAT, 955

- VIRUS, HERPES
  - CERVICAL CARCINOMA
    - REVIEW, 614*

- VIRUS, HERPES 2
  - CERVICAL CARCINOMA
    - SEROEPIDEMIOLOGIC STUDIES, 963

- VIRUS, HERPES SAIMIRI
  - LYMPHOMA INDUCTION
    - MONKEY, 788
  - LYMPHOMA, MALIGNANT
    - CELL CULTURE ESTABLISHMENT, MONKEY, 889*
    - LYMPH NODE, MONKEY, 858

- VIRUS, HERPES SIMPLEX
  - DNA
    - PLAQUE FORMING ABILITY, HELA CELLS, 914*
  - DNA SYNTHESIS
    - HELA CELLS, 862
  - INFECTION
    - ANTIGEN SYNTHESIS, HAMSTER CELLS, 867
  - TRANSFORMED CELLS
    - IMMUNOLOGIC MANIPULATION OF METASTASES, HAMSTER, 990*

- VIRUS, INFLUENZA
  - BENZO(A)PYRENE HYDROXYLASE ACTIVITY
    - LUNG TISSUE, MOUSE, 668

- VIRUS, LACTIC DEHYDROGENASE
  - ERYTHROPOIETIN
    - ERYTHROID RESPONSE, MOUSE, 871

- VIRUS, MAMMARY TUMOR
  - ANTIBODY
    - MOUSE, 864

  DNA
    NUCLEOTIDE SEQUENCE, MOUSE, 821
  IMMUNOSUPPRESSION
    MOUSE, 968
  INTRACYTOPLASMIC A PARTICLES
    ISOLATION, MOUSE, 793
  LACTOPEROXIDASE IODINATION
    STRUCTURAL CHARACTERISTICS, 814
  MAMMARY TUMOR CELLS
    HUMAN, 794
  MILK
    BREAST CANCER PATIENTS, 794
  TRANSMISSION BY SPERM
    MOUSE, 907*

VIRUS, MAREK'S DISEASE
  ANTIGEN
    CELL CULTURE, CHICK, 964
  HERPESVIRUS-ASSOCIATED ONCOGENESIS
    CHICKEN, 890*

VIRUS, MASON-PFIZER MONKEY
  INTERNAL COMPONENTS
    MORPHOLOGY, 913*

VIRUS, MOLONEY LEUKEMIA
  ANTIGEN
    LYMPHOCYTE CYTOTOXICITY, MOUSE, 956
  LACTOPEROXIDASE IODINATION
    STRUCTURAL CHARACTERISTICS, 814

VIRUS, MOLONEY SARCOMA
  DNA POLYMERASE INHIBITION
    POLYCYTIDYLIC ACID, 852
  TRANSFORMATION
    2-DEOXYGLUCOSE UPTAKE ALTERATIONS, RAT CELLS, 866

VIRUS, MURINE LEUKEMIA
  ANTIGEN
    GENETIC CONTROL, MOUSE, 922
    VIRAL ENVELOPE, 976

VIRUS, MURINE SARCOMA
  ANTIGEN
    VIRAL ENVELOPE, 976
  ANTIGEN, CELL SURFACE
    IMMUNOELECTRON MICROSCOPY, 941
  DNA POLYMERASE ACTIVITY, 790
  IMMUNE RESPONSE
    MOUSE, 974
  IMMUNOSUPPRESSION
    ONCOGENESIS, MOUSE, 939
  NATURALLY OCCURRING
    MOUSE, 806
  TRANSFORMED CELLS
    AGGLUTINATION, MOUSE, CAT, RAT, 832

VIRUS, MURINE SARCOMA-LEUKEMIA
  REPLICATION
    RAT CELL LINE, 838

VIRUS, ONCORNA
  LYMPH NODE
    MONKEY, 850
  SPONTANEOUS DIFFERENTIATION
    STABLE CELL LINE, MOUSE, 916*

VIRUS, PAPILLOMA
  ANTIBODY
    HUMAN, 959
  SEROLOGICAL STUDIES
    HUMAN, 959

VIRUS, PAPOVA
  PRESENCE IN URINE
    RENAL TRANSPLANTATION PATIENTS, 798

VIRUS, POLYOMA
  ALKALINE PHOSPHATASE
    TRANSFORMED CELL, HAMSTER, 851
  ANTIBODY
    CLINICAL STUDY, ENGLAND, 1021*
  DNA
    ASSAY, MOUSE CELLS, 800
  DNA POLYMERASE
    EMBRYO CELLS, RAT, 876
  INDUCTION BY FLUORESCENT LIGHT
    5-BROMODEOXYURIDINE, RAT, 785
  INFECTION SUSCEPTIBILITY
    CELL CYCLE, MOUSE, 796
  NUCLEOTIDE PHOSPHODIESTERASE
    TRANSFORMED CELL, HAMSTER, 851
  REPLICATION
    ADRENAL GLUCOCORTICOIDS, MOUSE CELLS, 799
    SUSCEPTIBILITY, MOUSE, 811
  RNA-LINKED DNA
    MOUSE CELL, 820
  SYNTHESIS INDUCTION
    HAMSTER CELLS, 807
  TRANSCRIPTION
    STRAND ORIENTATION, 863
  TRANSFORMATION
    CARBOHYDRATE CATABOLISM, HAMSTER CELLS, 883
    ENZYME ACTIVITIES, MOUSE CELLS, 868
    GALACTOSE UPTAKE, HAMSTER CELLS, 883
  TRANSFORMED BHK CELLS
    INVASIVENESS, CHORIOALLANTOIC MEMBRANE, CHICK, 813
  TRANSFORMED MOUSE CELLS
    EXOGENOUS PURINES, 805

VIRUS, POXVIRUS
  PAPILLOMA
    MARSUPIAL, 905*

VIRUS, PROGRESSIVE PNEUMONIA
  SARCOMA
    TRANSPLANTATION ANTIGENS, MOUSE, 970

VIRUS, RADIATION LEUKEMIA
  BACILLUS CALMETTE-GUERIN
    LEUKEMOGENESIS, MOUSE, 836
  LACTOPEROXIDASE IODINATION
    STRUCTURAL CHARACTERISTICS, 814

VIRUS, RAUSCHER LEUKEMIA
  DNA POLYMERASE INHIBITION
    1-BETA-D-ARABINOFURANOSYLCYTOSINETRIPHOSPHATE, MOUSE, 839

- ERYTHROBLASTOSIS
  - DEFECTIVITY, MOUSE, 845
- HISTOPROLIFERATIVE EFFECT
  - LYMPHATIC TISSUE, MOUSE, 871
- IMMUNE RESPONSE
  - MCUSE, 861
- INFECTION
  - ANCHORAGE DEPENDENCE, RAT FIBROBLASTS, 881
  - ERYTHROLEUKEMIA INHIBITION, MOUSE, 878
  - HEMOLYTIC ANEMIA INDUCTION, MOUSE, 878
- NASOPHARYNGEAL CARCINOMA
  - HOMOLOGOUS RNA, HUMAN, 854
- RADIOPROTECTIVE EFFECT
  - MOUSE, 910*
- RNA
  - REVERSE TRANSCRIPTASE, HUMAN LEUKEMIC CELLS, 825

S, REO 3
- BURKITT'S LYMPHOMA
  - INCIDENCE, CORRELATION, UGANDA, 1043

S, RNA TUMOR
- POLYADENYLIC ACID
  - MILK, HUMAN, 843

S, ROUS SARCOMA
- BRAIN TUMOR
  - DOG, 860
- FIBROBLAST TRANSFORMATION
  - ADENYLATE CYCLASE ACTIVITY, CHICK-EMBRYO, 795
- GLIOMA
  - GREY MATTER EDEMA, DOG, 896*
- INDUCTION
  - TRANSFORMED FIBROBLASTS, HAMSTER, 885
- NON-TRANSFORMING MUTANTS
  - ONCOGENICITY, CHICKEN, 801
- RNA
  - 5'TERMINUS CHARACTERIZATION, 833
- 30-40S RNA PRECURSOR, 884
- SYNCYTIA FORMATION
  - C-TYPE VIRUS, HUMAN CELLS, 879
- RANSFORMED CHICK-EMBRYO FIBROBLASTS
  - SUGAR TRANSPORT, 830
- UMOR ANTIGENS
  - IMMUNO-FERRITIN TECHNIQUES, 935
- UMOR CELLS
  - LYMPHOCYTE CYTOTOXICITY, QUAIL, 886
- UMOR FORMATICN
  - GROWTH REGULATION, RAT, 818

, SHOPE FIBROMA
- YTOTOXIC ANTIBODY RESPONSE
  - RABBIT, 784

, SIMIAN ADENO
- NA
  - CHARACTERIZATION, 848

, SV40
- NA
  - ENCAPSIDATION, 853
- DNA POLYMERASE
  - MONKEY CELL CULTURE, 791
- DNASE ACTIVITY INDUCTION
  - MONKEY CELL CULTURE, 791
- INFECTION
  - ANTIGENICITY, MOUSE CELLS, 891*
  - TUMORIGENICITY, MOUSE CELLS, 891*
- KIDNEY TUMOR
  - HAMSTER, 857
- PH DEPENDENT RESCUE
  - HAMSTER, MONKEY, MOUSE CELLS, 855
- PRODUCTION
  - SV40-TRANSFORMED RENAL CELLS, HUMAN, 797
- PROSTATE
  - HAMSTER, 857
- PROTECTION
  - MYELOMA ONCCGENESIS, MOUSE, 875
- PROTEIN-DNA COMPLEXES
  - SUPERCOILING, 789
- RESCUE
  - CELL FUSION, HAMSTER, 824
- REVERTANT CELL LINE
  - MORPHOLOGY, MCUSE, 803
- RNA
  - RNA POLYMERASE, E.COLI, 901*
- RNA SYNTHESIS
  - TRANSFORMED HUMAN CELLS, 787
- TRANSFORMATION
  - BIOCHEMICAL CHARACTERIZATION, MOUSE, 827
  - ENZYME ACTIVITIES, MCUSE CELLS, 868
  - HAMSTER EMBRYO CELLS, 870
  - SUGAR TRANSPORT, MOUSE CELLS, 911*
- TRANSFORMED CELL
  - CELL MIGRATION, MOUSE, HAMSTER, 882
- TRANSFORMED CELLS
  - GROWTH INHIBITION, LIPOPOLYSACCHARIDE, RAT, 888*
- TRANSFORMED KIDNEY CELLS
  - SURFACE CHARACTERISTICS, MOUSE, 856

VIRUS, VESICULAR STOMATITIS
- AVIAN MYELOBLASTOSIS
  - PSEUDOTYPE FORMATION, CHICK CELL, 840
- MAMMARY TUMOR
  - PSEUDOTYPE FORMATION, HUMAN CELLS, 950

VIRUS, VISNA
- RNA
  - POLYADENYLIC ACID, 898*
- SARCOMA
  - TRANSPLANTATION ANTIGENS, MOUSE, 970

VITAMIN A
- RNA METABOLIAM
  - HAMSTER TRACHEAL EPITHELIUM, 1109*

VITAMIN C
- AMINOPYRINE NITROSATION BLOCKING
  - MOUSE, 703

VULVA

CARCINOMA
CLINICAL STUDY, REVIEW, 645*

WILM'S TUMOR
CULTIVATION
CHICK EMBRYO, 1081
INDUCTION AND TREATMENT
EXPERIMENTAL MODEL, 1189*

YOLK SAC
ALPHA-FETOPROTEIN SYNTHESIS
HUMAN CONCEPTUS, 1122*

ZINC
PLEURAL FLUID
NEOPLASTIC DISEASE PATIENTS, 1196*

☆U.S. GOVERNMENT PRINTING OFFICE: 1973-544-909/2

LIBRARY U. OF I. URBANA-CHAMPAIGN

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list.

DHEW Publications No. (NIH) 72-301

MARCH 1973
Abstract & Citation Nos. 1201-1800

Vol. 11
No. 3

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health

Volume XI, No. 3
March

Abstract and Citation Nos. 1201 - 1800

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

Editor

Robert Love, M.D.
Jefferson Medical College, Philadelphia

Associate Editor

George P. Studzinski, M.D.
Jefferson Medical College, Philadelphia

NCI Staff Consultants

Elizabeth Weisburger, Ph.D.

Sidney Siegel, Ph.D.

THE LIBRARY OF THE
NOV 29 1973
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Etiology Area
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication approved by the Director of the Bureau of the Budget on July 25, 1967.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	U	unit(s)
kg	kilogram(s)	UV	ultraviolet
LD_{50}	median lethal dose(s)	WBC	white blood cells (leukocytes), white blood count
m	meter(s)		
M	molar	wk	week(s)
mEq	milliequivalent(s)	wt	weight(s)
mM	millimolar	yr	year(s)
μM	micromolar		
mC,μC	milli-,microcurie(s)		

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	1201-1231	179
CHEMICAL CARCINOGENESIS	(Chem)	1232-1299	185
PHYSICAL CARCINOGENESIS	(Phys)	1300-1320	199
VIRAL CARCINOGENESIS	(Viral)	1321-1435	202
IMMUNOLOGY	(Immun)	1436-1576	225
PATHOGENESIS	(Path)	1577-1587	246
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	1588-1606	248
MISCELLANEOUS	(Misc)	1607-1800	250
AUTHOR INDEX			
SUBJECT INDEX			

1201 IMPLICATIONS OF CANCER IMMUNITY. *(E.)*

Burnet, F. M. (Sch. Microbiol., U. Melbourne, Australia). *Aust NZ J Med* 3(1):71-77, 1973.

Recent work relating to tumor immunology, immune surveillance, and its clinical implications is reviewed. Tumor cells retain most of the immunologic qualities of normal cells, including the histocompatibility antigens, and are thus subject to rejection by the T-cell mediated immune system. Unlike normal cells, however, tumor cells contain their own unique and specific surface antigens. Experimental support for this has come from chemical- and viral-induced tumors in animals in which characteristic tumor-specific transplantation antigens have been identified. In addition, some tumors may produce antigens characteristic of normal embryonic or fetal cells. Besides such evidence from experimental animal tumors an α-fetoprotein has been identified in sera from some persons with hepatoma and carcino-embryonic antigen has been found in sera of patients with colonic carcinoma. Clinical observations, which include an active tumor-specific immune response in patients with spontaneously regressing tumors and a high incidence of malignancies in immunodeficient and immunosuppressed individuals, lend support to the concept of immunological surveillance. (17 references)

1202 CELLULAR PROLIFERATION IN CARCINOGENESIS.

(E.) Oehlert, W. (Pathol. Inst., U. Freiburg, W. Germany). *Cell Tissue Kinet* 6(3):325-335, 1973.

Literature bearing on the relationship between cellular proliferation and carcinogenesis is reviewed. More than 80% of spontaneously arising tumors in man arise in tissues which are rapidly changing their cell populations, such as the tracheobronchial, gastrointestinal and urogenital tracts and the skin. These tissues consist of both differentiated and rapidly proliferating cells, the latter class of cells serving as the precursors for cancer cells. *In vivo* experimental systems have provided results which support this model. Application of carcinogenic hydrocarbons to skin of experimental animals increases proliferative activity of the treated epithelium, which has been explained by an increase in the proliferative cell pool following injury to differentiated cells. This situation then results in tumorigenesis. An analogous mechanism has been implicated in radiation-induced carcinogenesis. Other studies have shown that mice treated with carcinogens while in stages of increased cell proliferation develop more papillomas and carcinomas and at an earlier time than animals treated in a phase of decreased proliferative activity. (48 references)

1203 CANCER PERMEATION: PROCESSES, PROBLEMS, AND PROSPECTS--A REVIEW. *(E.)*

Onuigbo, W. I. B. (Gen. Hosp., Enugu, Nigeria). *Cancer Res* 33(4):633-636, 1973.

Permeation is the scientific term introduced in 1905 by Handley to signify the spread of cancer cells in continuous columns within the lymph vessels. The phenomenon itself was known to workers of much earlier times and, to date, its processes largely have been documented. However, some problems have arisen. There is, for example, considerable confusion because different authorities have classified it under both direct and metastatic spread. Evidence is therefore adduced from the literature to show that the columns of cancer cells seen in permeation are not absolutely continuous but partially continuous. It is this element of discontinuity that confers on permeation the status of a metastatic phenomenon. If this status is recognized, a continuing source of confusion will disappear from the literature and new prospects will open in this important facet of oncology. (75 references)

1204 THE EXPRESSION OF NORMAL HISTOCOMPATIBILITY ANTIGENS IN TUMOR CELLS. *(E.)*

Lengerova, A. (Inst. Exp. Biol. Genetics, Czechoslovak Acad. Sci., Prague). *Adv Cancer Res* 16:235-271, 1972.

Recent systematic studies of the expression of normal cellular antigen in tumor cells are reviewed under two major headings: 1) quantitative and qualitative characteristics; and 2) regulation of the expression of cellular antigens as a possible site for impact of malignant transformation. Studies on the quantitative variation in histocompatibility phenotype of tumor cells, including the possible bearing of quantitative differences in H antigenicity on the resistance of tumor allografts; loss variants of H-2 heterozygous tumors, and the expression in tumor cells of normal stage-specific or tissue-specific antigens are discussed in detail. The cell cycle and variation in the expression of membrane antigens, the level of regulation revealed by analysis of somatic cell hybrids and the possible role of the specificity of membrane architecture in normal and pathological morphogenesis are reviewed in relation to their effect on the regulation of the expression of cellular antigens. (156 references)

1205 IMMUNOSUPPRESSION AND NEOPLASIA. I. A CRITICAL REVIEW OF EXPERIMENTAL CARCINOGENESIS AND THE IMMUNESURVEILLANCE THEORY. *(E.)*

Gleichmann, H. (Med. Sch. Hannover, Germany) and E. Gleichmann. *Klin Wochenschr* 51(6):255-259, 1973.

The immunesurveillance theory, which provides a simple explanation for the high frequency of cancer in conditions of impaired immunologic responsiveness, assumes that the correlation between immunosuppression and augmented tumor rates is due to a lack of cellular immune response against the tumor specific transplantation antigens (TSTA) of neoplastic cells and that a depression of cellular immunity causes an increased formation of spontaneous neoplasms. This theory is critically reviewed and a number of objections raised, as follows: 1) The promoting effect of immunosuppressive treatment on lymphomagenesis is facilitated by lymphatic hyperproliferation; immunosuppression alone is probably insufficient for inducing malignant lymphomas. 2) In viral carcinogenesis, the increase in tumor incidence under immunosuppression cannot clearly be attributed to a deficient immunologic response against the virus-induced TSTA. 3) Suppression of the cellular immune response during chemical carcinogenesis causes a moderate increase in tumor incidence. 4) The rate of spontaneous malignancies in animals with suppressed cellular immunity does not differ significantly from that of non-immunosuppressed animals. (161 references)

1206 MOLECULAR REPAIR, WOUND HEALING, AND CARCINOGENESIS: TUMOR PRODUCTION - A POSSIBLE OVERHEALING? *(E.)* Haddow, A. (Royal Cancer Hosp., London, England). *Adv Cancer Res* 16:181-234, 1972.

The characteristics and mechanisms of repair and wound healing at the molecular and cellular levels are related to events in carcinogenesis. Some of the broad areas considered include photoreactivation, cytology, mechanisms of radiation protection, and DNA repair. As long ago as 1902, studies of the implantation of tumors into wound margins to examine the repair process were advocated. If malignant change is regarded as a form of overhealing, the physiology of wound healing possesses a highly stimulating influence for problems of treatment. Such topics as the relation of allantoin, ascorbic acid, beryllium, zinc compounds, sulfhydryl compounds, protein therapy, chondroitin sulfuric acid, acid mucopolysaccharides, glycoproteins, and cell agglutinins to wound healing are discussed; the author recommends the testing and retesting of many of these substances, both micro- and macromolecular. The possible role of plant physiology in suggesting macromolecules for biological test is reviewed. Such substances would include agars, plant lignins, and biological sealants. (433 references)

1207 POLYSACCHARIDES IN CANCER. *(E.)* Nigam, V. N. (Notre Dame Hosp., Montreal, Canada) and A. Cantero. *Adv Cancer Res* 16:1-96, 1972.

The role of polysaccharides in the carcinogenic process as well as in the growth characteristics of tissues is reviewed. The function of hyaluronic acid and chondroitin sulfates as intercellular cement is well accepted and places them in a relevant position in cancer research, since loss in cell-cell association is often considered as a possible reason for the metastatic spread of tumors. Mucopolysaccharides are excreted by both normal and transformed cells, and thus they are an important constituent of the environment in which both normal and neoplastic cells survive. From various studies carried out in normal and tumor tissues, it has become evident that no chemical differences exist between mucopolysaccharide isolated from the two kinds of tissues. How far quantitative differences in mucopolysaccharide content between normal and cancer tissue are related to cancerous transformation is difficult to appreciate because emergence of a uniform pattern has not yet been shown. Numerous histochemical studies in human tumors have demonstrated the presence of mucopolysaccharides in and around cancers of many different tissues. Only mesotheliomas, however, are definitely known to produce large amounts of hyaluronic acid and chondroitin sulfate. Since tumor viruses alter mucopolysaccharide synthesis, it is suggested that it may be desirable to study mucopolysaccharide metabolism in human cell lines before and after transformation by oncogenic RNA and DNA viruses, and to compare transformed cell lines with cultured human tumors where abnormal production of mucopolysaccharides is evident. (461 references)

1208 TRANSFORMATION BY POLYOMA VIRUS AND SIMIAN VIRUS 40. *(E.)* Sambrook, J. (Cold Spring Harbor Lab., N.Y.). *Adv Cancer Res* 16:141-180, 1972.

Experiments that bear on the mechanism by which virus transformation occurs and the ways currently being used to isolate and characterize those virus proteins responsible for the phenomenon are reviewed. Polyoma virus and Simian virus (SV) 40 are viruses of simple structure and contain enough DNA to code for only a few proteins. About 12% of these two viruses is DNA; the rest is protein. This framework supports a variety of complicated interactions with cells, one of which is transformation, where virus infection causes the cells to assume malignant properties. At least one virus-coded function is necessary to establish transformation, and integrated viral DNA persists in the transformed cells. Although this integrated DNA may be necessary to provide for the genetic stability of the transformed state, it is not of itself sufficient to cause expression of the transformed phenotype, for which additional virus-coded functions are required. Most current research on polyoma virus and SV40 is directed toward cataloguing and identifying these functions. (307 references)

1209 LATE NEOPLASTIC CHANGES FOLLOWING MEDICAL IRRADIATION. *(E.)* Hutchison, G. B. (Harvard Sch. Public Hlth., Boston, Mass.). *Ther Radiol* 105(3):645-652, 1972.

Literature pertaining to possible associations between exposure to various types of radiation and subsequent development of cancer in humans is reviewed. A statistically significant correlation has been found between the following cancers and therapeutic or diagnostic procedures including local x-irradiation, fluoroscopy, and infusions: adult leukemias, childhood malignancies in general (following *in utero* exposure), cancers involving local organs (e.g., thyroid, G.I. tract, bronchus, bone, head) breast cancer, hepatoma, head sinus cancers and even generalized cancer. Patients included in this category had been treated for such diseases as ankylosing spondylitis, metropathia hemorrhagica, tinea capitis, and benign conditions of the neck.

A possible, though not significant, correlation has been found for leukemia following therapy for polycythemia vera or irradiation to the thymus, for malignancies in limited irradiated fields and therapy for metropathia hemorrhagica or thymic hypertrophy, and for neurofibrosarcoma following Thorotrast infusion. This and other data presented suggest that: 1) Irradiation may affect all types of malignancies; 2) All types of irradiation may be carcinogenic; 3) Risk may be calculated at one to five cases per 10^6 population per yr per rad; and 4) The latent period for clinical appearance of cancer varies with the type, being shortest for leukemia. (47 references)

1210 IMMUNOLOGICAL ENHANCEMENT, BLOCKING FACTORS, AND TOLERANCE TO TUMORS, TRANSPLANTS AND FOETUSES. *(E.)* Halliday, W. J. (Dept. Microbiol., U. Queensland, Brisbane, Australia). *Aust NZ J Med* 4(2):416-421, 1972.

Evidence supporting the theory that immunological nonreactivity ("tolerance") is involved in tumor growth and progression is reviewed. Tolerance as manifested towards progressing tumors, successful transplants and the developing fetus, may be accomplished through blocking factors containing the appropriate antigen in combination with specific antibody. These factors interfere with the lymphocyte-mediated cytotoxic immunity developed simultaneously against the growing "foreign" cells. It is postulated that a balanced situation is achieved by the establishment of conditions for moderate immunological responses towards antigens which must be continually synthesized and released. The successful initiation of primary tumors, pregnancy and most allogeneic transplants requires immunologically depressed or immature hosts; normally competent hosts will tolerate weakly antigenic cells (syngeneic tumors and some allogeneic transplants) only if the number of cells is large. In this way, the formation of antigen-containing blocking factors keeps pace with the development of cytotoxic immunity, resulting in immunological enhancement of the invading cells. (63 references)

1211 THE ROLE OF VIRUSES IN THE ORIGIN OF CANCER. *(E.)* Bentvelzen, P. (Radiobiol. Inst., Rijswijk, Netherlands). *TNO* 27(11):644-652, 1972.

Theories of carcinogenesis by oncornaviruses are reviewed. Research at the Radiobiological Institute with Rauscher leukemia virus (RLV) is emphasized. Indications are that the target of RLV is the hemopoietic stem cell. It is suggested that erythropoietic stimulation of RLV-infected stem cells gives rise to abnormal erythroblasts. Production of abnormal cells results in continuous erythropoietic stimulation, producing more abnormal cells. Viral aspects of cancer and aging are also discussed. The late appearance of virus-induced tumors may result from host-dependent resistance to virus, low virulence of virus or late emergence of an endogenous virus. In current theories, cells harboring the oncornavirus genome can release virus spontaneously (with advancing age of the host, e.g.), or in response to exogenous stimuli such as radiation or chemical carcinogens. (15 references)

1212 QUESTION: DO HERPESVIRUSES CAUSE CANCER? ANSWER: OF COURSE THEY DO! *(E.)* Rapp, F. (Hershey Med. Ctr., Pennsylvania State U., Hershey). *J Natl Cancer Inst* 50(4):825-832, 1973.

Evidence linking herpesviruses and human cancer is briefly reviewed. Herpesviruses, thought to cause cancer in frogs, chickens, monkeys and rabbits, meet many criteria for tumor viruses in humans. The Epstein-Barr virus (EBV) is discussed. The EBV genetic information is regularly found in human tumors and the virus can convert normal cells into cells with infinite replicative capabilities. Herpes simplex virus is also discussed. This virus is also a candidate for a human cancer virus on the basis of its epidemiological correlation with cervical cancer and its ability to transform human cells *in vitro*. (81 references)

1213 ASCORBIC ACID AND THE GLYCOSAMINOGLYCANS: AN ORTHOMOLECULAR APPROACH TO CANCER AND OTHER DISEASES. *(E.)* Cameron, E. (Vale Leven Hosp., Alexandria, Scotland) and L. Pauling. *Oncology* 27(2):181-192, 1973.

A theory is described according to which ascorbic acid may figure in the control of cell proliferation. Cells in tissues are embedded in a ground substance the composition of which influences all cellular behavior, including malignant proliferation. The ground substance is highly viscous due to the presence of glycosaminoglycans including hyaluronic acid. It is thought that the malignant proliferation of cells is held in check by the viscous nature of their ground substance *milieu*. Viscosity of the ground substance can be reduced, however, and cell proliferation unleashed, by the action of the enzyme hyaluronidase. Ordinarily, hyaluronidase in cells is inhibited by an agent which appears to require ascorbic acid for its synthesis. Evidence for the ascorbic acid theory, and its implications for cancer control, are discussed. (35 references)

1214 PHORBOL ESTERS, FATTY ACIDS AND TUMOUR PROMOTION. *(E.)* Rohrschneider, L. R. (U. Wisconsin Med. Ctr., Madison) and R, K. Boutwell. *Nature* [*New Biol*] 243(128):212-213, 1973.

The literature is reviewed in an attempt to explain the mechanism of action of the potent tumor promoter 12-*O*-tetradecanoylphorbol-13-acetate (TPA) the most active component of croton oil. The nature of the TAP molecule as well as recent research both suggest that TPA interacts with the cell membrane, and this interaction is the proposed initial event in its mechanism of action. The observed specificity of the reaction of TPA with target cells suggests that a TPA-specific membrane receptor is involved. The fact that TPA is of plant origin further suggests that it is most likely mimicking a structurally similar natural endogenous product and interacting at its membrane receptor. The striking structural similarity between TPA and the methyl esters of a polyunsaturated fatty acid (PUFA) are consistent with this latter substance as the natural endogenous product. The identification of a naturally occurring unsaturated fatty acid, oleic acid, and its methyl ester as moderately active tumor promoters lends further support to this hypothesis. (18 references)

1215 HUMAN MAMMARY TUMOR VIRUS. *(E.)* Hollmann, K. H. (Broussais Hosp., Paris, France). *Biomedicine* 18(2):103-108, 1973.

Evidence in support of the existence of a human mammary tumor virus is reviewed. The viral etiology of human breast cancer received initial support in 1971 with the discovery in human milk of B-like (MS) particles resembling murine mammary tumor virus (MTV). Of the three types of MS particles, MS-2 has been found in 30% of all human milks examined, whether or not the milk is from women with a family history of breast cancer. MS-1 and MS-2 particles are present in milk from women in India, the United States and France. In addition, virus-like particles almost identical to MTV have been observed in human breast tissues in four of nine cases of cancer and in two cases of cystic disease. Several lines of evidence suggest that the candidate human breast cancer virus is related to MTV. MTV preincubated with sera from humans with breast cancer is less tumorigenic than control virus following injection into mice. Some human milks contain RNA-dependent DNA polymerase as well as 60-70S RNA. Molecular hybridization studies indicate areas of homology between viral DNA from MTV and cytoplasmic RNA from human breast cancer cells. Finally, the high incidence of breast cancer in some families is consistent with vertical transmission of a human cancer virus. (30 references)

1216 POSSIBLE ETIOLOGIES OF CANCER OF THE CERVIX OTHER THAN HERPESVIRUS. *(E.)* Alexander, E. R. (Sch. Publ. Hlth., Community Med., U. Washington, Seattle). *Cancer Res* 33(6):1485-1496, 1973.

The evidence for noninfectious agents and for infectious agents, other than herpesvirus, as a cause of cervical cancer is reviewed. Although some carcinogenic potential for smegma may be inferred from experimental studies, epidemiological studies do not indicate a significant role for either smegma or circumcision in human cervical cancer. One hypothesis attempts to explain the importance of early coitus in cervical cancer by proposing that sperm is the nucleic acid vector and the potential mutagen for tissues undergoing active metaplasia. The most attractive aspect of this hypothesis is the suggestion that any sperm may be a potential mutagen, not because of some special intrinsic property but because of a particular susceptibility of the target tissue at certain times. Six infectious agents have an established association with cervical cancer. The association with *Trichomonas*, syphilis, and gonococcal infections in all probability merely reflects the fact that these infections are strongly associated with promiscuity. The correlation of mycoplasmal infection with mild cervical dysplasia, as well as with promiscuity, calls for more serious consideration of this infection, but there is no consistent association between mycoplasmal infection and cervical cancer. Chlamydial infections may be important because of their chronicity, their association with chronic inflammatory disease, and their venereal mode of transmission. Cytomegalovirus infections may be implicated upon similar grounds. The virus is known to be associated with chronic inflammatory disease, but evidence for a venereal mode of transmission and for male genital infection is less convincing. Whatever the evidence for the association of these agents with cervical cancer, none appears to be so likely a candidate for its cause as herpesvirus type 2. Any hypothesis on the etiology of cervical cancer must take into consideration the epidemiological characteristics of the disease: 1) at highest risk of cervical cancer are women who have had first coitus at an early age, have had multiple sex partners, are sexually promiscuous, and are of lower socioeconomic status; and 2) at lowest risk of cervical cancer are the celibate, and to a lesser degree the nulliparous and Jewish Women. (55 references)

1217 A COMPARISON REVIEW OF KEY EPIDEMIOLOGICAL STUDIES IN CERVICAL CANCER RELATED TO CURRENT SEARCHES FOR TRANSMISSIBLE AGENTS. *(E.)* Rotkin, I. D. (U. Illinois, Med. Ctr., Chicago). *Cancer Res* 33(6):1353-1367, 1973.

The search for a venereally transmissible oncogenic agent which may show relatedness to onset of cervical cancer rests upon three main evidential currents: demographic demonstration of cultural variables associated with increased risk, epidemiological focus upon sexual factors maximizing risk, and the assumption that cervical cancer begins and develops according to a multistage continuum. For this, an abbreviated model conforming multistage carcinogenesis in the human uterine cervix with the classical animal model introduces an epidemiological review. Assumptions required for this rationale include maximal biological activity in the cervical cellular matrix during adolescence, an oncogenic agent for initiation of neoplasia, and recognition of pivotal events directly related to risk. A set of comparison studies is presented in assessing historically described major influences upon risk and each is examined for relevance, using a comparison ratio for detection of excessive frequencies between patients and controls. It is shown that onset of sexuality before age 17 is the most powerfully discriminating variable in virtually all studies where this has been investigated and that a history of multiple sexual consorts is a supporting variable of some strength. Marital variables and those associated with sociosexual stability are cultural and relate to the biological variables of early coitus and multiple sexual partners. Coital frequencies, menstrual patterns, and gravidity seem to bear no discernible risk relationship. Noncircumcision of sexual partners is shown to have been limited in most studies to husbands and in some studies to selected or all coital mates, yet no study reveals differences between patients and controls for exposure to uncircumcised partners. Cervical cancer is the only solid neoplasm for which epidemiological findings in humans have been adapted to a model developed with laboratory animals, and it is the only tumor that has been studied with regard to a full universe of possible variables and attributes that might alter risk. This has resulted in the currently espoused hypothesis that *Herpesvirus hominis* type 2 may be an initiating or promoting carcinogenic agent which can be transmitted during coitus by the male donor to the female host at risk. (39 references)

1218 METABOLISM AS A FACTOR IN DETERMINING THE TOXIC ACTION OF THE AFLATOXINS IN DIFFERENT ANIMALS SPECIES. *(E.)* Patterson, D. S. P. (Central Vet. Lab., Weybridge, Surrey, England). *Food Cosmet Toxicol* 11:287-294, 1973.

The present state of knowledge of the metabolic fate of aflatoxin B1 in the livers of avian and mammalian species is reviewed and an attempt is made to relate metabolism to toxic action. Based on the time required for metabolism of an LD_{50} dose of aflatoxin, eight avian and mammalian species were classified as "fast" (rabbit, duck, guinea-pig), "intermediate" (chick, mouse, pig, sheep) and "slow" metabolizers (rat). Aflatoxin B_1 can be metabolized by one or more species to form the 2-hydroxy or hemiacetal derivative (aflatoxin B_2a), the 4-hydroxy derivative (aflatoxin M_1), the phenolic derivative (aflatoxin P) or the cyclopentol derivative (aflatoxicol). Fast metabolizers are all capable of hemiacetal formation, suggesting that hemiacetal formation by liver microsomal enzymes is an important factor in producing acute toxic effects in these animals. In slow metabolizers, however, such as the rat, untransformed toxin is almost certainly the active species, with chronic liver damage the probable outcome. (42 references)

1219 EVIDENCE FOR AN ONCOGENIC POTENTIAL OF THE EPSTEIN-BARR VIRUS. *(E.)* Henle, W. (Children's Hosp. Philadelphia, Pa.) and G. Henle. *Cancer Res* 33(6):1419-1423, 1973.

Literature pertaining to the different approaches being made to establish an etiologic role of Epstein-Barr virus (EBV) in Burkitt's lymphoma (BL) and/or nasopharyngeal carcinoma (NPC) is reviewed. The search for EBV particles in tumor biopsies by electron microscopy or for antigen-producing tumor cells by immunofluorescence has been unrewarding. Although only one in 100,000 biopsy cells might be early antigen (EA)-positive, as much as 10% of the same population expresses EA when established in cell culture. Most cells from BL biopsies express membrane antigens which, when not detectable immunologically, are coated with the patient's antibodies. Lymphoblast lines established from biopsy specimens harbor EBV, and EBV DNA has been demonstrated by hybridization techniques in every BL and NPC biopsy examined. Attempts to induce tumors by EBV inoculation into nonhuman primates also have not met with success, presumably due to a requirement for certain unknown cofactor(s). Results from *in vitro* transformation studies have established that EBV does stimulate lymphoproliferation and that it is most likely the sole requirement for establishing lymphoblast lines from normal cells. Serological testing has revealed the presence of elevated titers of antibody against EBV antigens in approximately 600 African, Oriental and Western BL and NPC patients. High antibody titers have been correlated with relapse and severe disease, whereas low titers are present during periods of remission. (43 references)

1220 THE BACKGROUND AND PROMISE OF TUMOR VIROLOGY, IMMUNOLOGY, AND CHEMOTHERAPY AND A CHALLENGE TO RADIOTHERAPY. *(E.)* Gellhorn, A. (Sch. Med., U. Pennsylvania, Philadelphia). *Am J Roentgenol Radium Ther Nucl Med* 117(3):489-501, 1973. (59 references)

1221 25 YEARS OF CANCER RESEARCH IN BIRMINGHAM - IN CLOSE COLLABORATION WITH OTHERS ACROSS THE NATION AND WORLD. *(E.)* Skipper, H. E. (Sloan-Kettering Inst., New York, N.Y.). *South Res Inst Ann Rep* 9-21, 1972. (No references)

1222 A DEVELOPMENTAL GENETIC APPROACH TO THE STUDY OF TERATOCARCINOGENESIS. *(E.)* Stevens, L. C. (Jackson Lab., Bar Harbor, Me.). *Bio Sci* 23(3):169-172, 1973. (30 references)

1223 CUTANEOUS BASOSQUAMOUS CARCINOMA. REVIEW OF THE LITERATURE AND REPORT OF 35 CASES. *(E.)* Borel, D. M. (U. Kansas Med. Ctr., Kansas City). *Arch Pathol* 95(5):293-297, 1973. (15 references)

1224 RESTRICTION ENZYMES: NEW TOOLS FOR STUDYING DNA. *(E.)* Marx, J. L. (No affiliation). *Science* 180(4085):482-485, 1973. (4 references)

1225 NON-HODGKIN'S LYMPHOMAS. IV. CLINICOPATHOLOGIC CORRELATION IN 405 CASES. *(E.)* Jones, S. E. (Stanford U. Sch. Med., Calif.), Z. Fuks, M. Bull, M. E. Kadin, R. F. Dorfman, H. S. Kaplan, S. A. Rosenberg and H. Kim. *Cancer* 31(4):806-825, 1973. (25 references)

1226 PRIMARY MALIGNANT MESOTHELIAL TUMORS IN CANADA, 1960-1968. A PATHOLOGIC REVIEW BY THE MESOTHELIOMA PANEL OF THE CANADIAN TUMOR REFERENCE CENTRE. *(E.)* McDonald, A. D. (Dept. Epidemiology, Hlth., McGill U., Montreal, Canada), D. Magner and G. Eyssen. *Cancer* 31(4):869-876, 1973. (5 references)

1227 CANCER VIRUS: LINK TO DISEASE IN MAN REPORTED AGAIN. *(E.)* Culliton, B. J. (No affiliation). *Science* 180(4086):572-574, 1973. (No references)

1228 PORCINE AND OVINE LYMPHOSARCOMA: A REVIEW. *(E.)* Bostock, D. E. (Sch. Vet. Med., U. Cambridge, England) and L. N. Owen. *J Natl Cancer Inst* 50(4):933-939, 1973. (59 references)

1229 ASBESTOS → LUNG CANCER → MESOTHELIOMA. *(E.)* Anonymous. *Lancet* (7807):815-816, 1973. (15 references)

1230 THE GENE IN RECOMBINATION AND MUTATION. *(E.)* Jain, H. K. (Indian Agricultural Res. Inst., New Delhi). *J Sci Ind Res* 31(9):463-464, 1972. (No references)

1231 ON MEDULLARY CARCINOMA OF THE THYROID. *(E.)* Ljungberg, O. (Gen. Hosp., Malmö, Sweden). *Acta Pathol Microbiol Scand* [A] (Suppl 231):1-57, 1972. (116 references)

1232 TRANSPLACENTAL AND COMBINED LONG-TERM EFFECT OF DDT IN FIVE GENERATIONS OF A-STRAIN MICE.

(E.) Shabad, L. M. (Acad. Med. Sci. USSR, Moscow), T. S. Kolesnichenko and T. V. Nikonova. *Int J Cancer* 11(3):688-693, 1973.

Results of previous experiments on the effects of dichloro-diphenyltrichloroethane (DDT) in organ culture *in vitro* are compared with its effects on mice *in vivo*. A-strain mice were given technical DDT in sunflower-seed oil at daily dose levels of 50 ppm during their entire life span for the first (F_o) generation and of 10 ppm for the first and 5 consecutive generations (F_o-F_5). Exposure to both levels of DDT resulted in a significant increase of lung adenomas in F_o generations: 36.66% by 50 ppm and 19.05% by 10 ppm of DDT. In F_1-F_5 generations exposed to 10 ppm of DDT lung tumors appeared in F_1 (15.38%), in F_2 (24.00%), in F_3 (46.34%), in F_4 (43.24%) and in F_5 (12.69%). In control A mice, lung tumors were not detected at 6 months of age, but were observed at 12 months in 10.88% and altogether (between 6 and 12 months) in 7.28%. In F_2, F_3 and F_4 treated generations the incidence of lung adenomas was significantly higher as compared to the controls. No other tumors were observed in the treated animals. The data obtained *in vivo* are compared with previous observations made in organ cultures of embryonic lung tissues of the same A-strain mice exposed to 10 ppm of DDT transplacentally. Hyperplastic lesions and nodular proliferation of the epithelium in the organ cultures are correlated with the results *in vivo*. The same dynamics of the total incidence and development of pretumorous and tumorous lesions was observed both *in vitro* and *in vivo*. DDT must be evaluated as a weak carcinogen.

1233 INFLUENCE OF AGE AND 3-METHYLCHOLANTHRENE ON AZO-DYE CARCINOGENESIS AND METABOLISM OF p-DIMETHYLAMINOAZOBENZENE IN RAT LIVER.

(E.) Decloitre, F. (Inst. Sci. Res. Cancer, Villejuif, France), J. Chauveau and M. Martin. *Int J Cancer* 11(3):676-680, 1973.

Two groups of male Sprague-Dawley rats of different ages received a carcinogenic diet containing 0.06% dimethylaminoazobenzene (DAB). Out of a group of 4- to 6-wk-old rats, 20% developed hepatomas 3½ months after the beginning of the diet; after 10 months the percentage of tumors reached 56%. Rats aged 10- to 11-wk-old did not present hepatomas before 6 months of DAB feeding: the yield was only 28% after 10 months of carcinogen ingestion. In control rats, which received an i.p. injection of arachid oil, *in vitro* DAB metabolites binding to DNA and proteins proceeding from microsomal activation of carcinogen decreased by 70% between 4 and 12 wk; within the same time azoreductase activity remained constant. A single injection of 3-methylcholanthrene (20 mg/kg) greatly increased binding but not azoreductase activity. Time-effect was similar in treated and control rats. The results clearly show differences in the incidence of hepatomas in the rats studied according to the age of the animals at the start of the carcinogenic diet. Hormonal factors, modified by aging or differences in the amount of carcinogen fed to the rats, may be involved. Enzymatic activities in azo-dye metabolism may also account for age-related responses. As far as yield of metabolites bound to macromolecules represents transformation of initial carcinogen to active derivatives, two physiological states are considered: 1) the high ability for DAB activation and normal detoxication would make young rats especially susceptible to the carcinogenic effect of DAB; 2) the low degree of activation and normal detoxication in older rats would make then more resistant.

1234 THE INDUCTION OF AZAGUANINE-RESISTANT MUTANTS IN CULTURED CHINESE HAMSTER CELLS BY REACTIVE DERIVATIVES OF CARCINOGENIC HYDROCARBONS.

(E.) Duncan, M. E. (Chem. Carcinogenesis Div., Chester Beatty Res. Inst., London, England) and P. Brookes. *Mutat Res* 21(2):107-118, 1973.

7-Bromomethylbenz[a]anthracene (7-BrMeBA), a weak carcinogen, and 7-bromomethyl-12-methylbenz[a]anthracene (7-BrMe12MeBA), an active carcinogen, were tested for their abilities to induce azaguanine-resistant mutants in azaguanine-sensitive V79 Chinese hamster cell cultures. Sensitive cells grown for 15 min in medium containing one of the carcinogens were recultured and azaguanine was added at different times. The induced mutation frequency increased arithmetically with the number of cell divisions which occurred following exposure to carcinogen and prior to addition of azaguanine, and reached a maximum after three or four divisions. The percentage of induced mutations declined sharply when cells were allowed to progress beyond four divisions. At a given concentration, ^{3}H-labeled 7-BrMeBa, the weaker carcinogen, bound five times more extensively to cellular DNA and RNA than did 7-BrMe12BA. Binding of both compounds to protein was 50-100 times less than to DNA or RNA. However, based on cell survival, 7-BrMeBA was twice as toxic as 7-BrMe12BA. At low doses both compounds gave a similar linear mutation response with a slope of about 5×10^{-5} induced mutants/survivor/μmole hydrocarbon bound/mole of DNA phosphorus. However, at extents of DNA binding greater than 8μmoles/mole phosphorus, 7-BrMeBA was much more mutagenic than 7-BrMe12BA. These data were consistent with the existence of two distinct mechanisms for the induction of mutants by these two hydrocarbon derivatives.

1235 ELECTROPHILIC *N*-ACETOXYAMINOARENES DERIVED FROM CARCINOGENIC *N*-HYDROXY-*N*-ACETYLAMINOARENES BY ENZYMATIC DEACETYLATION AND TRANSACETYLATION IN LIVER.

(E.) Bartsch, H. (U. Wisc. Med. Ctr., Madison), M. Dworkin, J. A. Miller and E. C. Miller. *Biochim Biophys Acta* 286(2):272-298, 1972.

Rat liver cytosol fraction has been shown to convert N-hydroxy-2-acetylaminofluorene (*N*-hydroxy-2-AAF) and similar *N*-hydroxy-N-acetylaminoarenes by enzymatic transacetylation to the strongly electrophilic *O*-acetyl derivatives of N-hydroxyaminoarenes which were identified by mass spectroscopy. In one reaction mixture, the apparent transacetylation mechanism involved transacetylation from the hydroaxamic acid to the hydroxylamine to yield an *N*-acetoxyaminoarene. This latter compound then reacted with *N*-acetylmethionine to yield a sulfonium derivative which decomposed to the methylmercaptoaminoarene. The transacetylase activity of rat liver was localized in the 105,000 x g supernatant (cytosol) fraction and was eluted from Sephadex G-75 columns at pH 7.4. Maximum activity occurred in the presence of pyridine nucleotide at pH 6.8 and was inhibited by thiol reagents. Of all compounds tested, only the aromatic hydroxamic acids served as effective sources of acetyl groups. Hepatic transacetylase activity was found in hamster and rabbit livers but not in mouse, guinea pig, dog or human (post-mortem) livers. Enzyme activity did not, however, correlate with susceptibility of a particular species to *N*-hydroxy-2-AAF hepatocarcinogenesis. Transacetylase activity of rat liver decreased to 20-30% of control following *N*-hydroxy-2-AAF feeding but was unaffected by oral administration of phenobarbital.

1236 EFFECTS OF AFLATOXIN B_1 ON THE FORMATION OF SUBRIBOSOMAL PARTICLES IN RAT LIVER.

(E.) Moule, Y. (Inst. Sci. Res. Cancer, Villejuif, France). *Cancer Res* 33(3):514-520, 1973.

The effect of aflatoxin B_1 on the posttranscriptional steps that convert a normal ribosomal precursor 45 S RNA into subribosomal particles has been studied in rat liver cells. Male Wistar rats, were injected i.p. with orotic acid-^{14}C in neutralized solution (170 µCi/kg), followed by aflatoxin B_1 dissolved in 0.3 ml of propylene glycol injected i.p. (1 mg/kg) after an interval of time long enough to permit the synthesis of a limited number of 45 S RNA molecules without allowing them to begin processing. Aflatoxin B_1 allows 18 S RNA to be excised and transferred into the cytoplasm where it appears in free, newly synthesized 40 S subunits; however, the subsequent emergence of 60 S particles is greatly impaired by the toxin. It may be that aflatoxin B_1 or an aflatoxin-derived metabolite induces some alteration in the properties of the 60S particles; thus, a specific defect in their stability could induce their immediate degradation inside the nucleus. Another possibility might be that aflatoxin B_1 impairs the normal molecular processing of 45 S RNA.

1237 INJECTION SITE TUMOURS AND PRECEDING PATHOLOGICAL CHANGES IN RATS TREATED SUBCUTANEOUSLY WITH SURFACTANTS AND CARCINOGENS.

(E.) Hooson, J. (British Industrial Biol. Res. Assoc., Carshalton, Surrey, England), P. Grasso and S. D. Gangolli. *Br J Cancer* 27(3):230-244, 1973.

The sequential histological changes and neoplastic response occurring in the s.c. tissue of rats after injection of surfactants or carcinogens were compared. Twice weekly s.c. injections of the surfactants Blue VRS and Light Green SF elicited a deranged connective tissue repair with continued proliferation of fibroblasts and extensive collagen desposition. In contrast, the carcinogens N-methyl-N-nitrosourea (MNU) and N-nitroquinoline-N-oxide (NQO) appeared to inhibit connective tissue repair and produce morphologically abnormal fibroblasts. The spectrum of neoplastic response was also found to differ. Surfactants gave rise to local sarcomata only after about 47 wk, whereas carcinogens produced local sarcomata and adenocarcinomata after 20 and 12 wk, resp. Another distinguishing feature is the dose of chemical required to produce a carcinogenic effect. At the termination of the surfactant experiments 1-5 g of dye had been administered. With the carcinogens, only about 40 mg of MNU and 20 mg NQO were given. A completely new tumorigenic response was elicited by MNU and NQO, namely a high percentage of mammary tumors, the majority in female rats. It appears that there are certain clear cut differences in the quality of the neoplastic response which make it possible to distinguish between sarcoma production denoting a true carcinogenic response and sarcomata arising from a derangement of connective tissue repair.

1238 THE EFFECTS OF LONG-TERM FEEDING OF DDT TO BALB/c MICE.

(E.) Terracini, B. (Natl. Inst. Study, Cure Tumors, Milan, Italy), M. C. Testa, J. R. Cabral and N. Day. *Int J Cancer* 11(3):747-764, 1973.

A multigenerational study on the long-term effects of three dose levels of dichloro-diphenyltrichloroethane (DDT) (2, 20 and 250 ppm in the diet) and one dose level of N-nitrosodimethylamine (DMN) (0.0003% in the drinking water) was started in 1968. Each treatment was administered to two separate colonies of BALB/c mice. Treatment of parent mice started at 4-5 wk of age. The results observed in the first two generations are presented. Survival in males was poor because of fighting. In females, the highest dose level of DDT produced liver-cell tumors in 44% of the parent mice and 74% of the first generation mice, whereas no liver-cell tumors were found in mice given the control diet, or lower dietary concentrations of DDT. Liver cell tumors did not give metastases, but grew after transplantation to syngeneic mice. The ability of DDT to induce liver-cell tumors was identical in both colonies of mice given 250 ppm DDT. Malignant lymphomas occurred in about 50% of the mice in all colonies given 0, 2 or 20 ppm DDT. In one of the two colonies given 250 ppm DDT, the incidence of malignant lymphomas was 14% whereas in the other it was 36%. The incidence of lung adenomas was not affected by DDT treatment. Tumors at sites other than liver, lymphatic system and lungs were highest in control mice and lowest in mice given 250 ppm DDT. However, no linear dose-response was found, and the decrease was more obvious in the parental than in the first generation. A study on the combinations of tumors in mice with more than one tumor and an analysis of litter distribution of liver-cell tumor-bearing mice did not reveal any particular trend in the distribution of liver-cell tumors. DMN consistently produced lung adenomas, liver blood cysts and hemangioendotheliomas.

1239 COMPARATIVE STUDIES OF THE HEPATOCARCINOGEN N,N-DIMETHYLNITROSAMINE *IN VIVO:* REACTION SITES IN RAT LIVER DNA AND THE SIGNIFICANCE OF THEIR RELATIVE STABILITIES. *(E.)* O'Connor, P. J. (Paterson Lab., Manchester, England), M. J. Capps and A. W. Craig. *Br J Cancer* 27(2):153-166, 1973.

The reaction of the hepatocarcinogen N,N-dimethylnitrosamine (DMN) was compared with that of methyl methanesulphonate (MMS), a methylating agent which is not a liver carcinogen. Male Wister rats were given single i.p. injections and were allowed access to food and water. Rats weighing 230-250 g received injections of MMS, 50 mg/kg body weight, and rats weighing 210-220 g were injected with DMN, 2 mg/kg. Consistent differences were observed in the reaction of rat liver DNA *in vivo* with these agents; O^6-alkylation and the production of unidentified acid-labile products were distinctive features of the reaction with the carcinogenic nitroso compound but were undetectable or in low yield, resp., after reaction with the alkyl sulphonate. Methylation of DNA by MMS was about 50% complete by the first 15 min and was maximal between 2 and 4 hr. With DMN, methylation was maximal in 5 hr. The comparison made between MMS and DMN treated animals suggests that if reaction with nucleic acids is germane to the process of liver carcinogenesis then those modifications to DNA structure most likely to be implicated are O-methylguanine and the unstable products yielding the early peak materials. The detection of excision processes for abnormal components suggests that events leading to the development of tumors may be related to the efficiency of cellular excision system for certain products of alkylation rather than to the level of alkylation obtained at a particular reaction site.

1240 PCB DIET. *(E.)* Andrews, E. J. (Coll. Med., Pennsylvania State U., Hershey); Jowett, D. (U. Wisconsin, Green Bay); Allen, J. R. (Med. Sch., U. Wisconsin, Madison) and D. H. Norback. *Science* 180(4083):255-257, 1973.

Two letters criticizing two different aspects of a previously published article (Allen and Norback, Feb. 2, 1973, p. 498) on the effects of feeding polychlorinated biphenyls (PCB) to macaques are presented as well as the original authors' reply to the criticisms. The first letter criticizes a suggestion in the original article that the carcinogenic potential of PCBs should be investigated because they caused a particular gastric mucosal lesion in the macaque. Andrews claims that these lesions have been reported only in *Macaca* species and that they occur in response to a variety of other agents as well. The authors reply that hypertrophic gastritis, with a similar invasion of the muscularis mucosae, occurs in man and has been reported in three species of the genus *Macaca*, in the rat, and in the baboon. The evidence cited indicates that this is not a lesion peculiar to macaques, and the high phylogenetic position of this animal makes it more suitable for the investigation of disease processes of man than the more primitive primates. The second letter takes exception to the statement that the concentration of PCBs in the experimental diet was less than an order of magnitude greater than that occurring in random samples of food sold in the U.S. It is claimed that the experimental diets contained concentrations at least two orders of magnitude greater than those in the general diet. The authors reply that their figures were those for the highest concentration of PCBs found in food samples randomly taken by the FDA. It is also pointed out that the time factor must be taken into consideration and that, whereas the animals developed the lesions within three months, dietary contamination by the compound would ensure intake for indefinite lengths of time.

1241 RETICULUM CELL NEOPLASMS IN NORMAL AND BENZENE TREATED HAIRLESS MICE. *(E.)* Laerum, O. D. (Inst. Path., U. Oslo, Norway). *Acta Pathol Microbiol Scand (A)* 81(1):57-63, 1973.

The frequency of reticulum cell neoplasms in benzene treated hairless mice in which no previous carcinogenic initiation had been performed was investigated. The spontaneous frequency and histological types of reticulum cell neoplasms in this same strain of mice was also examined. Hairless mice of the strain hr/hr, 6 to 8 wk old at the beginning of the study, were used. Half of the animals (35) were painted regularly twice weekly on the back skin with benzene. The other half (36) of the mice were not given any treatment. The animals were observed for up to 2 yr. The tumor frequency was about the same in the benzene treated (37.1%) and the untreated animals (38.9%). Of the total number of animals, 38% had various kinds of reticulum cell neoplasms, including reticuloses, malignant lymphomas, and leukemia. The following conclusions were drawn from this study: 1) the strain of hairless mice investigated has a high spontaneous frequency of reticulum cell neoplasms with increasing age; these are mostly generalized reticuloses which affect several organs; 2) the spontaneous frequency of these neoplasms is not affected by applications of benzene when painted on the skin twice weekly during the whole life span of the animals; and 3) the most frequent type of reticulosis in this strain of mice is reticulolymphogranulomatosis, resembling Hodgkin's disease in man.

1242 CO-CARCINOGENIC ACTION OF SACCHARIN IN THE CHEMICAL INDUCTION OF BLADDER CANCER. *(E.)* Hicks, R. M. (Middlesex Hosp. Med. Sch., London, England), J. St. J. Wakefield and J. Chowaniec. *Nature* 243(5406):347-349, 1973.

The effect of dietary saccharin (2 g/kg body wt/day) on the development of bladder carcinoma was studied in female Wistar rats which received a single intravesicular instillation of N-methyl-N-nitrosourea

(NMU). Whereas controls which received NMU alone showed a transient hyperplastic response of the bladder epithelium, 9 of 12 animals receiving both NMU and saccharin developed gross hyperplasia with capillary invasion (4 animals), papillary or nodular outgrowths (2 animals), or frank invasive carcinoma (3 animals). Controls fed saccharin alone showed slowly developing, mild bladder hyperplasia over the same time period.

1243 CHROMOSOMES OF SIX PRIMARY SARCOMAS INDUCED IN THE CHINESE HAMSTER BY 7,12-DIMETHYLBENZ(a)ANTHRACENE. *(E.)* Mitelman, F. (Inst. Pathol., Univ. Lund, Sweden), J. Mark and G. Levan. *Hereditas* 72(2):311-318, 1972.

The karyotypic patterns of six primary 7,12-dimethylbenz(a)anthracene-induced Chinese hamster sarcomas were studied. Four of the sarcomas had normal diploid stemlines, one stemline was hypodiploid and one was hypertriploid. Three of the four diploid lines showed a marked nonrandom tendency to develop hyperdiploid and polyploid sidelines. The variation in three of four of these sidelines, as well as in the hypertriploid and hypodiploid stemlines, preferentially involved chromosome number 11. Less regular but frequent changes were also observed with chromosomes numbers 5 and 7. One of the diploid stemlines which tended to develop heteroploid sidelines also had a 32% incidence of small accessory "double-minute" chromosomes. The chromosomal changes induced by DMBA were distinctly different from those in morphologically identical sarcomas induced in Chinese hamsters by Rous sarcoma virus.

1244 REACTION OF DIETHYL PYROCARBONATE WITH NUCLEIC ACID COMPONENTS. BASES AND NUCLEOSIDES DERIVED FROM GUANINE, CYTOSINE, AND URACIL. *(E.)* Vincze, A. (Sch. Chem. Sci., U. Illinois, Urbana), R. E. L. Henderson, J. J. McDonald and N. J. Leonard. *J Am Chem Soc* 95(8):2677-2682, 1973.

The reaction of diethyl pyrocarbonate (DP) with the major and some minor nucleic acid bases and nucleosides in aqueous media was studied at pH values customarily used when DP is utilized for nuclease inhibition and nucleic acid isolation. Reaction products were identified by their absorption spectra obtained from several different physical methods. Adenosine and guanosine reacted with excess DP to give ring-opened dicarbethoxylated derivatives as the major products. Unlike adenine, the free base, guanosine, did not react with DP in the pH range studied. Cytosine and 5-hydroxymethylcytosine reacted with DP to give respectively 1-carbethoxycytosine and an unstable 1-carbethoxylated derivative. Cytidine reacted to give N-carbethoxycytidine. Uracil and thymine, at a slightly basic pH, both give closely related ring-substituted 1-carbethoxylated derivatives. Uridine and ribothymidine, however, reacted to form unstable products tentatively identified as the 3-carbethoxylated derivatives. Pseudouridine C, found in tRNA, reacted with DP to produce the 1-acylated derivative. In light of these results, careful consideration should be given before using DP as a nuclease inhibitor during nucleic acid extraction when unaltered molecules are desired.

1245 BINDING OF BENZO(a)PYRENE METABOLITES(S) TO MAIN AND SATELLITE CALF THYMUS DNA'S *(IN VITRO)*. *(E.)* Meunier, M. (Inst. Sci. Res. Cancer, Villejuif, France) and J. Chauveau. *FEBS Letters* 31(3):327-331, 1973.

The binding of ^{3}H-benzo(a)pyrene (^{3}H-BP) to purified calf thymus DNA was studied *in vitro*. Cs_2SO_4-Ag^+ gradient centrifugation of DNA preincubated for 15 min with ^{3}H-BP showed that, although ^{3}H-BP and/or its metabolites did not bind selectively to any one satellite DNA fraction, it did bind to a 22-37% greater extent to the total satellite DNA fraction than to total nonsatellite DNA. The results suggested that the specific activity of each DNA fraction could be correlated with its buoyant density in CsCl.

1246 CARCINOGENICITY OF CONTAMINANTS IN INDIGENOUS EDIBLE OILS. *(E.)* Ranadive, K. J. (Cancer Res Inst., Tata Mem. Ctr., Bombay, India), S. V. Gothoskar and B. U. Tezabwala. *Int J Cancer* 10(3):652-666, 1973.

Two batches of solvent-extracted ground-nut oil samples and pure argemone oil or argemone oil in combination with mustard or soybean oil were tested for carcinogenicity in two strains of inbred C17 and in inbred Swiss mice following cutaneous application, s.c. injection or gastric intubation. Five out of six solvent-extracted ground-nut oil samples (1966 batch) indigenous to India were carcinogenic by all three routes of administration in all three mouse strains, producing skin carcinomas and sarcomas and stomach tumors. A second batch of five ground-nut oils extracted with an improved food-grade n-hexane solvent in 1969 was considerably less carcinogenic than the 1966 batch. No frank carcinomas developed and no s.c. sarcomas were observed. None of 51 mice injected singly or multiply with pure argemone oil and observed for their full life span developed tumors. Likewise, none of the 58 mice which received argemone oil in soybean oil and only two of 174 mice which received argemone oil in authentic mustard oil developed tumors. Soybean or authentic mustard oil alone were noncarcinogenic. However, mice treated with a local Indian market sample of mustard oil showed a weak carcinogenic response. The response was significantly increased by addition of argemone oil.

1247 THE EFFECT OF URINARY β-GLUCURONIDASE INHIBITOR ON THE INDUCTION OF BLADDER TUMORS WITH 2-ACETYLAMINOFLUORENE IN RATS. *(E.)* Miyakawa, M. (Kyoto U. Sch. Med., Japan), O.

Yoshida, T. Harada and T. Kato. *Invest Urol* 10(4):256-261, 1973.

The effects of 2,5-di-O-acetyl-D-glucosaccharo-(1→4) (6→3)-dilactone (SLA), a potent inhibitor of β-glucuronidase, on the changes in the vesical mucosa of rats maintained on a vitamin B_6-deficient, 2-acetylaminofluorene-containing diet and of those maintained on feed containing *d,l*-tryptophan and 2-acetylaminofluorene were studied. Male Wistar rats, 7 to 8 wk old and weighing an average of 150 g were used in the experiments. The effect of β-glucuronidase inhibitor was evident in the group of animals (65 rats) receiving vitamin B_6-deficient, 2-acetylaminofluorene-containing feed with respect to ^3H-thymidine labeling index of the vesical mucosa as well as incidences of tumor development within 25 wk of dosing; early development of bladder tumors by the administration of 2-acetylaminofluorene was apparently suppressed by the β-glucuronidase inhibitor. With the groups of rats fed the *d,l*-tryptophan and 2-acetylaminofluorene-containing diets (50 rats and 51 controls which received no SLA), drug administration had no significant effect on reducing the incidence of tumor formation. The results suggest the possibility that the chemical carcinogen may be excreted in the urine not as a glucuronide but as another form of metabolite biochemically irrelevant to β-glucuronidase and acting upon the vesical mucosa to cause tumor formation.

1248 FRUCTOSE-1,6-DIPHOSPHATASE IN DMBA-INDUCED RAT MAMMARY TUMORS. *(E.)*

Lustig, V. (Dept. Pathol. Chem., U Toronto, Canada) and J. A. Kellen. *Oncology* 27(3):266-280, 1973.

The presence and some basic properties of fructose-1,6-diphosphatase (FDP-ase) in 7,12-dimethylbenz(a)anthracene (DMBA)-induced rat mammary tumors, correlations between levels of enzyme activity and tumor type, and the possibile physiological roles of this enzyme in rat mammary tumors were studied. Tumors induced in female Sprague-Dawley rats were removed by surgery, homogenized, passed through glass wool, and the supernatant used for the enzyme assay. Ten assays were performed employing the same enzyme preparation; the coefficient of variation was 3.49. Levels of activity appeared to be solely related to the secretory status of the tumor, irrespective of the grade of malignancy. The highest levels of activity were recorded in the secretory adenomas and in the secretory neoplastic growths. Tumor weight, growth rate, and the interval between induction by DMBA and the removal of tumors, did not seem to bear any relationship to the enzyme activity levels. The secretory tumors seemed to be similar with respect to FDP-ase activity to pregnant mammary glands rather than to the resting mammary gland. Enzyme levels may be correlated with the ability of the tumor to perform some of the functions of the pregnant mammary gland. These tumors may possess gluconeogenic potential and may be able, under certain circumstances, to synthesize glucose.

1249 *IN VITRO* SYNTHESIS OF RNA WITH "AGGREGATE" ENZYME, CHROMATIN, AND DNA FROM LIVER OF METHYLAZOXYMETHANOL ACETATE-TREATED RATS. *(E.)*

Grab, D. J. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), M. S. Zedeck, N. I. Swislocki and M. Sonenberg. *Chem Biol Interact* 6(4): 259-267, 1973.

Inhibition of hepatic RNA synthesis using "aggregate" enzyme, chromatin, and DNA fractions isolated from methylazoxymethanol (MAM) acetate-treated rats was examined to localize more precisely the nuclear site of such inhibition. Immature weanling male Sprague-Dawley rats, weighing 50-70 g, were injected i.v. in the tail vein with 70 mg/kg MAM acetate in 0.9% NaCl solution. Animals given injections of 0.9% NaCl solution served as controls in all experiments. The animals were killed 1 or 3 hr after treatment and "aggregate" enzyme, chromatin and DNA preparations isolated from their livers. DNA template activity for RNA synthesis *in vitro* was unimpaired while the template activity of chromatin was slightly reduced. There was a marked inhibition of UTP incorporation into RNA when the "aggregate" enzyme preparation was the source of both template and RNA polymerase. Circular dichroism analysis of the "aggregate" enzyme preparation indicated a change in conformation of the protein component. The results suggest that MAM acetate interacts with nuclear proteins and produces conformational changes which result in an increased binding of protein to DNA and, thus, in a decreased RNA synthesis.

1250 MUTATIONS AND LOSS OF TRANSFORMING ACTIVITY OF DNA CAUSED BY THE O-GLUCURONIDE CONJUGATE OF THE CARCINOGEN, N-HYDROXY-2-AMINOFLUORENE. *(E.)*

Maher, V. M. (Michigan Cancer Fdn., Detroit) and M. A. Reuter. *Mutation Res* 21(2):63-71, 1973.

The highly reactive *o*-glucuronide conjugate of N-hydroxy-2-aminofluorene (N-GL*o*-AF) which may be formed *in vivo* as a transient metabolic intermediate of the liver carcinogen, 2-acetylaminofluorene (AAF) was incubated at neutral pH and 37 C from 2 to 20 min with biologically active transforming DNA from *Bacillus subtilis* prototroph SB19. Exposure of DNA to N-GL*o*-AF for as little as 2 min resulted in marked loss (to 6% of control) of the ability of the DNA to transform the tryptophan dependent T3 recipient to tryptophan independence. Longer incubation periods did not result in more extensive DNA damage due to the short halflife of N-GL*o*-AF in aqueous solution. N-GL*o*-AAF also showed strong mutagenic activity as determined by its ability to produce fluorescent, indole-dependent colonies among the tryptophan-independent transformants. The mutagenic ability was directly correlated with its ability to inactivate transforming DNA. The majority (59%) of the mutations did not revert to indole-independence spontaneously and may thus have represented deletions.

1251 AN IMPROVED METHOD FOR MEASURING CROSS-LINKS IN THE DNA OF MAMMALIAN CELLS: THE EFFECT OF NITROGEN MUSTARD. *(E.)* Jolley, G. M. (Chester Beatty Res. Inst., Belmont, England) and M. G. Ormerod. *Biochem Biophys Acta* 308(2):242-251, 1973.

A method is described for quantitatively measuring both the number of interstrand cross-links and the number of strand breaks introduced into the DNA of cells by a drug. L5178Y murine lymphoma cells were labeled overnight with ^{3}H-thymidine, treated for one hour at 37 C with nitrogen mustard, x-irradiated and pipetted into an alkaline solution of detergent. The denatured DNA solution was neutralized, thus allowing the cross-linked DNA to re-nature. The solution was mechanically sheared and the denatured, sheared DNA and the re-natured, cross-linked DNA were separated by CsCl equilibrium density centrifugation. Treatment with 1 μM nitrogen mustard introduced two cross-links and one strand-break per 10^9 daltons of DNA. One cross-link per 2×10^9 daltons of DNA could be detected by this method.

1252 ARYL HYDROXYLASE INDUCTION IN THE CHICK EMBRYO BY POLYCYCLIC HYDROCARBONS. *(E.)* Jellinck, P. H. (Dept. Biochem., Queen's U., Kingston, Canada) and G. Smith. *Biochim Biophys Acta* 304(2):520-525, 1973.

The ability of various polycyclic hydocarbons to induce liver microsomal enzymes which convert 7,12-[^{14}C]dimethylbenz(a)anthracene to water-soluble products was compared in the chick embryo and the rat. The hydrocarbons (1 mg) dissolved in sesame oil (0.2 ml) were injected through the chorioallantois into the fluids surrounding a 17-18 day old embryo. The embryo was killed 24 hr later and a liver microsomal fraction prepared. Male Holtzman rats, 26-28 days old and weighing 80-90 g were injected i.p. with 1 mg hydrocarbon in 0.25 ml sesame oil and the animals killed 24 hr later. A microsomal liver fraction was prepared. A good correlation in response to the 18 hydrocarbons tested was obtained but the chick embryo was found to provide a better assay system which was more sensitive and virtually independent of external variables. The effect of pretreatment with 3-methylcholanthrene was maintained for a very much longer period in the closed system provided by the egg. Induction with 3-methylcholanthrene was both rapid and prolonged. A marked increase in the ability of liver microsomes to convert [^{14}C]dimethylbenzanthracene to water-soluble products could be seen within 4 hr after injection of 1 mg of the hydrocarbon and the effect persisted for at least 7 days when injected into younger (12-day-old) embryos. The nature of the diethyl ether-soluble dimethylbenzanthracene metabolites formed by the liver microsomes of chick embryos was shown to differ from those formed in the rat but they were not identified. No embryo mortality occurred during the experiments until the dose of hydrocarbons reached high levels.

1253 A NITROSATED ARGININE DERIVATIVE, A POWERFUL MUTAGEN. *(E.)* Endo, H. (Fac. Med., Kyushu U., Katakasu, Japan) and K. Takahashi. *Biochem Biophys Res Commun* 52(1):254-262, 1973.

E. coli and *S. typhimurium* were exposed to benzoyl-L-arginineamide (BAA), an arginine derivative. Colonies evolving after exposure were found to be *try*$^+$ and *his*$^+$, indicating that revertants were induced by a mutagenic principle in BAA. The active mutagenic principle was found to be 4-benzoylamido-4-carboxamido-n(N-nitroso)butylcyanamide. The mutagenic activity of BAA was 30 times greater than that of N-methyl-N'-nitro-N-nitrosoguanidine at neutral pH.

1254 NUCLEOSIDE TRIPHOSPHATASE ACTIVITY IN INDUCED MAMMARY TUMOURS IN RATS. *(E.)* Toth, J. (Res. Inst. Oncopathol., Budapest, Hungary), O. Csuka and J. Sugar. *Virchows Arch [Zellpathol]* 12(1):84-90, 1972.

Wistar rats were administered 20 mg 7,12-dimethylbenz-(a)anthracene (DMBA) orally and 5 mg stilbestrol i.m. to induce mammary tumors. ATPase and UTPase were assayed in DMBA-induced mammary adenomas and in a transplanted tumor from a DMBA-induced tumor. In primary tumors, ATPase and UTPase were seen on the surfaces of epithelial and myoepithelial cells. In the transplanted tumor, UTPase activity was as intensive as in the primary tumors, while ATPase was reduced, being observed on the surfaces of some cells only.

1255 EFFECTS OF 1-(4-DIMETHYLAMINOBENZYLIDENE)-INDENE (SYN. NSC 80087, 14M21, DABI) ON INBRED STRAINS OF MICE. *(E.)* Gabrielides, C. G. (Theagenion Cancer Inst., Thessaloniki, Greece), T. Sirmakechian, C. T. Bahner and A. Symeonidis. *Path Europ* 7(2):155-159, 1972.

Mice of five inbred strains were fed with a diet containing 0.6, 0.06, 0.03 or 0.02% 1-(4-dimethylaminobenzylidene)indene (DABI). DABI was highly toxic for the mice. DABI had some antitumor effect on treated mice. Nine of 17 untreated C_3H and C_3Ha^{vy} mice developed mammary tumors while none of the DABI-treated mice of these strains developed tumors. DABI was also tumorigenic, producing higher incidences of ovarian tumors, sarcomas and hepatomas than were seen in controls.

1256 A COCARCINOGEN IN THE EYE-LENS OF FISH? *(E.)* Bon, W. F. (Dept. Protein Res., U. Amsterdam, Netherlands). *Acta Morphol Neerl Scand* 10(3):223-232, 1972.

An extract having the features of a diphosphonucleotide was prepared from codfish eye lenses. The extract showed strong light absorbancy at wavelength 360 mμ. The extract was diluted to a 0.5% solution and injected s.c. into S strain female mice in amounts of 0.5 ml. Some mice were also treated with benzo(a)pyrene, 7,12-dimethylbenz(a)anthracene or 3-methylcholanthrene to determine whether the eye lens extract acted as a cocarcinogen. Mice in groups given carcinogen plus eye lens extract developed more carcinomas than either mice in groups given extract only or mice in groups given carcinogen only.

1257 HOST RESISTANCE TO GASTRIC ADENOCARCINOMATOSIS IN THREE STRAINS OF RATS INGESTING N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. *(E.)* Bralow, S. P. (Fels Inst., Philadelphia, Pa.), M. Gruenstein and D. R. Meranze. *Oncology* 27(2):168-180, 1973.

Male random-bred Wistar rats were given N-methyl-N'-nitro-N-nitrosoguanidine (NG) in drinking water in amounts of 83 mg/l for 25 or 52 weeks. Wistar-Mai-Furth and Buffalo-Mai rats were treated similarly and stomach carcinogenesis was observed. After 25 wk on NG 38% of Wistar random-bred animals developed invasive cancer of the glandular stomach, while 50% developed cancer after 52 wk on NG. Twenty-five and 52 wk on NG produced stomach carcinomas in 12% and 10%, resp., of Wistar-Mai-Furth rats. NG produced no stomach carcinomas in Buffalo-Mai rats.

1258 ASBESTOS AND MALIGNANCY. *(E.)* Webster, I. (Natl. Res. Inst. Occupational Dis. South African Med. Res. Council, Johannesburg). *S Afr Med J* 47(5):165-171, 1973.

The relative toxicity and hazards of three types of asbestos mined in South Africa is examined and efforts to determine the nature of the relationship between asbestos and malignancy are described. A series of 232 cases of diffuse pleural mesothelioma has been divided into those with occupational, environmental or no history of asbestos exposure. It is suggested that there are as yet unsolved problems in the relationship of asbestos to malignancy. The exact relationship will only be known when there is an explanation of the difference in the number of cases of mesothelioma occurring in people exposed to Cape Blue, Transvaal Blue, and amosite asbestos. Only an occassional case of mesothelioma has been found in a population exposed to Transvaal Blue and very few cases in those exposed to amosite asbestos. It is postulated that Cape crocidolite, and possibly amosite, must be associated with another factor before malignancy occurs. It is possible that another mineral is inhaled with Cape Blue and not with Transvaal Blue asbestos.

1259 TRANSPLANTABLE BREAST TUMOURS IN RATS. STUDIES ON HORMONE RESPONSE. *(E.)* Sander, S. (Rikshospitalet, Oslo, Norway), A. Jakobsen and J. Sander. *Acta Pathol Microbiol Scand (A)* 81(1):91-94, 1973.

The induction and transplantation of breast tumors in rats was examined. Breast tumors were induced in Fisher-S.P.D. rats by i.v. administration of 7,12-dimethylbenz(a)anthracene (DMBA). Among the 13 animals which received DMBA, only three developed tumors suitable for transplantation after a latency period of 2-3 months. These tumors were hormone responsive. One induced tumor was transplanted to seven ovariectomized recipients and three controls. None of the ovariectomized recipients developed tumors, whereas tumors developed in the three controls. One hormone-responsive tumor, in a state of regression after overiectomy of the host, was transplanted to five recipients which developed 12 tumors. When the tumors reached appropriate size, overiectomy was carried out; it became apparent that all the transplant tumors were hormone-unresponsive. While transplantation of hormone-responsive tumors may result in the development of hormone-unresponsive transplant tumors, the opposite was not observed. It is suggested that this change in hormone-response can be explained as a selection phenomenon rather than a transformation of the tumor during the transplant procedure. The change in hormone-response was paralleled by a change in tumor morphology.

1260 CHROMOSOME ABERRATIONS IN XERODERMA PIGMENTOSUM CELLS EXPOSED TO THE CARCINOGENS, 4-NITROQUINOLINE-1-OXIDE AND *N*-METHYL-*N'*-NITRO-NITROSOGUANIDINE. *(E.)* Stich, H. F. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada), W. Stich and R. H. C. San. *Proc Soc Exp Biol Med* 142(4):1141-1144, 1973.

The frequency of chromosome aberrations in cultured cells of a patient with xeroderma pigmentosum (XP) and of control persons following exposure to 4-nitroquinoline-1-oxide (4NQO) and *N*-methyl-*N'*-nitro-nitrosoguanidine (MNNG) was examined. The XP cells were obtained from a small punch biopsy taken from the skin of one XP patient (female, 18 yr old). Fibroblasts from two nonafflicted persons of comparable age and sex were used as controls. The 4NQO was dissolved in ethanol and the MNNG in distilled water. These stock solutions were mixed with culture medium to the desired concentration. The level of DNA repair synthesis in the XP cells was about 21% of that found in cells of nonafflicted persons following exposure to 4NQO. The XP cells show an elevated sensitivity to the chromosome damaging effect of 4NQO. Doses of 4NQO which had no effect on control cells, increased significantly the frequency of metaphase plates with chromosome aberrations in the XP cells over a 2 day period posttreatment. The frequency of chromatid breaks and exchanges of XP cells resembled that found in control cells exposed to equimolar concentrations of MNNG. According to the authors, it appears likely, but unproven, that the different sensitivity of the XP cells towards 4NQO and MNNG is linked with their different capacity to repair 4NQO and MNNG induced DNA alterations.

1261 MODIFICATION OF DIMETHYLNITROSAMINE-INDUCED CHANGES IN RENAL METABOLISM AND SUBSEQUENT EFFECT ON CARCINOGENIC ACTIVITY OF ACTINOMYCIN D AND CYCLOHEXIMIDE. *(E.)* Stewart, B. W. (Middlesex Hosp. Med. Sch., London, England) and P. N. Magee. *Eur J Cancer* 9(1):37-47, 1973.

Nucleic acid and protein metabolism in the kidney of protein-depleted female Wistar rats during the week following a single injection of dimethylnitrosamine that would cause a high incidence of renal tumors was investigated. The maximum doses of actinomycin D and cycloheximide that could be tolerated when administered to rats simultaneously with or shortly after i.p. injection of 40 mg/kg of body weight dimethylnitrosamine were determined. When administered to dimethylnitrosamine-treated rats the inhibitors did not affect the rate of metabolism of the nitrosamine or the level of alkylation of liver and kidney nucleic acids. These doses of actinomycin D and cycloheximide were shown to be sufficient to inhibit renal RNA, protein and DNA synthesis in otherwise untreated rats, and were found to have a similar effect on RNA and protein synthesis in dimethylnitrosamine-treated rats. Stimulation of renal DNA synthesis by dimethylnitrosamine 2-3 days after administration was prevented by injection of actinomycin D and cycloheximide. Renal DNA synthesis at other times was also affected by these compounds but to a lesser extent. The inhibitors did not modify the incidence of dimethylnitrosamine-induced renal tumors but actinomycin D decreased the survival time of rats bearing the tumors. It is possible that inhibition of discrete biochemical reactions in the kidney, perhaps before the first cell division after administration of dimethylnitrosamine, will prevent the induction of cancer. The available evidence suggests, however, that once this period has passed the elimination of a specific cell population, already morphologically different from normal interstitial cells, is necessary to prevent neoplasia.

1262 1'-HYDROXYSAFROLE, A PROXIMATE CARCINOGENIC METABOLITE OF SAFROLE IN THE RAT AND MOUSE. *(E.)* Borchert, P. (U. Wisconsin Med. Ctr., Madison), J. A. Miller, E. C. Miller and T. K. Shires. *Cancer Res* 33(3):590-600, 1973.

When fed as 0.5% of the diet for 8 to 10 months, 1'-hydroxysafrole induced a high incidence of hepatocellular carcinomas in male rats; safrole induced only a low incidence under these conditions. 1'-Acetoxysafrole, because of its toxicity, was fed to rats at only 0.6 the molar level of the above compounds. These rats did not develop hepatic tumors, but all of those that survived at least 6 months developed multiple papillomas of the forestomach; squamous cell carcinomas were found in the forestomachs of two of these rats. A few isolated papillomas of the forestomach were observed in rats fed 1'-hydroxysafrole. Eighty-four and 82%, respectively, of male mice given injections of 1'-hydroxy- or 1'-acetoxysafrole at 1 to 21 days of age (total dose, 9.5 μmoles) and killed at 12 to 14 months of age had liver tumors; the incidences were 40% for male mice given injections of safrole and 8% for the controls. Of the adult male mice fed 0.4 or 0.5% of safrole for 13 months and killed at 16 months, 30% had liver tumors; the incidence was 11% in the controls. Some liver tumors also developed in adult male mice fed 1'-hydroxysafrole, but the number of survivors was low. All of these liver tumors were diagnosed as highly differentiated hepatic carcinomas. Between the 14th and 16th months, 46% of the mice that were fed 1'-hydroxysafrole and that survived at least 12 months developed sarcomas, most of which were diagnosed as angiosarcomas, in the interscapular region; only two safrole-fed mice and one control mouse developed tumors at this site. Twenty s.c. injections of 18.6 μmoles each of 1'-acetoxysafrole or 1'-hydroxysafrole induced sarcomas in 30 and 8%, respectively, of adult male rats. These sarcomas were observed 12 to 18 months after the 1st injection. Skin tumors did not develop in mice treated topically 14 times with 1.8 μmoles of safrole, its 1'-hydroxy, 1'-methoxy, or 1'-acetoxy derivatives, or the corresponding 2',3'-dihydrosafrole derivatives and then given twice weekly applications of phorbol-12,13-didecanoate. On the basis of the above data and the finding that a conjugate of 1'-hydroxysafrole is excreted in the urine of rats and mice given p.o. or i.p. safrole, it is concluded that 1'-hydroxysafrole is a proximate carcinogenic metabolite of safrole.

1263 TRANSFORMATION OF RAT EMBRYO CELLS *IN VITRO* BY CHEMICAL CARCINOGENS. *(E.)* Rhim, J. S. (Microbiol. Associates, Inc., Bethesda, Md.) and R. J. Huebner. *Cancer Res* 33(4):695-700, 1973.

A cell line derived from Fischer rat embryo was treated *in vitro* with various levels of polycyclic aromatic hydrocarbons [3-methylcholanthrene, 7,12-dimethylbenz(a)anthracene, and benzo(*a*)pyrene or dimethyl sulfoxide (control)]. Cells treated only with the carcinogens underwent transformation *in vitro* and produced progressively growing transplantable tumors when injected into homologous hosts. The rat cells transformed by chemical carcinogens had the following properties: 1) the carcinogen-treated cells were morphologically altered and grew as randomly oriented multilayers; 2) the treated cells showed loss of contact inhibition and increased growth rates; and 3) the 3-methylcholanthrene-treated transformed cells were more resistant to the toxic effect of 3-methylcholanthrene.

1264 PHYSICAL BINDING OF LABELED 20-METHYLCHOLANTHRENE TO MOUSE EPIDERMAL SUBCELLULAR FRACTIONS. *(E.)* Slaga, T. J. (Pacific Northwest Res. Fdn., Seattle, Wash.), J. D. Scribner and J. M. Rice. *Cancer Res* 33(5):1032-1037, 1973.

The level and degree of selectivity of noncovalent binding that might take place between a carcinogenic hydrocarbon and mouse epidermal subcellular fractions was examined. When applied to the skin of female Charles River CD1 mice, ^{3}H-labeled 20-methylcholanthrene (MC) is physically absorbed by the chromatin, mitochondria, microsomes, and cytosol of mouse epidermis. Label remained associated with cytosol protein after removal of free MC and any low-molecular-weight metabolites by dialysis, gel filtration, and ultrafiltration; this association reached a maximum around 3 hr after application of MC to mouse skin. Although previous work reported covalent binding to mouse skin cytosol, most of the material bound to cytosol protein after mild isolation of cytosol and removal of low-molecular-weight radioactivity is easily removed by precipitation of the protein with ethanol or trichloroacetic acid. Enzymatic digestion established that most of the MC loosely bound to the cytosol or chromatin was in fact bound to protein. Electrophoresis of the MC-bound cytosol protein on 7% basic polyacrylamide gels showed that most of the activity was associated with a protein fraction that had the same mobility as bovine or mouse serum albumin. The binding to the cytosol fraction and to chromatin was inhibited by the simultaneous application of 7,12-dimethylbenz(a)anthracene or the promoter 12-*O*-tetradecanoyl-phorbol-13-acetate, and was less inhibited by benz(a)anthracene or the nonpromoter phorbol-13,20-diacetate. The binding to the cytosol receptor protein and to chromatin may be involved in the promoting stage of mouse skin tumorigenesis.

1265 IDENTIFICATION OF THE CHROMOSOMES THAT CONTROL MALIGNANCY. *(E.)* Yamamoto, T. (Dept. Genetics, Weizmann Inst. Sci., Rehovot, Israel), Z. Rabinowitz and L. Sachs. *Nature* [*New Biol*] 243(129):247-250, 1973.

The chromosomes of a line of golden hamster cells transformed after treatment with dimethylnitrosamine, nonmalignant revertants from these cells, and malignant segregants from the revertants were studied in an attempt to identify those responsible for the control of malignancy. According to the authors' model, malignant cell transformation and its reversion are controlled by the balance between genes for expression (E) and suppression (S) of malignancy. The results using the Giemsa banding technique showed an addition of about half the 5_7 chromosome which was lost in revertants and regained in *in vitro* re-revertants. A piece of 5_{10} missing in revertants was also regained in transformed cells and re-revertants. Tumors induced *in vivo* by transformed cells and *in vitro* re-revertants of these tumor cells maintained the additional piece of 5_7. These findings were consistent with the presence on 5_7 of E for malignancy and on 5_{10} of E for transformation. A piece of 7_3 was added in revertants and lost in *in vitro* re-revertants. One of the 7_3 chromosomes was lost in tumors formed by segregants from revertants and by transformed cells, and in *in vitro* re-revertants. The half of 7_3 lost in *in vitro* re-reversion was not lost in tumors from revertants which still showed suppression of transformation, whereas the trisomy in 7_2 seen in transformed cells was lost in the tumors. These results suggested that 7_3 carries S associated with transformation and 7_2 carries S for malignancy.

1266 CHANGE IN TRANSCRIPTION OF REPETITIVE DNA SEQUENCES IN *N*-METHYL-*N*'-NITRO-*N*-NITROSOGUANIDINE-TRANSFORMED HAMSTER CELLS. *(E.)* Muramatsu, M. (Cancer Inst., Tokyo, Japan), N. Nemoto, N. Inui and S. Takayama. *Cancer Res* 33(4):739-744, 1973.

Transcription of repetitive DNA in *in vitro*-transformed hamster cells was compared with that of normal control cells by the technique of DNA-RNA hybridization. Hybridization efficiencies of rapidly labeled RNA extracted from these cells were almost identical, indicating no significant increase in the concentration of hybridizable RNA in the transformed cells. Hybridization-competition experiments indicated that certain segments of repetitive DNA which had not been transcribed in the normal cells were activated in the transformed cells, whereas no detectable deletion occurred in the RNA population of transformed cells as compared with normal fibroblasts. On the other hand, the total stable RNA population was not significantly different between these two types of cells.

1267 INDUCTION BY DIMETHYLHYDRAZINE OF INTESTINAL CARCINOMA IN NORMAL RATS AND RATS FED HIGH OR LOW LEVELS OF VITAMIN A. *(E.)* Rogers, A. E. (Dept. Nutrition, Food Sci., Massachusetts Inst. Technology, Cambridge), B. J. Herndon and P. M. Newberne. *Cancer Res* 33(5):1003-1009, 1973.

Induction of intestinal tumors by intragastric administration of 1,2-dimethylhydrazine (DMH) was studied in male CD Sprague-Dawley rats, weighing 40 to 50 g and fed a semisynthetic diet containing either high, adequate, or low levels of vitamin A. Sixty to 100% of the treated rats had intestinal carcinoma 6 months after administration of the first dose of DMH. Total doses in amounts ranging from 195 to 420 mg/kg did not affect tumor incidence; however, rats given the largest total dose in the largest individual doses (30 mg/kg) developed more tumors in the colon than in other sites. Tumors were polypoid or intramural adenocarcinomas, of which a variable number produced mucus. In many cases they caused passage of bloody stools, intestinal obstruction, and/or intussusception. Tumor incidence and morphology were in all ways comparable to those reported in studies in which DMH was administered s.c. A high level of vitamin A in the diet (sufficient to raise the vitamin content in the serum and liver and reduce growth) did not change the incidence of colon tumors but decreased the number of tumors per rat at the highest DMH dose. Chronic dietary deficiency of vitamin A increased the incidence of tumors slightly and may have reduced induction time.

1268 CHEMICALLY INDUCED BINDING OF THE HEPATOCARCINOGEN *N*-MONOMETHYL-4-AMINOAZOBENZENE TO NUCLEIC ACIDS *IN VITRO*. *(E.)* Lin, J.-K. (Coll. Med., Natl. Taiwan U., Taipei, Republic of China) and K.-F. Fok. *Cancer Res* 33(3):529-535, 1973.

Chemical reactions between the hepatocarcinogen *N*-monomethyl-4-aminoazobenzene (MAB) and nucleic acids were carried out in neutral aqueous solution and at room temperature (25 C) in the presence of potassium persulfate or iodine. The data presented are consistent with the hypothesized formation of a covalent linkage between MAB and DNA or RNA under these mild conditions. The covalently linked MAB cannot be extracted by organic solvents that remove the physically bound MAB from the nucleic acid. A chemical linkage was further indicated by gel filtration and equilibrium dialysis of the nucleic acid:MAB complexes. Hydrolysis of the persulfate-induced DNA- or RNA-bound dye with deoxyribonuclease or alkali, phosphodiesterase, and alkaline phosphatase yields various nucleoside-bound dyes. The reaction of persulfate provides a new model system for a study of the activation mechanism of MAB *in vitro*.

1269 CHEMICAL CARCINOGENESIS IN TRANSPOSED INTESTINAL SEGMENTS. *(E.)* Gennaro, A. R. (Temple U. Hlth. Sci. Ctr., Philadelphia, Pa.), R. Villanueva, Y. Sukonthaman, V. Vathanophas and G. P. Rosemond. *Cancer Res* 33(3):536-541, 1973.

A research model has been devised to assist in an assessment of the relative susceptibility of small bowel and colonic mucosa to potential intestinal carcinogens, and an application of this model to azoxymethane is reported. Intestinal transpositions were performed on Sprague-Dawley rats. In one group, segments of small intestine were transposed to the ascending and descending colon and, in another group, colon segments were transposed to the level of the mid small bowel. Weekly i.m. injections of azoxymethane were started several weeks after operation. Usually, after 14 injections (although sometimes earlier) some of the rats died or presented clinical evidence of intestinal tumors. This preliminary report is based on findings obtained from specimens in these animals and in a few animals from the control groups. Eighteen rats that had a colon transposition and that received azoxymethane developed adenocarcinoma in the transposed segment. None of the 17 rats that had a small bowel transposition and that received the carcinogen developed cancer in the transposed small intestine; however, each had one or more colon cancers. Control animals that received the carcinogen, but which had no operation, developed colon cancer. The animals in both control and operated groups that received the carcinogen developed small intestinal tumors that were localized in the duodenojejunal region. Control animals that had intestinal transpositions but no carcinogen did not develop cancer. These early findings suggest that colon mucosa, regardless of its location in the intestinal tract, is susceptible to the carcinogen azoxymethane but that mucosa of the small intestine is not.

1270 INDUCTION OF ADENOCARCINOMAS OF THE COLON IN MICE BY WEEKLY INJECTIONS OF 1,2-DIMETHYLHYDRAZINE. *(E.)* Thurnherr, N. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), E. E. Deschner, E. H. Stonehill and M. Lipkin. *Cancer Res* 33(5):940-945, 1973.

Thirty-four CF1 mice were given weekly s.c. injections of 20 mg of 1,2-dimethylhydrazine hydrochloride (DMH) per kg; this treatment induced colonic carcinomas in more than 90% of the animals after 186 days. The earliest change was found to be a widening of the proliferative compartment within the colonic crypts; at a later stage, there was an increase of the tritiated thymidine-labeling index from 9.3% at day 45 to 16.2% at day 87. The first histological changes were focal hyperplastic changes and focal atypias on the tip of the folds of the distal colonic mucosa (day 38) confined to single crypts. The colonic tumors were located in the distal colon and rectum, with a high incidence of squamous cell cancers originating from the anal canal. Saline extracts of colonic tumor material precipitated specific antimouse embryonic rabbit serum. Assays of hypoxanthine phosphoribosyltransferase in the villus fraction of the small bowel of the DMH-treated tumor-carrier mice indicated lower activity, compared to that of controls. This model of chemical carcinogenesis with DMH is similar in many respects to the development of neoplasms in the human colon. The action of the carcinogen induced an enlargement of the proliferative zone in the colonic crypts, led to an increase in the total number of labeled cells in the crypts, and decreased the activity of the enzyme, hypoxanthine phosphoribosyltransferase, all of which develop in human colonic neoplasms.

1271 THE INHIBITORY EFFECT OF COPPER ON DL-ETHIONINE CARCINOGENESIS IN RATS. *(E.)* Kamamoto, Y. (Nara Med. U., Kashihara, Japan), S. Makiura, S. Sugihara, Y. Hiasa, M. Arai and N. Ito. *Cancer Res* 33(5):1129-1135, 1973.

The inhibitory effects of cupric acetate (CU) on various hepatic changes in male, Wistar rats, including the development of hepatomas induced by DL-ethionine (ethionine) were examined. By the addition of 0.25% CU, hepatoma induction was prevented in rats fed diets containing 0.25% ethionine for 24 wk. The inhibitory effect of CU depended on the administration period. The induction of hepatomas after 24 wk by diets that contained ethionine was completely prevented by the addition of CU for 12, 16, or 20 wk but not by its addition for only 4 or 8 wk. CU did not inhibit the proliferation of nodular hyperplasia induced in rats by ethionine. Nodular hyperplasia was not seen in the liver of rats treated with ethionine and CU for 20 wk, but it developed in the liver of rats treated with ethionine and CU for 4 to 16 wk. Histochemical examination showed deposits of copper in the liver in parenchymal cells in periportal areas but not in Kupffer cells, oval cells, or cells of bile duct epithelium. Electron microscopy showed dense bodies surrounded by a single membrane in the liver parenchymal cells of rats treated with ethionine and CU but not in those of rats treated with ethionine alone. Biochemical examination showed that the copper content of the liver increased in rats treated either with ethionine plus CU or with CU alone. After treatment with ethionine and CU, the nuclear fraction had the highest copper concentration, followed by the supernatant fraction. From these results, it is suggested that copper was bound to ethionine and deposited in the nuclei. The reason for the protective effect of copper in ethionine carcinogenesis is unknown.

1272 REACTION OF *N*-METHYL-*N*-NITROSOUREA WITH DNA OF NEURONAL AND GLIAL CELLS *IN VIVO*.

(E.) Kleihues, P. (Middlesex Hosp. Med. Sch., London, England), P. N. Magee, J. Austoker, D. Cox and A. P. Mathias. *FEBS Letters* 32(1):105-108, 1973.

Nuclei isolated from the brains of female Wistar rats that had received, two hr previously, an i.v. injection of *N*[^{14}C]methyl-*N*-nitrosourea (^{14}C-NMU), were fractionated according to cell type into five fractions in discontinuous sucrose gradients. Hydrolyzed DNA of each nuclear fraction was assayed by ion exchange chromatography for 7-methylguanine. The results indicated that, although inducing almost exclusively gliomas, NMU reacted more extensively with DNA from nuclei of the neuronal fraction than with that from astrocytes and oligodendrocytes. It was thus concluded that there is no difference between the extent of initial interaction of NMU with the neuronal and glial cells and that the failure of neuronal tumor induction is possibly determined by the inability of these cells to divide.

1273 CHANGES IN THE PHOSPHOLIPID COMPOSITION OF THE LIVER ENDOPLASMIC RETICULUM AFTER INDUCTION WITH DRUGS AND CARCINOGENS.'

(E.) Davison, S. C. (St. Bartholomew's Hosp. Med. Coll., London, England) and E. D. Wills. *Biochem Soc Trans* 1(2): 362-364, 1973.

Liver microsomal phospholipid synthesis was studied in male Wistar rats injected i.p. with phenobarbital or 20-methylcholanthrene (20-MC). Neither phenobarbital nor 20-MC altered the proportions of the different phospholipids in the endoplasmic reticulum which consisted primarily of phosphatidylcholine (51.3%), phosphatidylethanolamine (20.2%), phosphatidylinositol (10.3%), phosphatidylserine (5.0%) and sphingomyelin (3.9%). Phenobarbital administration produced a decrease in the percentage of $C_{20:4}$ and $C_{22:6}$ fatty acids, especially in phosphatidylcholine and phosphatidylethanolamine. 20-MC, however, had no effect on the proportions of saturated and unsaturated fatty acids in phospholipid. 20-MC, but not phenobarbital, stimulated *in vivo* transethylase activity as determined from the extent of [1,2-^{14}C]-ethanolamine incorporation into phospholipid. Although phenobarbital stimulated microsomal S-adenosylmethionine-phosphatidylethanolamine methyltransferase activity *in vitro*, 20-MC failed to do so. It was concluded, therefore, that the previously observed increase in membrane phospholipids following phenobarbital administration was not due to increased synthesis of phosphatidylcholine by the transmethylaction pathway.

1274 THE METABOLISM OF THE NATURALLY OCCURRING HEPATOCARCINOGEN SAFROLE TO 1'-HYDROXYSAFROLE AND THE ELECTROPHILIC REACTIVITY OF 1'-ACETOXYSAFROLE.

(E.) Borchert, P. (U. Wisconsin Med. Ctr., Madison), P. G. Wislocki, J. A. Miller and E. C. Miller. *Cancer Res* 33(3):575-589, 1973.

A conjugated form of 1'-hydroxysafrole was identified as a urinary metabolite of safrole; the conjugate was cleaved by commercial β-glucuronidase preparations. The conjugated 1'-hydroxysafrole accounted for 1 to 3% of an i.p. dose of safrole administered to rats, hamsters, or guinea pigs. Pretreatment of rats with phenobarbital or 3-methylcholanthrene increased about 10-fold the excretion of 1'-hydroxysafrole conjugate(s) after a dose of safrole; these treatments had little effect on the urinary excretion of conjugated 1'-hydroxysafrole after injection of the 1'-hydroxy derivative. Pretreatment with phenobarbital did not increase the excretion of conjugated 1'-hydroxysafrole by guinea pigs or hamsters given safrole injections. Male mice excreted 30% or more of a dose of safrole or 1'-hydroxysafrole as a conjugate of the latter compound. The 1'-hydroxysafrole released by β-glucuronidase treatment of urine from rats given safrole injections was isolated and characterized by comparison of its ultraviolet, nuclear magnetic resonance, and mass spectra with those of the synthetic compound. The synthetic model ester 1'-acetoxysafrole is an electrophilic reactant. It reacts at neutrality with methionine to yield 3'-methylmercaptoisosafrole, which was characterized by the identity of its ultraviolet, nuclear magnetic resonance, and mass spectra with those of the compound prepared from 3'-bromoisosafrole and methylmercaptan. 1'-Acetoxysafrole also reacts with guanosine and adenosine and, probably to a small extent, with cytidine as evidenced by the formation in reaction mixtures containing ^{14}C-labeled nucleosides of ^{14}C-containing products which are less polar than the parent nucleosides. These products did not contain a significant quantity of ^{3}H from 1'-acetoxysafrole-acetoxy-^{3}H. A major nucleotide product was isolated from a large-scale reaction of 1'-acetoxysafrole with guanosine monophosphate. This nucleotide was degraded to the nucleoside, which was acetylated. The ultraviolet, nuclear magnetic resonance, and mass spectra of the acetylated nucleoside are consistent with its characterization of *O*-6-(isosafrol-3'-yl)-*N*-2-acetylguanosine-2',3',5'-triacetate.

1275 MACROMOLECULAR SYNTHESIS FOLLOWING A SINGLE APPLICATION OF ALKYLATING AGENTS USED AS INITIATORS OF MOUSE SKIN TUMORIGENESIS.

(E.) Slaga, T. J. (U. Wisconsin Med. Ctr., Madison), G. T. Bowden, B. G. Shapas and R. K. Boutwell. *Cancer Res* 33(4): 769-776, 1973.

After the application of initiating dosages of certain alkylating agents, the incorporation of thymidine-^{3}H, cytidine-^{3}H, and leucine-^{3}H into DNA, RNA, and protein, resp., in mouse skin epidermal preparations was determined. Groups of 20 to 30 female Charles River CD1 mice, 7 to 9 wk old, received a single topical application of an alkylating agent. This treatment was followed, beginning 1 wk later by 2 applications of 0.25% croton oil weekly for the duration of the experiment. Histological changes were compared with the metabolic activities. β-Propiolactone, γ-propane sultone, and bis(chloromethyl) ether inhibited DNA synthesis for periods up to 24 hr; this was followed by stimulation and in turn, by epidermal hyperplasia. The maximal rate of RNA synthesis with both β-propiolactone and bis(chloromethyl) ether was attained at 12 hr, whereas the major peak with γ-propane sultone was observed at 2 days. Urethane depressed DNA synthesis for 24 hr without a subsequent increase and without detectable epidermal hyperplasia. Chloromethyl methyl ether, a weak or inactive initiating agent, produced no change in macromolecular synthesis, and the skin sections appeared normal. When either β-propiolactone or urethan was applied to mouse skin in which DNA synthesis had been prestimulated with a noncarcinogenic irritant, these agents blocked DNA synthesis.

1276 HISTONE IV BINDING OF 3,4-BENZPYRENE, PYRENE, ANTHRACENE, AND 9-METHYLANTHRACENE. *(E.)* Small, E. W. (Dept. Biochem., Biophys., Oregon St. U., Corvallis), A. M. Craig and I. Isenberg. *Biopolymers* 12(5):1149-1160, 1973.

Ultraviolet absorption, fluorescence excitation and emission, and fluorescence anisotropy demonstrate that the polycyclic aromatic hydrocarbons 3,4-benzypyrene, pyrene, anthracene, and 9-methylanthracene bind to histone IV after the slow conformational change in the presence of divalent phosphate anion. These hydrocarbons do not bind, within the limits of detection of this study to histone IV in the disordered form at low pH or to the form produced by the fast conformational change when the slow change is prevented. On binding the wavelength maxima of both the excitation and emission spectra of the hydrocarbons shift 2-8 mm to the red, a small shift compared to that observed on binding to DNA. High anisotropy values for the bound hydrocarbon indicate a high degree of aggregation for the histone IV; the hydrocarbon molecules must be rigidly fixed with respect to the aggregate.

1277 BENZIDINE EXPOSURE AS A CAUSE OF BLADDER TUMORS. *(E.)* Zavon, M. R. (U. Cincinnati Coll. Med., Ohio), U. Hoegg and E. Bingham. *Arch Environ Health* 27(1):1-7, 1973.

The results of a 13-year observance of 25 men who were employed in the manufacture of benzidine are reported. Thirteen of twenty-five exposed men developed tumors of the bladder. The common chemical to which all were exposed was benzidine. However, multiple exposures to other highly suspect chemicals did occur, and they cannot be entirely discounted. The respiratory route is of major importance as a portal of entry, at least under some manufacturing conditions. Urinary pap smears should be the basic surveillance of persons at special risk of cancer of the urinary bladder. When a skilled and experienced reader is not available, education in the significance of hematuria can be very effective. A reason was sought for the initiation of this epidemic of bladder carcinoma, the case reported in 1958 being the first noted in more than 25 years of manufacture. Despite evaluation of changes in the process over the years, a particular process change that might be the cause of this cluster of cases could not be identified. The number of persons exposed should be limited. Rotation of employees does not decrease risk to the employee but does increase the number at risk.

1278 ERYTHROLEUKAEMIA IN A MELPHALAN-TREATED PATIENT WITH PRIMARY MACROGLOBULINAEMIA. *(E.)* Petersen, H. S. (U. Dept. Internal Med. II, Amtssygehuset, Aarhus, Denmark). *Scand J Haematol* 10(1):5-11, 1973.

The case of a 78-yr-old man who was treated for 3½ yr with melphalan (procarbazine) for primary macroglobulinemia and who subsequently developed erythroleukemia is reported. The patient, who, at the time treatment was initiated, had a normocytic, normochronic anemia with a hemoglobin of 8.1 g/100 ml, received a loading dose of 5 mg melphalan daily for four weeks followed by maintenance with 2 to 6 mg per week. He also received androgen therapy. Eventually, the patient's bone marrow became hyperplastic, with erythroblasts which comprised 95% of the marrow population. A sharp rise in peripheral erythrocytes occurred. Terminally, peripheral myeloblasts also increased. Autopsy revealed leukemic infiltration of the liver, lymph nodes, adrenals, leptomeninges, basal ganglia, spleen and kidneys. A total of eight patients who developed acute leukemia following prolonged melphalan therapy have so far been reported, suggesting a possible hazard for patients receiving such a therapeutic regimen.

1279 NEOPLASMS IN WISTAR RATS AFTER AN N-METHYL-N-NITROSOUREA INJECTION. *(E.)* Murthy, A. S. K. (Children's Cancer Res. Fdn., Boston, Mass.), G. F. Vawter and A. Bhaktaviziam. *Arch Pathol* 96(1):53-57, 1973.

A variety of nonneoplastic and neoplastic lesions were induced in 30-day old Wistar rats after a single i.v. injection of N-methyl-N-nitrosourea at a dose of 70 mg/kg body weight. Nonneoplastic lesions included retinal atrophy, cataract, hyperkeratosis, and cystic degeneration of pancreatic islets. In treated rats, tumors developed in the kidney, lung ovary, uterus, colon, jaw, pituitary gland, and mammary glands. Lymphomas, leiomyoma and hemangioma also developed. Both the renal tumors and lymphomas proliferated in cell culture. A sexual dimorphism in the morphologic structure of renal tumors was not apparent, and these tumors could not be transplanted in unconditioned adult hosts of the same strain. An association between renal and endocrine tumors suggests that the kidney, aside from its sensitivity to steroids, may have the potentiality of an endocrine gland.

1280 LACK OF SIGNIFICANT VISIBLE C-TYPE VIRUS ACTIVATION IN THE BONE MARROW AND THYMUS OF MICE GIVEN A LEUKAEMOGENIC DOSE OF METHYLNITROSOUREA. *(E.)* Frei, J. V. (Dept. Pathol., U. Western Ontario, London, Canada), R. Iwasutiak and G. Viragos. *Chem Biol Interact* 6(5):333-337, 1973.

Bone marrow aspirates and sections of thymus from female CFW/D mice were examined under the electron microscope for C-type viruses at various times up to 20 days following i.p. injection with a leukemogenic dose (0.6 mmole/kg) of methylnitrosourea. A small increase in C-type particles, ranging from two to four times control numbers, was observed in both tissues from treated mice. No significant difference in the number of particles was observed between bone marrow and thymus. The viruses were almost invariably seen budding off undifferentiated cells. The presence of virus in control tissues and the small increase in particles in tissues from the treated mice suggest that this increase may have been related to tissue regeneration rather than to chemical activation.

1281 CONTRIBUTION OF HOST IMMUNITY TO CYCLOPHOSPHAMIDE THERAPY OF A CHEMICALLY-INDUCED MURINE SARCOMA. *(E.)* Moore, M. (Robert Jones, Agnes Hunt Orthopaedic Hosp., Oswestry, Shropshire, England) and D. E. Williams. *Int J Cancer* 11(2):358-368, 1973.

1282 CALCITONIN AND OSTEOGENIC SARCOMA. *(E.)* Walton, I. G. (Western Gen. Hosp., Edinburgh, Scotland) and J. A. Strong. *Lancet* (7808):887-888, 1973.

1283 TISSUE ZINC IN MALIGNANT DISEASE. *(E.)* Griffith, K. (Whittington Hosp., London, England), E. B. Wright and T. L. Dormandy. *Nature* 241(5384):60, 1973.

1284 FACTORS INFLUENCING THE RATE OF FORMATION OF NITROSOMORPHOLINE FROM MORPHOLINE AND NITRITE: ACCELERATION BY THIOCYANATE AND OTHER ANIONS. *(E.)* Fan, T.-Y. (Dept. Nutrition, Food Sci., Massachusetts Inst. Technol., Cambridge) and S. R. Tannenbaum. *J Agric Food Chem* 21(2):237-240, 1973.

1285 THE MUTAGENICITY OF BENZIMIDAZOLE AND BENZIMIDAZOLE DERIVATIVES. III. THE INFLUENCE OF THE 2-SUBSTITUENT IN BENZIMIDAZOLE ON THE MUTAGENIC ACTIVITY. *(E.)* Seiler, J. P. (Swiss Federal Res. Station Arboriculture, Viticulture, Horticulture, Wädenswil, Switzerland) and H. Limacher. *Chimia* 27(2):68-70, 1973.

1286 EFFECTS OF INSTALLATION OF PARAFFINS IN TISSUES. *(E.)* Campbell, J. S. (Ottawa Gen. Hosp. Ontario, Canada) and I. W. D. Henderson. *Lancet* (7806): 775-776, 1973.

1287 SARCOMA AFTER INJECTION OF INTRAMUSCULAR IRON. *(E.)* MacKinnon, A. E. (Royal Manchester Childrens Hosp., England) and J. Bancewicz. *Br Med J* (5861): 277-279, 1973.

1288 EPOXY DERIVATIVES OF AROMATIC POLYCYCLIC HYDROCARBONS. THE PREPARATION AND METABOLISM OF EPOXIDES RELATED TO 7,12-DIMETHYLBENZ[*a*]ANTHRACENE. *(E.)* Sims, P. (Chester Beatty Res. Inst., London, England). *Biochem J* 131(2):405-413, 1973.

1289 FEEDING STUDIES OF NITRILOTRIACETIC ACID AND DERIVATIVES IN RATS. *(E.)* Lijinsky, W. (U. Nebraska Med. Ctr., Omaha), M. Greenblatt and C. Kommineni. *J Natl Cancer Inst* 50(4):1061-1063, 1973.

1290 ASBESTOS AND CANCER OF THE HEAD AND NECK. *(E.)* Holmes, S. (Asbestos Res. Council, Rochdale, England). *Lancet* (7807):839-840, 1973.

1291 CYTOKINETIC STUDY ON TOXIC ACTION OF SODIUM CYCLAMATE AND CYCLOHEXYLAMINE. *(E.)* Koizumi, A. (Fac. Med., U. Tokyo, Japan), Y. Tachibana, Y. Dobashi, K. Tsuda, T. Okino and H. Katsunuma. *Ind Health* 9(4):188-193, 1971.

1292 AN ATTEMPT ON A CAUSE AND PREVENTION OF OESOPHAGEAL CANCER IN THE TRANSKEI BANTU. *(E.)* Roach, W. A. (Bantu Cancer Chem. Res., Butterworth, Transkei, South Africa). *S Afr Med Tydskr* 47(8):347-348, 1973.

1293 STANDARDS FOR CARCINOGENS. *(E.)* Stellman, J. M. (Oil, Chem., Atomic Workers Internatl. Union, Denver, Colo.). *Science* 181(4095):109, 1973.

1294 STEATORRHOEA AND JEJUNAL RETICULUM CELL SARCOMA OCCURRING IN A PATIENT ON LONG-TERM COLCHICINE THERAPY FOR GOUT. *(E.)* Boucher, B. J. (London Hosp., England) and J. T. Wright. *Postgrad Med J* 49(568):106-107, 1973.

1295 EFFECT OF CIGARETTE SMOKING ON PRIMARY AND SECONDARY HUMORAL RESPONSES OF MICE. *(E.)* Thomas, W. (U. Western Australia, Sch. Med., Perth), P. G. Holt and D. Keast. *Nature* 243(5404):240-241, 1973.

1296 STIMULATION OF CELL DIVISION BY CROTON OIL IN BLUE-GREEN BACTERIA. *(E.)* Ingram, L. O. (Oak Ridge Natl. Lab., Tenn.) and W. D. Fisher. *J Bacteriol* 114(2):874-875, 1973.

1297 PHYSIOLOGIC DISPOSITION OF 1-(2-CHLOROETHYL)-3-CYCLOHEXYL-1-NITROSOUREA (CCNU) AND 1-(2-CHLOROETHYL)-3-(4-METHYL CYCLOHEXYL)-1-NITROSOUREA (MeCCNU) IN MAN. *(E.)* Sponzo, R. W. (Natl. Cancer Inst., Bethesda, Md.), V. T. DeVita and V. T. Oliverio. *Cancer* 31(5):1154-1159, 1973.

1298 POLYCHLORINATED BIPHENYLS: A NEW TYPE OF INDUCER OF CYTOCHROME P-448 IN THE LIVER.

(E.) Alavares, A. P. (Rockefeller U., New York, N.Y.), D. R. Bickers and A. Kappas. *Proc Natl Acad Sci USA* 70(5):1321-1325, 1973.

1299 LUNG CANCER IN CHLOROMETHYL METHYL ETHER WORKERS. *(E.)* Figueroa, W. G. (Germantown Dispensary Hosp., Philadelphia, Pa.), R. Raszkowski and W. Weiss. *N Engl J Med* 288(21):1096-1097, 1973.

See also:

* (Rev): 1214, 1218, 1229
* (Viral): 1363, 1409
* (Immun): 1546, 1548
* (Path): 1583
* (Epid-Biom): 1589

1300 TRANSFORMATION OF MAMMALIAN CELLS *IN VITRO* BY LOW DOSES OF X-RAYS. *(E.)* Borek, C. (Dept. Radiol., Columbia U., New York, N.Y.) and E. J. Hall. *Nature* 243(5407):450-453, 1973.

Primary cultures of golden hamster whole embryo cells were X-irradiated with 1-600 rad, cloned, and the number of transformed clones was determined in order to obtain a dose-response curve. Transformation was evaluated on the basis of morphology, cloning ability in semi-soft agar, and agglutinability by concanavalin A and wheat germ agglutinin. X-irradiation with as little as one rad induced transformation. The percentage of transformed cells increased with increasing dosage to a plateau of 1% at 150-300 rad. At higher doses, there was a marked decrease in the number of transformed cells. Transformed cells tended to be aneuploid, but no gross chromosomal abberations were observed. No C-type viruses were seen in any of the X-ray transformed cells, and no transformed clones were observed in control cultures.

1301 RELATIONSHIP OF DNA REPAIR TO CARCINOGENESIS IN XERODERMA PIGMENTOSUM. *(E.)* Robbins, J. H. (Natl. Cancer Inst., Bethesda, Md.) and P. G. Burk. *Cancer Res* 33(5):929-935, 1973.

Radioautograms of intact ultraviolet (UV)-irradiated epidermis from a patient with xeroderma pigmentosum showed no detectable abnormality in UV-induced thymidine-^{3}H incorporation. This result is consistent with findings in this patient's UV-irradiated skin fibroblasts and lymphocytes and contrasts with findings in cells from typical xeroderma pigmentosum patients, all of which exhibit an impaired ability to repair UV-damaged DNA. The development of numerous tumors in the presence of apparently normal DNA repair suggests that some mechanism other than enhancement of UV carcinogenesis by defective DNA repair may be responsible for skin tumor formation in this patient and perhaps in all patients with xeroderma pigmentosum.

1302 INHIBITION BY PROGESTERONE OF RADIATION-ESTROGEN-INDUCED MAMMARY CANCER IN THE RAT. *(E.)* Segaloff, A. (Alton Ochsner Med. Fdn., New Orleans, La.). *Cancer Res* 33(5):1136-1137, 1973.

The effect of progesterone on radiation-estrogen induced mammary cancer in female A x C rats was examined. The data from this preliminary experiment make it appear that continuous administration of progesterone protects animals from the synergism between estrogen and radiation. However, it also appears that the progesterone has by itself a protective effect against the individual carcinogenic agents since fewer tumors occurred on the opposite side as well as on the radiated side. In this preliminary experiment none of the animals that bore just progesterone pellets and were radiated developed mammary carcinomata.

1303 THE EFFECT OF VARIATIONS IN *LET* AND CELL CYCLE ON RADIATION HEPATOCARCINOGENESIS. *(E.)* Wiley, A. L., Jr. (U. Wisconsin Med. Ctr., Madison), H. H. Vogel, Jr., and K. H. Clifton. *Radiat Res* 54(2):284-293, 1973.

The effect of variations in linear energy transfer (*LET*) and cell cycle on radiation carcinogenesis was studied in LAF$_1$/Jax mouse liver. It was found that both the quality (*LET*) of the radiation and the addition of a proliferative stimulus significantly alter the hepatocarcinogenic endpoint. A single, whole-body, 200-rad dose of ^{137}Cs gamma rays increased the hepatoma incidence by approximately 3- to 4-fold over the control rates, when a pure proliferative stimulus (partial hepatectomy) was added. Also a single, whole-body, 200-rad dose of fission neutron irradiation increased the hepatoma incidence by approximately 6- to 9-fold over the control rate, when combined with partial hepatectomy. Furthermore, both the ^{137}Cs and fission neutron hepatocarcinogenic rates were enhanced regardless of whether the irradiation was given either during liver regeneration or 8-13 weeks prior to partial hepatectomy. However, neither ^{137}Cs nor fission neutron irradiation alone (i.e., without partial hepatectomy), significantly increased the incidence of hepatomas. The highest incidence of hepatomas observed was nine times the unirradiated control incidence and 50% greater than that in the next highest experimental groups, and occurred in mice irradiated with fission neutrons 24 hours after partial hepatectomy. The difference between this group and those irradiated with neutrons 36.5 and 43.5 hours after hepatectomy was not, however, statistically significant. These and other data indicate that the role of the cell cycle phase in susceptibility to carcinogenesis warrants further investigation and that radiation appears to be the carcinogenic agent of choice in such investigations.

1304 EFFECT OF DUST OF RARE EARTH METALS CONTAINING A RADIOACTIVE COMPONENT ON INCIDENCE OF RETICULOSARCOMA OF THE LUNG. *(E.)* Likhachev, Yu. P. (Inst. Work Hyg. Occupational Dis., Acad. Med. Sci. USSR, Moscow), P. P. Lyarskii and L. T. Elovskaya. *Bull Exp Bio Med (USSR)* 74(11):1426-1429, 1973.

The effect of radioactive dusts on the incidence of reticulosarcoma of the lungs of random-bred albino rats was studied. Groups of rats received single intratracheal injections of saline-suspended dusts of the rare earths which contained thorium at specific activities ranging from 10^{-11} to 10^{-7} Ci/g. Lung tumors developed in 13 of 250 animals (5.2%) between 15 and 21 months following thorium dust injection. Nine other animals exhibited precancerous states (e.g., bronchial squamous metaplasia, epithelial proliferation, hyperplasia). Tumor incidence, which was eight to ten times higher than in the control group, was related to the amount of radioactivity given. Histologically, the tumors consisted of 11 reticulosarcomas, one endothelioma and one anaplastic carcinoma. The dust and, especially, the radioactivity were considered to play the major role in pathogenesis of tumors in the treated animals.

1305 LETHAL DAMAGE AFTER X- AND UV-IRRADIATION AND ITS MODIFICATION BY HYPOXIA AND CYCLOHEXIMIDE. *(E.)* Horsley, R. J. (Hamilton Clin., Ontario, Canada), S. N. Banerjee, A. Laszlo and K. Parker. *Rad Res* 54(1):110-120, 1973.

The effects of hypoxia and cycloheximide on giant cell formation after x- or uv-irradiation of *Oedogonium cardiacum* haploid zoospores have been studied. Oxygenated cells irradiated with either 200-1500 rads x-rays or 3 kerg/mm^2 uv-light yielded 86% giant cells. Cells grown and irradiated in a nitrogen atmosphere showed about the same yield of giant cells; however, the peak value for cells irradiated in nitrogen occurred at twice the dose as that for cells irradiated in oxygen. This difference in dose response was presumably due to a decreased reproductive capacity in the presence of nitrogen. Treatment of G_2 cells with 1 μg/ml cycloheximide following x- or uv-irradiation increased cell survival and concomitantly reduced the number of progeny giant cells. It was postulated that giant cells were formed due to abnormal protein synthesis, presumably due to malformed microtubules in the cell wall, and that cycloheximide treatment prevented this expression of potentially lethal radiation damage.

1306 THYROID CANCER AFTER RADIOACTIVE IODINE DIAGNOSTIC PROCEDURES IN CHILDHOOD. *(E.)* Pilch, B. Z. (Natl. Cancer Inst., Bethesda, Md.), C. R. Kahn, A. S. Ketcham and D. Henson. *Pediatrics* 51(5):898-902, 1973.

A case is presented in which thyroid carcinoma was found in a 20-yr-old woman who had undergone diagnostic radioactive iodine uptake studies at the ages of 4 and 12 yr for suspected hyperthyroidism. She had no history of external radiation to the neck, and the only family history of thyroid disease was benign goiter in her mother and grandmother. The patient presented at 20 yr with an asymptomatic right neck mass the biopsy of which provided a histologic diagnosis of metastatic papillary carcinoma consistant with primary thyroid gland involvement. Histologic studies of resected tissues at surgery revealed a mixed papillary and follicular carcinoma in the right lower and left upper lobes of the thyroid, a fetal adenoma in the right lower lobe, and metastatic papillary carcinoma in the right cervical nodes and in one left paratracheal node. Tumor was also present in the surrounding areas of soft tissue. There was no evidence of radiation damage to the thyroid. Calculation of the probable radiation the patient received as a child from her ^{131}I uptake tests indicated that she had received a sufficient dose to place her at an increased risk for developing thyroid carcinoma at an older age.

1307 THE EFFECT OF FRACTIONATION ON TUMOR INDUCTION AND HAIR FOLLICLE DAMAGE IN RAT SKIN. *(E.)* Burns, F. J. (New York U. Med. Ctr., N.Y.), R. E. Albert, I. P. Sinclair and P. Bennett. *Radiat Res* 53(2):235-240, 1973.

Male, albino rats were irradiated with 750 rads X-irradiation at 28 days of age. At 59 days of age, previously irradiated rats and unirradiated rats were irradiated with 0, 750, 1500, 2250 or 3000 rads. Recovery kinetics of skin tumor induction and hair follicle atrophy in irradiated rats was observed. The differences (D_2-D_1) between fractionated doses and single doses of radiation producing the same damage were calculated. Tumor incidence was a function of time after irradiation. For the 3000 and 2250 rad doses, tumors first appeared at 12-16 wk postirradiation, while for the 750 rad dose tumors appeared after 48 wk. The D_2-D_1 values for skin tumor induction and hair follicle atrophy were 650 and 600 rads, resp.

1308 CHARGE TRANSFER IN BIO-MOLECULAR DAMAGE BY LOW ENERGY PROTONS. *(E.)* Johnson, R. E. (Dept. Eng. Sci. System, U. Virginia, Charlottesville), E. T. Trevisani and J. H. Harberger. *Phys Med Biol* 18(2):287-289, 1973.

The role which charge transfer plays in the inactivation of RNase by incident low energy protons has been determined. Early studies had led to the suggestion that charge transfer, rather than quasi-elastic scattering, might be the dominant damage mechanism at proton energies below 5 keV. Results from calculating the fraction of beam particles which are ions as a function of penetration depth indicated that quasi-elastic damage most likely dominates at low proton energies, with damage via charge transfer being very small beyond about 1 nm of the target material. Therefore, the observed energy dependence in the inactivation cross-section for photons in RNase is not likely a result of fragmentation by charge transfer, the primary role of which is to control the charge state of the beam in the target.

1309 RADIATION-INDUCED CARCINOMA OF THE TRANSVERSE COLON: REPORT OF A CASE. *(E.)* Cunningham, M. P. (St. Francis Hosp., Evanston, Ill.) and R. Wilhoite. *Dis Colon Rectum* 16(2):145-148, 1973.

1310 STUDY OF THE EFFECT OF ULTRA-VIOLET LIGHT ON BIOMEMBRANES. I. ALTERATION OF OXIDATIVE PHOSPHORYLATION AND PROTEIN STATE CHANGES DURING IRRADIATION OF ISOLATED MITOCHONDRIA. *(Rus.)* Roshchupkin, D. I. (2nd Moscow Med. Inst., USSR), A. I. Marzoev and Yu. A. Vladimirov. *Biofizika* 18(1):83-88, 1973.

1311 EFFECT OF X-IRRADIATION ON MELANOSOMAL TYROSINASE ACTIVITY. *(E.)* Woert, M. H. van (Yale U. Sch. Med., New Haven, Conn.) and F. Korb. *Radiat Res* 53(3):435-443, 1973.

1312 MYELOID LEUKEMIA: DOES BLOOD LOSS INCREASE THE INCIDENCE IN X-IRRADIATED RATS? *(E.)* Maloney, M. A. (Lab. Radiobiol., U. California, San Francisco), A. R. Margulis and H. M. Patt. *Science* 180(4087):761-762, 1973.

1313 A HUMAN ENDONUCLEASE ACTIVITY FOR GAMMA-IRRADIATED DNA. *(E.)* Brent, T. P. (St. Jude Children's Res. Hosp., Memphis, Tenn.). *Biophys J* 13(4):399-401, 1973.

1314 CHONDROSARCOMA ARISING IN A CARTILAGINOUS AREA OF PREVIOUSLY IRRADIATED FIBROUS DYSPLASIA. *(E.)* Feintuch, T. A. (Baroness Erlanger Hosp., Chattanooga, Tenn.). *Cancer* 31(4):877-871, 1973.

1315 α-S-CYSTEINYL-5,6-DIHYDROTHYMINE: A POSSIBLE MODEL FOR RADIATION-INDUCED CROSS-LINKING OF DNA AND PROTEIN. *(E.)* Varghese, A. J. (Ontario Cancer Inst., Toronto, Canada). *Biochem Biophys Res Commun* 51(4):858-862, 1973.

1316 POST-IRRADIATION DARK RECOVERY OF PHOTODAMAGE TO DNA INDUCED BY FUROCOUMARINS. *(E.)* Chandra, P. (Inst. Therap. Biochem., U. Frankfurt, West Germany), R. K. Biswas, F. Dall'Acqua, S. Marciani, F. Baccichetti, D. Vedaldi and C. Rodighiero. *Biophysic* 9(2):113-119, 1973.

1317 ALTERATIONS OF THE IR SPECTRA OF DNAs AND DNPs RESULTING FROM EXPOSURE TO IONIZING AND ULTRAVIOLET RADIATION. *(Rus.)* Zhizhina, G. I. (Inst. Chem. Phys. Moscow, USSR), S. I. Bobovich and K. Ye Kruglyakova. *Dokl Acad Nauk SSSR* 209(5):1142-1145, 1973.

1318 NUMERICAL INCREASE OF GOMORI POSITIVE GLIAL CELLS IN THE BRAINS OF MICE IRRADIATED WITH LOW-ENERGY X-RAYS. *(E.)* Srebro, Z. (Med. Acad., Krakow, Poland) and H. Lach. *Acta Neuropathol* 23(1):84-89, 1973.

1319 RADIATION-INDUCED EXTRASKELETAL OSTEOSARCOMA. *(E.)* Alpert, L. I. (Methodist Hosp. Brooklyn, New York, N.Y.), I. F. Abaci and S. Werthamer. *Cancer* 31(6):1359-1363, 1973.

1320 RADIATION-INDUCED SARCOMATOUS DEGENERATION OF THE LOWER ABDOMINAL WALL: REPORT OF A CASE. *(E.)* Skinner, D. G. (U. California Sch. Med., Los Angeles). *J Urol* 108(6):879-881, 1972.

See also:

* (Rev): 1209
* (Epid-Biom): 1588

1321 TUMOR INDUCTION BY IMMUNOLOGICALLY ACTIVATED MURINE LEUKEMIA VIRUS. *(E.)*

Armstrong, M. Y. K. (Yale U. Sch. Med., New Haven, Conn.) N. H. Ruddle, M. B. Lipman and F. F. Richards. *J Exp Med* 137(5):1163-1179, 1973.

The possibility that oncogenic viruses are activated during the process of development of lymphoreticular malignancies secondary to the graft-versus-host reaction (GVHR) in hybrid mice was examined. Twelve of 29 spleen cell-free extracts (CFE) from (Balb/c x A/J)F_1 or (Balb/cJ x C57BL/6J)F_1 hybrid mice which had received an injection of parental spleen cells were tumorigenic in newborn syngeneic mice. XC plaque assays and complement fixation tests showed that several of these oncogenic CFE contained infectious murine leukemia virus (MuLV) and/or MuLV antigen. Second, third and fourth *in vitro* passage CFE were also positive for infectious MuLV and MuLV complement-fixing antigen, as well as for morphologically recognizable MuLV-like particles. These serially passed CFE remained tumorigenic in syngeneic mice. Since CFE from normal control mice matched for strain, age and sex were not oncogenic and contained no MuLV antigen or infectious virus, it was concluded that the GVHR can activate oncogenic C-type virus.

1322 SPONTANEOUS AND INDUCED PRODUCTION OF ENDOGENOUS TYPE-C RNA VIRUS FROM A CLONAL LINE OF SPONTANEOUSLY TRANSFORMED BALB/3T3. *(E.)*

Lieber, M. M. (Natl. Cancer Inst., Bethesda, Md.) and G. J. Todaro. *Int J Cancer* 11(3):616-627, 1973.

The different levels of expression of the endogenous type-C viral genome in a spontaneously transformed, tumor-producing clone (S16) derived from the BALB/3T3 murine cell line were examined. Four of nine subclones of S16 were found to continuously release high titers of endogenous virus. Five other S16 subclones were virus-nonproducers, but could be readily induced with 5'-bromodeoxyuridine (BrdU) to release large quantities of the endogenous virus. The transformed subclones were much more readily induced to produce virus compared to the parent BALB/3T3 cell line. All virus-producing subclones and one non-virus-producing subclone contained considerably larger quantities of the major group-specific antigens of the mouse type-C viruses than did the untransformed BALB/3T3 cells themselves. Kinetics of induction of type-C virus was studied in two clones that did not spontaneously release virus. Extracellular viral polymerase could be detected within 24 hr of BrdU application; it increased exponentially over a thousand-fold range during the next 48 hr and then slowly declined. It is concluded that the probability of expression of the endogenous type-C viral genome is increased in spontaneously transformed BALB/3T3 cells.

1323 STUDIES OF NEOPLASTIC MYELOMONOCYTIC CELLS IN BALB/C MICE PRODUCING INFECTIOUS C-TYPE VIRUSES. *(E.)*

Sanel, F. T. (Baltimore Cancer Res. Ctr., Md.). *Cancer Res* 33(4):671-678, 1973.

C-type viruses were identified by electron microscopy in muramidase-producing transplantable leukemic cells of BALB/c mice. Neoplastic cells contained peroxidase-positive cytoplasmic granules and consisted of atypical monocytic and granulocytic precursors, undifferentiated stem cells, and histiocytes. Viral particles were present in all tumors studied and were principally of the intracisternal A and immature C types. Exceptionally, mature C-type virions were noted in large cytoplasmic vacuoles of tumor cells, in bone marrow megakaryocytes, and in extracellular spaces. Unusual cytoplasmic structures that may represent structural precursors of viral nucleoprotein assembly were also observed in some cells. Bone marrow cells from animals treated with cell-free filtrates contained numerous cylindrical inclusions, several of which demonstrated viral budding. Complement fixation and mouse embryo cytopathogenicity tests on tumor extracts identified the viral particles as members of the murine leukemia-sarcoma group and demonstrated high titers of infectivity.

1324 CYTOPLASMIC DNA'S CONSISTING OF UNIQUE NUCLEAR SEQUENCES IN HAMSTER CELLS. A POSSIBLE ROLE IN CELL DIFFERENTIATION AND ONCOGENESIS. *(E.)*

Koch, J. (Inst. Biol. Chem. Nutrition Sci. U. Hohenheim, Stuttgart-Hohenheim, W. Germany). *FEBS Letters* 32(1):22-26, 1973.

The cytoplasmic DNA (c-DNA) species isolated from several normal and transformed hamster cell lines were studied. CsCl gradient centrifugation of c-DNA purified from ^{3}H-thymidine labeled normal or polyoma virus-transformed BHK21 cells showed peaks at 10S and 16S in addition to the mitochondrial DNA peak. Analysis of pulse labeled c-DNA from several cell lines showed a correlation between the presence of c-DNA and the transformed state. The highest amounts of c-DNA were detected in cell lines derived from tumor tissues or from whole embryos. No c-DNA was found in nonpermanent cell lines derived from differentiated tissues. An intermediate amount of c-DNA was isolated from virus-transformed cell lines. In cell lines derived from whole embryos or virus-induced tumor cells, the c-DNA was by far the most prominently labeled cytoplasmic DNA species. Results of studies of reassociation kinetics of ^{3}H-labeled c-DNA from virus-transformed cells with nuclear DNA from nontransformed cells indicated that c-DNA was derived from a specific region of the nuclear genome. The presence of specific c-DNA sequences in transformed cells is consistent with a role in oncogenesis.

1325 COMPARISON OF FREE AND 80S RNA-ASSOCIATED RNAs OF MOUSE L CELL VIRIONS. *(E.)*

Nichols, J. L. (Duke U. Med. Ctr., Durham, N.C.) and M. Waddell. *Nature [New Biol]* 243(129):236-238, 1973.

Chromatographic "fingerprinting" techniques were used to compare the nucleotide compositions of 4S and 7S RNA purified from L-cell virion (LCV) with

4S and 7S RNA prepared by heat denaturation of 80S LCV RNA (R-4S and R-7S, resp.). The fingerprints of T_1 RNase digests of 7S and R-7S RNA were identical, suggesting that they had identical nucleotide sequences. LCV 7S RNA was unrelated to "7S" RNA released by heat denaturation of L-cell 5S-RNA. Fingerprint patterns of T_1 RNase digests of LCV 4S and R-4S RNAs indicated that they were different molecules. That LCV 4S RNA was tRNA was suggested by the presence in larger amounts of 5'-terminus pGp and of several different modified nucleotides. No modified nucleotides were detected in LCV 7S RNA. Neither LCV 4S nor 7S RNA appeared to be adventitiously associated with the exterior of the virus particles since treatment of the intact LCV particles with pancreatic RNase did not increase the amounts of recoverable 4S or 7S RNA or alter their mobilities in polyacryl-amide gels, relative to untreated cells.

1326 A MAJOR GENETIC LOCUS AFFECTING RESISTANCE TO INFECTION WITH MURINE LEUKEMIA VIRUSES. III. ASSIGNMENT OF THE *Fv-1* LOCUS TO LINKAGE GROUP VIII OF THE MOUSE.

(E.) Rowe, W. P. (Natl. Inst. Allergy Infectious Dis., Bethesda, Md.), J. B. Humphrey and F. Lilly. *J Exp Med* 137(3):850-853, 1973.

C57BR x (BALB/c x AKR)F_1 mice were typed with respect to the *Fv-1* locus, the major genetic locus affecting the magnitude of response of mice to infection with murine leukemia viruses. A highly significant degree of association was found between the *Fv-1* locus and the *b* locus on linkage group VIII which determines coat color. A high degree of correlation was also found between *b* and virus expression, which was due to the linkage of *b* to the *Fv-1* locus. Preliminary results suggested that the *Fv-1* locus may be close to the *Gpd-1* locus, which is on linkage group VIII about 32 map units from the *b* locus.

1327 THE IDENTIFICATION OF THE CIRCULAR FORMS OF SV40 DNA IN WHOLE INFECTED CELL PREPARATIONS.

(E.) Harley, E. H. (University Coll. Hosp. Med. Sch., London, England) and J. S. White. *Virology* 52(2):395-407, 1973.

The supercoiled and nicked circular forms of SV40 DNA were separated from each other and from linear DNA by electrophoresis of crude cell extracts in polyacrylamide gels (PAGE). Analysis of ^{3}H-labeled DNA from infected CV-1 monkey kidney cells showed that material which sedimented as a broad band between 10-30S in neutral glycerol density gradients was separated into three sharp bands by PAGE. The degree and pattern of separation characteristically depended on the concentration of the gels used. Comparison of the migration patterns of ^{3}H-labeled DNA from infected cells with purified ^{14}C-labeled supercoiled, nicked circular and linear DNA showed that supercoiled DNA migrated faster than linear DNA in 2% gels, but slower than linear DNA in 3% gels. Nicked circular DNA migrated more slowly than either of these two peaks at either gel concentration.

1328 THE EFFECT OF DIBUTYRYL CYCLIC AMP ON TRANSFORMATION BY ONCOGENIC VIRUSES.

(E.) Smith, B. J. (Imperial Res. Fund Lab., London, England), V. Defendi and N. M. Wigglesworth. *Virology* 51(1):230-232, 1973.

The effect of dibutyryl cyclic AMP (dc-AMP) on transformation of BHK21/13 cells by SV40 and Chinese hamster embryo (CHE) cells by polyoma virus was studied. Infected cultures were treated with dc-AMP and transformation was assayed on the basis of colony morphology and the ability of trypsinized treated cells to grow in soft agar. dc-AMP (0.6mM) increased the frequency of transformation of both BHK 21/13 and CHE cells by as much as ten-fold over controls treated with virus only. The same concentrations of adenosine and 5'-AMP had no such effect. To obtain an increase in transformation, dc-AMP had to be in contact with cells from the eighth hr onward after infection, i.e., when DNA synthesis was stimulated and when integration of viral DNA into host cell DNA was occurring.

1329 ENHANCEMENT OF VIRUS-INDUCED LEUKEMIAS AND SARCOMAS IN F_1 HYBRID MICE INOCULATED WITH PARENTAL SPLEEN CELLS.

(E.) Varet, B. (St. Louis Hosp., Paris, France), A. Cannat, N. Feingold, J. Wechsler and J. P. Levy. *Cancer Res* 33(4):759-763, 1973.

The inoculation of parental spleen cells in F_1 hybrid mice favors the growth of both Graffi leukemia virus-induced lymphomas and murine sarcoma virus-induced rhabdomyosarcomas. Histocompatibility typing showed that the lymphomas originated from the hybrids and not from the parents. These observations from experiments with a known leukemogenic virus support the theory of the role of an "endogenous" leukemia virus in previously reported lymphomas arising during chronic allogeneic disease, but they suggest that the parental spleen cells play an active role in enhancing the phenomenon. Three hypotheses are proposed to explain the mechanisms of this action: 1) a modification of the target cells of the host for viral oncogenesis; 2) an increase in viral production by immunologically stimulated lymphoid cells; and 3) a depression of the immune response directed against viral antigens or virus-induced antigens of the tumor cell surface.

1330 INHERITANCE OF SUSCEPTIBILITY TO FRIEND MOUSE LEUKEMIA VIRUS. X. SEPARATE GENETIC CONTROL OF TWO VIRUSES IN FRIEND VIRUS PREPARATION.

(E.) Odaka, T. (Inst. Med. Sci., Tokyo, Japan). *Int J Cancer* 11(3):567-574, 1973.

Susceptibility of mice to viral components present in Friend leukemia virus (FLV) preparation, lymphatic leukemia virus (LLV) and spleen focus-forming virus (SFFV), was studied by separately determining end point for infection of each component. DDD, DDD-Fv^r, C57BL/6-Fv^s, C57BL/6, and BALB/c mice were used. Mice with Fv-1^n genotype were much more susceptible to LLV of N-tropic FLV than those with Fv-1^b genotype. This difference in susceptibility to LLV was shown to be controlled by a single gene locus with the recessive allele for susceptibility. Mouse strains congenic except for Fv locus showed marked differences in susceptibility to SFFV according to their Fv genotypes; mice with Fv^s genotype were much more susceptible to SFFV than those with Fv^r genotype. These results indicate that the innate susceptibility to FLV in mice is under the simultaneous control of two separate gene loci, one locus (probably Fv-1) for LLV multiplication and the other (Fv or FV-2) for proliferation of infected spleen cells (spleen focus formation and splenomegaly induction) as well as SFFV multiplication. All the data obtained support Lilly's interpretation for two-gene control.

1331 ENHANCEMENT OF ADENOVIRUS TRANSFORMATION BY PRETREATMENT OF HAMSTER CELLS WITH CARCINOGENIC POLYCYCLIC HYDROCARBONS.

(E.) Casto, B. C. (Biolabs, Inc., Northbrook, Ill.), W. J. Pieczynski and J. A. DiPaolo. *Cancer Res* 33(4):819-824, 1973.

Treatment of hamster embryo cells with benzo(*a*)pyrene, 3-methylcholanthrene, 7,12-dimethylbenz(a)anthracene, dibenz(a,h)anthracene, or dibenz(a,c)anthracene for 18 hr prior to the addition of an oncogenic adenovirus, SA7, enhanced the frequency of viral transformation. However, treatment of cells 5 hr after virus addition resulted in inhibition of transformation. The noncarcinogenic polycyclic hydrocarbons, phenanthrene, pyrene, and perylene, failed to stimulate SA7 transformation. The enhancement of transformation was directly related to chemical concentration, but treatment of cells with concentrations of chemicals above those necessary to demonstrate maximal enhancement resulted in a decrease in enhancement or complete inhibition of viral transformation. The increase in transformation frequency was not due to the selection of transformation-sensitive cells, since 6-fold increases in SA7 foci were observed in the absence of cell death. The data indicate that the stimulation of viral transformation resulted from a direct effect of the chemicals upon the cells, increasing their sensitivity to adenovirus transformation.

1332 CHROMOSOMAL CONTROL OF MALIGNANCY IN TUMORS FROM CELLS TRANSFORMED BY POLYOMA VIRUS.

(E.) Yamamoto, T. (Dept. Genetics, Weizmann Inst. Sci., Rehovoth, Israel), M. Hayashi, Z. Rabinowitz and L. Sachs. *Int J Cancer* 11(3):555-566, 1973.

The chromosome banding pattern was analyzed in clones of male and female golden hamster cells independently transformed by polyoma virus and tumors derived from these cells to identify the chromosomes with the genes of expression and suppression for malignancy. All the tumors showed either a loss of a piece of chromosome 5_3 or a gain of a piece or a whole chromosome of 5_6. An additional 7_2 chromosome in one line of transformed cells was lost in the tumors derived from this line. The results support the authors' gene balance model of malignancy and indicate that genes of expression of malignancy in these cells were located on chromosome 5_6 and that genes for suppression of malignancy were located on a piece of chromosome 5_3 and on chromosome 7_2. All transformed lines and their tumors showed a loss of a piece of one or both X chromosomes in male and female cells, resp. This loss of a piece of X chromosome was also found in non-malignant revertants from polyoma-transformed cells, so that this change in the X chromosome was not specific for malignant cells.

1333 AN ENZYMATIC FUNCTION ASSOCIATED WITH TRANSFORMATION OF FIBROBLASTS BY ONCOGENIC VIRUSES. I. CHICK EMBRYO FIBROBLAST CULTURES TRANSFORMED BY AVIAN RNA TUMOR VIRUSES.

(E.) Unkeless, J. C. (Rockefeller U., New York, N.Y.), A. Tobia, L. Ossowski, J. P. Quigley, D. B. Rifkin and E. Reich. *J Exp Med* 137(1):85-111, 1973.

Rous sarcoma virus (RSV)-transformed chick embryo fibroblasts (CEF), unlike normal, untransformed CEF, showed fibrinolysin activity, as determined by the ability to solubilize ^{125}I-fibrin *in vitro*. A similarly enhanced fibrinolytic activity was observed in CEF cells transformed by a number of different transforming viruses. However, cultures infected with nontransforming viruses, with a temperature-sensitive RSV mutant at permissive temperatures, or with various cytocidal viruses, failed to show significant fibrinolytic activity. The appearance of fibrinolytic activity in CEF cells infected with the RSV mutant following a shift to permissive temperatures was dependent on RNA and protein synthesis, but not on DNA synthesis. Of all sera tested, those of chicken and monkey origin were most active in promoting fibrinolysis by transformed cells. Other sera, including fetal bovine, adult bovine, turkey, dog and guinea pig, were considerably less effective and some (e.g., fetal bovine) contained a fibrinolysin inhibitor. Normal CEF did not secrete fibrinolysin inhibitor nor was one found in the sera from normal birds. Sera from sarcoma-bearing chickens did, however, contain an inhibitor of fibrinolysin.

1334 RNA IN MAMMALIAN SARCOMA VIRUS TRANSFORMED NONPRODUCER CELLS HOMOLOGOUS TO MURINE LEUKEMIA VIRUS RNA.

(E.) Benveniste, R. E. (Natl. Cancer Inst., Bethesda, Md.) and E. M. Scolnick. *Virol* 51(2):370-382, 1973.

Various normal and Kirsten and Moloney sarcoma virus transformed, gs antigen negative nonproducer clones of mouse 3T3 and rat kidney cells were studied by nucleic acid hybridization techniques for the presence of RNA homologous to murine leukemia virus (MuLV) RNA. Total cellular RNA extracted from the transformed lines was annealed to ^{3}H-DNA synthesized in an *in vitro* reverse transcriptase reaction from purified MuLV RNA. Unhybridized nucleic acid was hydrolyzed with mung bean or *A. oryzae* S-1 nuclease. Results showed that RNA from transformed nonproducer and producer cell lines hybridized to approximately 22-40% and 65% of the total input ^{3}H-DNA, resp. Normal rat cell RNA showed no significant hybridization. Analogous results were obtained with polysomal RNA from sarcoma virus transformed mouse cell lines. Enzymatic digestion with nuclease proved to be more sensitive than the conventional Cs_2SO_4 centrifugation in detecting RNA-DNA hybrid formation.

1335 INTERACTION OF GENES CONTROLLING RESISTANCE TO RSV(RAV-0).

(E.) Crittenden, L. B. (Animal Physiol. Genetics Inst., Beltsville, Md.) E. J. Wendel and J. V. Motta. *Virology* 52(2): 373-384, 1973.

Four inbred chicken lines, 6, 7, 15 and 100, and some of their F_1 and backcross chicken embryos were studied for resistance to subgroups A, B, C, and E avian sarcoma viruses, and for the occurrence of the R_1 erythrocyte and group-specific (gs) antigens in primary cell cultures. Each of the four lines investigated differs with respect to the tumor virus (*tv*)*a* and (*tv*)*b* loci and the R_1 and gs antigens. Embryos from the same matings were assayed on the chorioallantoic membrane for resistance to subgroup B and E viruses, by agglutination tests for R_1 antigen, and by complement fixation tests for gs antigen. The results suggested that: (1) RSV(RAV-0) cannot readily infect subgroup B virus resistant cells; (2) the dominant gene, I^e, probably exists in line 6 and is associated with the occurrence of the gs antigen; (3) the occurrence of the R_1 erythrocyte antigen is associated with enhancement of susceptibility to RSV(RAV-0) in the presence of the dominant inhibitor (I^e); (4) the R_1 antigen is associated with the occurrence of gs antigen in the absence of I^e, (5) genes exist which cause resistance to RSV(RAV-0) on the chorioallantoic membrane, but not in culture. These results imply that the tumor virus *b* locus controls the existence of a receptor for subgroup B and E viruses, but that other genes, associated with the occurrence of viral antigens (gs and R_1), interact to inhibit or enhance susceptibility to RSV(RAV-0).

1336 STUDIES OF THE MECHANISM OF INDUCTION OF INFECTIOUS MURINE LEUKEMIA VIRUS FROM AKR MOUSE EMBRYO CELL LINES BY 5-IODODEOXYURIDINE AND 5-BROMODEOXYURIDINE.

(E.) Teich, N. (Natl. Inst. Allergy Infectious Dis., Bethesda, Md.) D. R. Lowry, J. W. Hartley and W. P. Rowe. *Virology* 51(1):163-173, 1973.

The mechanism by which 5-iododeoxyuridine (IUdR) and 5-bromodeoxyuridine (BUdR) induce release of mouse leukemia virus (MuLV) from three nonproducer AKR mouse cell clones was studied. Although 5-iododeoxycytidine, 3-methylcholanthrene, 7,12-dimethylbenzanthracene and mitomycin C also induced MuLV release, BUdR and IUdR were most effective. The efficiency of virus activation of BUdR and IUdR increased with increasing drug concentration and duration of treatment to a maximum and then declined. MuLV induction, as measured by the XC plaque assay, was inhibited by cytosine arabinoside, thymidine and by incubation in serum-depleted medium. Virus induction was stimulated by fluorodeoxyuridine and by visible and UV light irradiation of analogue-treated cells. It thus appeared that BUdR and IUdR incorporation into cellular DNA was necessary for virus activation.

1337 SCANNING ELECTRON MICROSCOPY OF CELLS INFECTED WITH A MURINE LEUKEMIA VIRUS.

(E.) DeHarven, E. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), N. Lampen, T. Sato and C. Friend. *Virology* 51(1):240-243, 1973.

Scanning electron microscopy (SEM) was used to study the surfaces of murine Friend erythroleukemia cells. Cells were collected from suspension cultures on silver membranes, fixed in glutaraldehyde and osmium tetroxide, shadowed with carbon and gold, and examined. Cells viewed at 10,000X showed numerous, small "knobs" on their membrane surface. Treatment of leukemic cells with either bromodeoxyuridine or dimethylformamide, two substances known to enhance the release of RNA tumor viruses, resulted in a three- to four-fold increase in the number of "knobs." Transmission electron microscopy of similarly treated cultures showed a parallel increase in the number of budding viruses. It was thus concluded that the "knobs" seen with SEM represented budding viruses. Budding, which was randomly distributed over the cell surface, was observed in over 95% of the cells.

1338 ISOLATION OF LEUKOSIS-TYPE VIRUS FROM PHEASANT EMBRYO CELLS: POSSIBLE PRESENCE OF VIRAL GENES IN CELLS.

(E.) Hanafusa, T. (Public Hlth. Res. Inst. City New York, N.Y.) and H. Hanafusa. *Virology* 51(1):247-251, 1973.

A new virus (RAV-61) was recovered from pheasant embryo cells after infection with the Bryan strain of Rous sarcoma virus (B-RSV) in the presence of UV-irradiated Sendai virus. RAV-61 purified by two limiting dilution passages in gs^-h^- chicken cells was used to determine its subgroup specificity by interference tests. RAV-61 induced no interference with any virus tested which belonged to subgroups A through D, but it did interfere with RSV activated by itself and with RSV(RAV-60), a subgroup E virus. The host range of purified RAV-61 was the same as RSV obtained directly from transformed pheasant cells. Based on these results, RAV-61 can be classified separately in a new subgroup, F, among avian leukoviruses. Like other avian leukoviruses, RAV-61 banded in sucrose gradients at 1.16g/cm and its RNA sedimented at 60-70 S. RAV-61 also contained RNA-dependent DNA polymerase activity. In addition, cells derived from 14 of 44 pheasant embryos spontaneously released a virus-like agent capable of acting as a helper for B-RSV which resembled, in some respects, RAV-0, which has been found in certain flocks of chickens.

1339 THE LYMPHOCYTE RESPONSE TO PRIMARY MOLONEY SARCOMA VIRUS TUMORS IN BALB/c MICE: DEFINITION OF THE ACTIVE SUBPOPULATIONS AT DIFFERENT TIMES AFTER INFECTION.

(E.) Lamon, E. W. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), H. Wigzell, E. Klein, B. Andersson and H. M. Skurzak. *J Exp Med* 137(6):1472-1493, 1973.

Lymphocyte populations from Moloney sarcoma virus (MSV)-infected male and female adult Balb/c mice were examined in an *in vitro* microcytotoxicity test for activity against three Moloney leukemia virus (MLV) antigen-positive target cell lines every five days for 30 days after infection. Antigen-specific activity was present in unfractionated lymphocytes from animals which either had not yet developed palpable tumors or had developed tumors which had since regressed. Macrophage-free T-cell and B-cell subpopulations from MSV-infected mice were also examined. T-cells were active against target cells just before tumor development and just after regression; however, activity decreased considerably thereafter. The B-cell subpopulation was also active before tumor development and after regression, but, unlike T-cells, anti-target cell activity continued to rise after regression to a maximum at 30 days. Neither T- nor B-cells were active when tumor size was maximum. The activity was MLV antigen specific.

1340 PREVENTION OF CELL-TO-CELL SPREAD OF HERPES SIMPLEX VIRUS BY LEUKOCYTES.

(E.) D. L. Lodmell (Natl. Inst. Dental Res., Bethesda, Md.), A. Niwa, K. Hayashi and A. L. Notkins. *J Exp Med* 137(3):706-720, 1973.

The factors involved in preventing direct cell-to-cell spread of herpes simplex virus (HSV) type 1 were studied. HSV-infected primary rabbit kidney cells were destroyed when placed in medium containing anti-HSV antibody plus complement. Although infected cells were destroyed, the spread of infection within the monolayer was not prevented. Virus spread from infected to uninfected cells before the infected cells became susceptible to immunological destruction. However, when infected cultures were incubated with leukocytes from New Zealand white rabbits, the spread of virus was inhibited due to nonspecific damage of both infected and uninfected cells and possibly also to breakage of intracellular bridges. Following removal of the leukocytes from these cultures, plaques once again appeared. Plaque development was prevented when anti-HSV antibody and complement were added to the monolayers before the leukocytes were removed, suggesting that both antibody and leukocytes were required to "cure" the HSV infection.

1341 THE INFLUENCE OF THE PARTNER CELL ON THE PRODUCTION OF L VIRUS AND THE EXPRESSION OF VIRAL SURFACE ANTIGEN IN HYBRID CELLS.

(E.) Fenyö, E. M. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), G. Grunder, F. Wiener, E. Klein, G. Klein and H. Harris. *J Exp Med* 137(5):1240-1255, 1973.

C-type virus particles produced by the A9 and A9HT sublines of mouse L cells could infect C3H (N type), but not C57BL (B type), mouse embryo fibroblast cultures as determined by the XC-plaque assay. It was thus concluded that L cell virus is N tropic. Sendai virus-induced fusion of A9 or A9HT cells with cells from various mouse tumor and normal lines resulted in introduction by the A9 component of the of the ability of the cell hybrid to produce Moloney-type surface antigen and to release infectious virus. Virus production, measured by antigen induction on JLS-V9 cells, was suppressed in those hybrids in which the partner cell had a genotype determining low infectibility with that particular virus (i.e., a B-type cell). It thus appeared as if the *Fv-1* locus, the major locus affecting resistance to infection with leukemia viruses, was also regulating virus production in the somatic cell hybrids. This genetic locus did not seem to govern the expression of all virus-related functions since virus-determined membrane antigen was detected in many of the N x B hybrids in which production of infectious virus was suppressed.

1342 AN ENZYMATIC FUNCTION ASSOCIATED WITH TRANSFORMATION OF FIBROBLASTS BY ONCOGENIC VIRUSES. II. MAMMALIAN FIBROBLAST CULTURES TRANSFORMED BY DNA AND RNA TUMOR VIRUSES.

(E.) Ossowski, L. (Rockefeller U. New York, N.Y.), J. C. Unkeless, A. Tobia, J. P. Quigley, D. B. Rifkin and E. Reich. *J Exp Med* 137(1):112-126, 1973.

SV40-transformed hamster embryo, SV40- or mouse sarcoma virus-transformed mouse embryo and mouse 3T3 cells, and avian B-77 sarcoma virus transformed-rat embryo cells were tested for fibrinolysin activity based on their ability to solubilize ^{125}I-fibrin *in vitro*. As previously reported for virus-transformed chick fibroblasts, the transformed mammalian cells exhibited a greatly increased enzyme activity compared with nontransformed cells. The factor produced by the transformed cells required the presence of a second factor in the serum of the incubation medium for fibrinolytic activity to occur. Dog, hamster and monkey sera were most effective in producing fibrinolysin activity by transformed hamster cells, whereas dog, monkey and human sera were most effective for transformed mouse and rat cells. Fetal bovine and chicken serum were inhibitory to both hamster and mouse fibrinolysin activity. Increased fibrinolysin activity was correlated with morphological transformation in all four transformed mammalian cell lines.

1343 EPSTEIN-BARR VIRUS. HETEROPHILE RESPONSES IN SQUIRREL MONKEYS INOCULATED WITH VIRUS-TRANSFORMED AUTOLOGOUS LEUKOCYTES.

(E.) Shope, T. (Yale U. Sch. Med., New Haven, Conn.) and G. Miller. *J Exp Med* 137(1):140-147, 1973.

Three female squirrel monkeys shown to be Epstein-Barr virus (EBV) negative received five or six i.v. injections of autologous EBV-transformed lymphoblasts over a 30-day period. Seven months later, the sensitized animals were challenged with a single injection of live autologous EBV-transformed cells, ultrafiltrates of EBV-transformed cells, or ghosts of the EBV-transformed cells prepared by freezing and thawing. All three animals developed heterophile agglutinins and EBV-specific antibodies following sensitization. None developed palpable tumors or hematologic disorders. The heterophile agglutinins disappeared 2-8 wk following the last EBV-transformed lymphocyte inoculation, and EBV-specific antibodies detectable by complement fixation or immunofluorescence disappeared after 4-18 wk. Heterophile agglutination and EBV-specific antigen production were again stimulated by challenge with EBV-transformed whole cells and cell ghosts, but not by the ultrafiltrates.

1344 *IN VITRO* CULTURE OF CLONED HAMSTER MELANOMA CELLS CONTAINING R-TYPE VIRUS. *(E.)*

Epstein, W. L. (U. California, Sch. Med., San Francisco) and K. Fukuyama. *Cancer Res* 33(4):825-831, 1973.

A transplantable melanoma of golden Syrian hamsters was grown in tissue culture and four stable cell lines, 3 pigmented and 1 with little or nor pigment, were obtained by cloning techniques. These four cell lines were examined by light and electron microscopy and found to differ from each other in growth patterns as well as in production of radial-type virus and synthesis and degradation of melanosomes. Cells of one line produced large amounts of virus and little or no pigmented melanosomes, which were degraded in the early stages of formation. Another cell line produced virtually no virus but melanosome formation remained intact. The other two cell lines produced varying amounts of both virus and melanosome and degraded pigment in "melanosome complexes." The findings demonstrate the fact that a seemingly homogenous tumor can yield several cell lines of differing growth rates, patterns, and degrees of differentiation. Acid phosphatase activity occurred in all cell lines but ultrastructural evidence of melanin degradation varied in the different lines. It is suggested that isolated cell lines may provide a model to investigate the relationship of radial-type viruses to differentiation in malignant cells.

1345 THERMOSENSITIVE EVENTS IN ADENOVIRUS TRANSFORMED CELLS. I. RESISTANCE TO HERPES SIMPLEX VIRUS. *(E.)*

Sprecher-Goldberger, S. (Pasteur Inst., Brabant, Belgium), N. Lefebvre, J. DeBruyne, R. Pochet and L. Thiry. *Arch Gesamte Virusforsch* 40(3-4):236-247, 1973.

Growth of herpes simplex viruses type 1 and 2 was inhibited in three hamster cell lines transformed by adenovirus type 12 but the viruses grew to the same high titers in two non-transformed hamster lines as well as in one of these lines transformed by polyomavirus. Sindbis virus multiplied normally in the adenovirus transformed cells. In these cells, inhibition of herpesvirus growth was much greater at 37 than at 35 C. In trying to determine a thermosensitive event in the transformed cell line NILTr$_1$ not infected with herpesvirus, it was found that adenylate cyclase activity was not decreased at 37 C, that the turn over of nucleic acid and protein synthesis was not thermosensitive, but that the incorporation of (14C) glucosamine was much greater at 35 than at 37 C. This thermosensitive event was however probably distinct from that responsible for inhibition of herpesvirus growth since in NILTr$_1$ cells, as in the non-transformed NIL cells, herpes simplex virus type 2 induced an increased glucosamine incorporation, even at 39 C. In contrast, the syntheses of herpesvirus DNA and proteins were inhibited at 37 C in NILTr$_1$ cells, while they were only suppressed at 39 C in the non-transformed cell line. Inhibition of herpesvirus replication by adenovirus genome may be related to one of the following situations: 1) competition between adenoviruses and herpesviruses for some cellular metabolic pathway, occurring whether or not the adenovirus is oncogenic; 2) repression by adenovirus of some cellular function which is necessary for the synthesis of herpesvirus DNA; or 3) the replacement of some host gene by an adenovirus gene.

1346 EVOLUTIONARY SIGNIFICANCE OF PERSISTENCE OF LATENT ONCOGENIC VIRUS INFORMATION IN VERTEBRATES. *(E.)*

Hirschhorn, K. (Mount Sinai Sch. Med., City U. New York, N.Y.), N. Gabelman, P. M. Price and S. Waxman. *Lancet* 1(7813):1158-1159, 1973.

A hypothesis which attempts to correlate data resulting from virological and immunological approaches to cancer research is proposed. It is suggested that oncogenic-virus information is a stable integral part of normal DNA in most vertebrate species, including man, and that this information normally benefits the species, rather than doing harm, as is generally suggested for oncogenic viruses. Transcription of RNA tumor viruses by derepression of such genetic information by carcinogenic agents leads to new antigenic information on the surfaces of transformed cells. These new antigens lead to recognition and rejection of the abnormal cell by the host's cellular immune system. It is postulated therefore that viruses, rather than causing cancer, may help to prevent it.

1347 *IN UTERO* TUMOR INDUCTION BY MURINE SARCOMA VIRUS (MOLONEY) IN THE RAT. I. BIOLOGICAL CHARACTERISTICS. *(E.)*

Vandeputte, M. (Rega Inst., U. Leuven, Belgium), H. Sobis, A. Billiau, B. Van De Maele and R. Leyten. *Int J Cancer* 11(3):536-542, 1973.

The pregnant rat uterus was used as the site for virus inoculation to examine whether its particular immunological status would allow tumor development and to characterize the biology and morphology of any tumors obtained by this procedure. Female R rats mated with an R male and inoculated *in utero*, after fetectomy, with murine sarcoma virus (MSV) (Moloney), developed tumors. These tumors originated in the uterus and were of fetal origin. I.v. or i.p. injection of the virus induced similar tumors. Infectious virus could not be isolated from the tumor cells kept as transplantable lines or cultured *in vitro*. However, the presence of the MSV genome could be demonstrated by a direct rescue test. In non-pregnant rats treated in the same way no neoplasms were recorded. Three explanations for these results are discussed. First, the possibility is considered that the fetal membranes, and more particularly the yolk sac, are extremely sensitive to neoplastic transformation perhaps due to the multipotentiality of the cells in the yolk sac and their growth potential. A second mechanism which could permit outgrowth of fetal tumors in adult female rats would be the non-immunogenicity of these tumors. The third explanation is based on the particular immunological mother-fetus relationship existing during pregnancy and confined to the pregnant uterus. Whether this intra-uterine immunologic protection is based upon the existence of a trophoblast quarantining layer or upon an afferent or efferent deficient immunologic mechanism is not yet clear.

1348 *IN UTERO* TUMOR INDUCTION BY MURINE SARCOMA VIRUS (MOLONEY) IN THE RAT. II. HISTOLOGICAL AND ULTRASTRUCTURAL CHARACTERISTICS. *(E.)* Sobis, H. (Rega Inst., U. Leuven, Belgium) and M. Vandeputte. *Int J Cancer* 11(3):543-554, 1973.

The histological and ultrastructural characteristics of *in utero* murine sarcoma virus (MSV)-induced tumors in pregnant R rats was examined. On histological examination the twelve MSV-induced tumors in the uterus observed in the study proved to be of fetal origin. Ten of the 12 tumors had the histological and ultrastructural characteristics of a yolk-sac tumor. The two other tumors examined had a different morphological structure. One was classified as a teratoma with embryonal carcinoma, the other as a carcinosarcoma. In the primary neoplasms virus particles were found. Their ultrastructure was similar to that of MSV. The morphology of the metastases was similar to that of the primary neoplasms and did not change significantly upon transplantation or after *in vitro* culturing. There was little or no tendency to differentiate into other tissues. In contrast, two tumors, one a teratoma with embryonal carcinoma and the other probably a carcinosarcoma, with a variety of different cell types in the primary neoplasms were observed. The both cases, areas of embryonal carcinoma and of yolk-sac tumor were observed in the metastases, indicating that the primary tumor contained multipotential cells capable of differentiation. The experiments also show that murine sarcoma virus is capable of provoking the induction of tumors having an epithelial structure.

1349 BURKITT'S LYMPHOMA AND *HERPESVIRUS SAIMIRI* LYMPHOMA: COMPARATIVE ASPECTS. *(E.)* Epstein, M. A. (U. Bristol Med. Sch., England). *Bol Of Sanit Panam* 7(1):58-63, 1973.

Burkitt's lymphoma is a malignant tumor of children. Epidemiologic and numerous *in vitro* studies have implicated Epstein-Barr virus (EBV), a herpes virus, as at least one of its etiologic agents. However, the fact that one is dealing with a human tumor has rendered impossible the types of experiments necessary to establish beyond a doubt the role that EBV probably plays in the pathogenesis of Burkitt's lymphoma. The recent discovery of the ability to induce a similar type of lymphoma in marmosets and owl monkeys with *Herpesvirus saimiri* has opened up the possibility for further study of the mechanism of viral oncogenesis of Burkitt's lymphoma by analogy. There are, however, important differences between EBV and *H. saimiri* and the tumors with which they are associated. Unlike Burkitt's lymphoma, *H. saimiri* lymphoma does not appear to occur in its natural host. *H. saimiri* lymphoma consists of marked, widespread reticulum cell invasion of many organs, whereas, Burkitt's lymphoma does not generally show widespread involvement. Unlike EBV, *H. saimiri* is cytocidal in tissue culture and does not produce cellular transformation. Despite these differences, *H. saimiri* will probably be of use in studying malignant human disease of the lymphoreticular type.

1350 ULTRAVIOLET INACTIVATION OF MURINE LEUKEMIA VIRUS AND ITS ASSAY IN PERMISSIVE AND NON-PERMISSIVE CELLS. *(E.)* Yoshikura, H. (Inst. Med. Sci., U. Tokyo, Japan). *Int J Cancer* 11(3):739-746, 1973.

The previously reported different titration patterns of murine leukemia viruses (MuLV) in permissive and non-permissive cells were examined and confirmed. Titration kinetics was one-hit in the permissive host and two-hit in the restrictive host. The following phenomena were also observed: (1) ultraviolet (UV)-inactivation curves were nearly the same, regardless of whether the virus was titrated in permissive or restrictive cells; (2) infection by MuLV of its restrictive host was enhanced by the UV-inactivated homologous MuLV, (3) UV-inactivated NB-tropic virus propagated in N-type cells enhanced plaque formation of N-tropic virus in B-type cells and not that of B-tropic virus in N-type cells. UV-inactivated NB-tropic virus propagated in B-type cells enhanced plaque formation of B-tropic virus in N-type cells, but not that of N-tropic virus in B-type cells.

1351 A RAPID DESTRUCTIVE EFFECT OF THE SCHMIDT-RUPPIN STRAIN OF ROUS SARCOMA VIRUS ON TRANSFORMED RAT CELL LINES. *(E.)* Reinisch, C. L. (Johns Hopkins U., Sch. Hygiene, Publ. Hlth., Baltimore, Md.) and F. B. Bang. *Int J Cancer* 11(3):774-779, 1973.

The *in vitro* effect of the Schmidt-Ruppin strain of Rous sarcoma virus (RSV-SR) on XC cells and on other transformed cell lines was examined. A cytopathic effect was induced by RSV-SR in 5 of 9 rat tumor cell lines tested. The cells showed irreversible cytopathic change as early as 24-48 hr after infection. Other leukemogenic and tumorigenic viral agents, such as RSV-Bryan, RAVs 1,2 and 50, SV40 and GAL virus were also tested on XC cells. Only RSV-SR induced the rapid Rous cytopathic effect. Virus was recovered 8 days after inoculation of the tumor cell lines XC and M 41. The effect was neutralized by specific anti-RSV-SR antiserum. Correlation between oncogenicity of the rat tumor lines in the syngeneic host and their susceptibility to RSV-SR is incomplete.

1352 PREFERRED SITE FOR INITIATION OF RNA TRANSCRIPTION BY *ESCHERICHIA COLI* RNA

POLYMERASE WITHIN THE SIMIAN VIRUS 40 DNA SEGMENT OF THE NONDEFECTIVE ADENOVIRUS-SIMIAN VIRUS 40 HYBRID VIRUSES Ad2+ND$_1$ AND Ad2+ND$_3$. *(E.)* Zain, B. S. (Yale U. Sch. Med., New Haven, Conn.), R. Dhar, S. M. Weissman, P. Lebowitz and A. M. Lewis, Jr. *J Virol* 11(5):682-693, 1973.

The transcription of purified SV40 DNA by *E. coli* RNA polymerase has been studied *in vitro*. ^{32}P-labeled SV40 complementary RNA synthesized *in vitro* from SV40 DNA by *E. coli* polymerase at 18 or 24 C was annealed to purified DNA from either Ad2+ND$_1$ or Ad2+ND$_3$, two nondefective adenovirus-SV40 hybrids. Following removal of nonannealed RNA by RNase treatment, DNA bound ^{32}P-labeled RNA was eluted and treated with T1 or pancreatic RNase and the resulting oligonucleotides were analyzed by two-dimensional electrophoresis and chromatography. Comparison of the oligonucleotide patterns obtained from short incubations showed that all the principal products obtained were derived from a segment of DNA common to both Ad2+ND$_1$ and Ad$_2$ND$_3$. This same set of oligonucleotides was also obtained from RNA digested and mapped without prior hybridization. These results indicated that, under these conditions of restricted RNA transcription, there was a preferred site for initiation of RNA synthesis on SV40 DNA which lay within the segment of SV40 DNA common to both Ad2+ND$_1$ and Ad2+ND$_3$. The initiation site for transcription lay at least 100 nucleotides from the 5'phosphorylated end of the transcribed strand of the SV40 region of Ad2+ND$_1$ and Ad2+ND$_3$ DNA.

1353 INTERFERENCE WITH POLYOMA VIRUS REPLICATION BY HOMOLOGOUS INCOMPLETE VIRUS. *(E.)* Hakura, A. (Res. Inst. Microbial Dis., Osaka U., Japan) and J. Kamahora. *Biken J* 15(4):207-213, 1973.

Unlike normal control polyoma (py) virus, py virus grown in SVT cells (SH-PV) derived from an SV40-induced hamster tumor was unable to replicate when inoculated onto monolayer cultures of A31 cells derived from Balb/c mice. The inability of SH-PV to replicate was due to the presence in the stock of defective virus which interfered with normal virus also present in the stock. The fact that these defective particles lost their ability to interfere with normal virus replication when UV-irradiated indicated that functional DNA was necessary for their action. This interference was observed when defective particles were added to cultures up to nine hr after infection with normal, complete virus. Interference of normal py virus replication in A31 cells by SH-PV was not due to a lethal effect of the SH-PV stock on the host cells before multiplication of the coinfected complete virus.

1354 IMMUNOLOGICAL CONTROL OF POLYOMA VIRUS ONCOGENESIS IN MICE. *(E.)* Gaugas, J. M. (Natl. Inst. Med. Res., Mill Hill, England), A. C. Allison, F. C. Chesterman, R. J. W. Rees and M. S. Hirsch. *Br J Cancer* 27(1):10-17, 1973.

Adult female, thymectomized CBA mice treated with antilymphocytic globulin (ALG) and inoculated with human leprosy organisms were accidentally infected with polyoma virus and all developed tumors. After cessation of ALG administration, some animals were given spleen cells from syngeneic donors immunized with polyoma virus; none developed tumors. Similar results were obtained in mice deliberately infected with polyoma virus but not with leprosy organisms. Passive transfer of antibody before but not after virus inoculation prevented tumor formation in immunosuppressed recipients. Virus infection in thymectomized, lethally irradiated and bone marrow reconstituted mice resulted in only a very low incidence of tumors. These results emphasize the role of immunological surveillance in preventing polyoma tumor formation under natural conditions.

1355 ACTIVATION OF ENDOGENOUS C-TYPE ONCORNAVIRUS BY 5-BROMODEOXYURIDINE IN CULTURED EMBRYONIC RAT FIBROBLASTS. *(E.)* Grollé, P. F. (Radiobiol. Inst. TNO, Rijswijk, The Netherlands), P. Bentvelzen and M. J. Van Noord. *Biomed* 19(4):148-151, 1973.

Cultured non-producer embryonic fibroblasts of the inbred WAG/Rij rat strain could be induced to release C-type virus particles by growth for 24 hr in medium containing 0.2 mg/ml 5-bromodeoxyuridine followed by growth for three days in medium containing 2% dimethylsulphoxide. Immunodiffusion studies indicated that the C-type virus contained an antigen in common with the murine leukemia viruses; however, immunofluorescence studies suggested that there was a sharp serological distinction between the retrieved rat virus and the murine leukemia viruses. The retrieved rat virus did not show focus-forming activity in cultures of embryonic rat fibroblasts, and inoculation of the virus into newborn WAG/Rij rats and Balb/c mice produced no signs of disease.

1356 FRIEND LEUKEMIA VIRUS (FLV) ELIMINATION IN FECES OF FLIES AND FLEAS. *(E.)* Rehácek, J. (Inst. Virol., Slovak Acad. Sci., Bratislava) R. G. Fischer and D. H. Luecke. *Acta Virol* 17(2):167, 1973.

The elimination of Friend leukemia virus (FLV) in feces of infected stable flies and cat fleas was studied. In 2 of 12 experiments, cat fleas which were allowed to feed on virus-infected mice eliminated FLV in their feces for up to one day after their meal. Stable flies did not eliminate FLV in their feces even though they ingested large amounts of virus. The potential for FLV survival demonstrated in flea feces suggests that transmission via insect feces is a possible factor in the natural spread of Friend leukemia.

1357 STUDIED ON THE REPLICATION OF POLYOMA DNA: PHYSICOCHEMICAL PROPERTIES OF VIRAL DNA SYNTHESIZED WHEN PROTEIN SYNTHESIS IS INHIBITED. *(E.)* Branton, P. E. (Ontario Cancer Inst., Toronto, Canada) and R. Sheinin. *Can J Biochem* 51(3):305-317, 1973.

Physicochemical, electron microscopic and enzymatic studies were conducted to analyze the structure of the polyoma (py) DNA synthesized under conditions in which *de novo* protein synthesis was inhibited late during infection, and in which (as previously shown) the maturation of progeny viral DNA to yield superhelical, fully covalently closed form I DNA was blocked. Neutral and alkaline sucrose gradient centrifugation of 16-21S py DNA synthesized during infection of mouse embryo cell cultures in the presence of cycloheximide showed that most of this newly synthesized DNA behaved as intermediate form II DNA rather than as form I, thus confirming earlier results. Comparison by CsCl equilibrium density centrifugation of normal ^{14}C-labeled marker with ^{3}H-labeled 16-20S py DNA synthesized during protein inhibition suggested that the 16-20S py DNA did not vary significantly from the normal py DNA in its base composition or in its association with protein. Electron micrographs showed that 50% of the py DNA synthesized in the presence of cycloheximide consisted of double-stranded molecules with linear "tails" of double-stranded DNA of varying lengths.

1358 PROPERTIES OF AN SV40 TRANSFORMED AFRICAN GREEN MONKEY KIDNEY (BSC-1) CELL LINE: INTERACTIONS BETWEEN SV40 AND CELLULAR DNA METABOLISM.

(E.) Mousset, S. (Inst. Res. Sci. Cancer, Villejuif, France) and D. Blangy. *Virology* 52(2): 385-394, 1973.

Normal and SV40-transformed African green monkey (BSC-1) cell lines were used to study the interactions between SV40 and cellular DNA synthesis following initial SV40 infection and superinfection. $CsCl_2$ equilibrium density centrifugation studies of ^{3}H-thymidine-labeled DNA from infected BSC-1 cells showed the presence of closed circular and nicked viral DNA, as well as a low molecular wt linear double-stranded DNA shown by DNA-DNA hybridization studies to be of cellular origin. No low molecular wt cellular DNA was present in uninfected cells. It did, however, appear in newly infected BSC-1 cells in a time- and multiplicity-dependent fashion which paralleled that observed for viral and total cellular DNA synthesis. ^{3}H-Thymidine incorporation into the low molecular wt DNA occurred in an SV40-infected BSC-1 clone (BSC/SV) and did not increase above this level following superinfection, indicating that production of this DNA species was unaffected by superinfection. Whereas infection of BSC-1 cells released the inhibition of DNA replication caused by 5-bromodeoxyuridine (BUdR), no additional increase in replication was found in superinfected cells. These results suggested that no additional control of DNA metabolism was supplied by superinfecting virus and that BUdR could not block DNA replication in transformed cells.

1359 STUDIES ON TUMORIGENICITY OF RAT BONE TUMOR VIRUS (SD-MSV-M) IN MICE, RATS AND HAMSTERS.

(E.) Dmochowski, L. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.), J. L. East, J. M. Bowen, M. L. Lewis and T. Shigematsu. *Tex Rep Biol Med* 30(4):301-312, 1972.

The tumorigenicity and transforming ability of the Moloney strain of murine sarcoma virus (SD-MSV-M) which produces bone tumors was studied *in vivo* and *in vitro*. SD-MSV-M previously adapted for tumorigenicity in New Zealand (NZ) black rats was tumorigenic and produced effusions in hamsters, mice and rats following i.p. injection. Hamster-adapted SD-MSV-M was tumorigenic only when injected into hamsters. Cell-free material from SD-MSV-M transformed mouse 3T3 fibroblast cultures was also tumorigenic in hamsters, mice and rats. Electron micrographs of these tumors revealed mature and budding C-type virus particles. Cell-free culture medium from transformed 3T3 cultures or from rat bone tumors or effusions of infected mice induced foci in mouse embryo (ME) and rat embryo (RE) but not in hamster embryo (HE) cultures. Virus from transformed ME and RE cultures was tumorigenic in mice and rats, but not in hamsters. Cell-free extracts of hamster bone tumors and from hamster bone tumor cultures were not tumorigenic in rats or mice; however, they did produce focus formation in HE, ME, RE and 3T3 cell cultures. Medium from the transformed HE, RE and 3T3 cultures did not regain the capacity for inducing tumors in rats or mice, but were tumorigenic in hamsters. Medium from the ME cells transformed by the hamster-adapted virus did, however, acquire the ability to induce tumors in mice, and, in fact, lost tumorigenicity in hamsters.

1360 ENHANCEMENT OF VIRAL ONCOGENICITY BY THE PRIOR ADMINISTRATION OF DIMETHYL SULFOXIDE.

(E.) Warren, J. (Leo Goodwin Inst. Cancer Res., Fort Lauderdale, Fla.), M. R. Sacksteder, B. M. Ellis and R. D. Schwartz. *Cancer Res* 33(3):618-622, 1973.

The ability of dimethyl sulfoxide (DMSO), given prior to infection, to modify the response of animal tissues to two oncogenic viruses in quail and mice was examined. The i.m. injection of DMSO prior to the infection of quail with Rous sarcoma virus strain 0 and of mice with Moloney sarcoma virus resulted in increased virus titers, decreased incubation time to tumors and, in the case of Moloney sarcoma virus, decreased spontaneous regressions. DMSO administered at the time of or after infection was without effect. This compound also increased focus formation by strain 0-Rous sarcoma virus in monolayer cultures of quail embryo fibroblasts. The basis for the promoting action of DMSO is unknown, but increased cell permeation could be involved. It is suggested that large-scale resumption of clinical use of DMSO in man should continue to be discouraged until its potential for enhancing oncogenicity is further understood.

1361 EFFECT OF HUMAN MILK ON THE MOUSE MAMMARY TUMOR VIRUS.

(E.) Sarkar, N. H. (Inst. Med. Res., Camden, N.J.), J. Charney, A. S. Dion and D. H. Moore. *Cancer Res* 33(3):626-629, 1973.

The extent of mouse mammary tumor virus (MuMTV) virion degradation, the loss of infectivity, and the reduction in RNA-dependent DNA polymerase caused by some human milks was examined. MuMTV when mixed with whole, skimmed, or cream fractions of some human milks and incubated at 37 C for 18 hr resulted in degradation of the particle morphology and decreased infectivity and reverse transcriptase activity of the virions. The cream fraction was more effective in the physical, biochemical, and biological degradation of the virions than the whole or skimmed milk, but the nature of the degrading component(s) has not been determined. It segregates with the fat fraction of the human milk; either a chemical, enzymatic, or physical mechanism is possible. It appears that the primary effect upon the virus is to render the virion permeable. This may suffice to degrade the virion structure and in turn reduce infectivity. The presence of these degrading components in human milk may account for the difficulty of detection of the putative human mammary tumor agent.

1362 SYNERGISM BETWEEN POLYOMA AND INACTIVATED SENDAI VIRUS IN HUMAN ASTROCYTES, FIBROBLASTS AND HELA CELLS. *(E.)* Levy, N. L. (Duke U. Med. Ctr., Durham, N.C.). *J Neuropathol Exp Neurol* 31(4):639-644, 1972.

A small plaque variety of polyoma virus could be induced to replicate in normally resistant cultured human astrocytes, fibroblasts or HeLa cells when cells were fused by inactivated Sendai virus following polyoma infection. No apparent polyoma replication occurred in unfused cells or in cells fused before polyoma absorption. Fusion did not induce permanent changes in polyoma host range as virus propagated in fused cells failed to replicate upon re-inoculation into fresh cultures of any of the human cell types. Infectivity was neutralized by rabbit anti-polyoma IgG but was unaffected by mouse anti-Sendai serum or by rabbit anti-herpes simplex IgG. Although polyoma replicated to a similar extent in all three cell types, it produced a cytopathic effect only in astrocytes. Long-term cultivation of fused polyoma infected astrocytes or fibroblasts revealed no evidence of transformation.

1363 THE COMBINED EFFECT OF CHEMICAL MUTAGEN AND VIRUS ON DNA REPAIR, CHROMOSOME ABERRATIONS AND NEOPLASTIC TRANSFORMATION. *(E.)* Stich, H. F. (Dept. Zool., U. British Columbia, Vancouver), O. Hammerberg and B. Casto. *Can J Genet Cytol* 14(4):911-917, 1972.

The combined effect of 4-nitroquinoline-1-oxide (4NQO) and human (AD12) or simian (SA7) adenovirus infection on cultured Syrian hamster embryo cells was studied. 4NQO-induced DNA repair, as determined by the extent of unscheduled incorporation of ^{3}H-thymidine into stationary cells grown in arginine-deficient medium, was not influenced by infection with either virus. 4NQO treatment followed by virus infection resulted in an enhanced number of chromosomal aberrations compared to the number seen following 4NQO or virus treatment alone. Virus infection following 4NQO exposure produced up to a 23-fold increase in the rate of virus-induced cell transformation as determined by cellular expression of T-antigen within morphologically transformed foci 33 days after infection. Whether or not the virus enhanced chemical transformation could not be answered.

1364 CHROMOSOMES OF SA7 TRANSFORMED HAMSTER CELLS. II. STUDIES ON UNMATCHED CHROMOSOMES. *(E.)* Bey, E. M. (Poliomyelitis Res. Fdn., Johannesburg, S. Africa) and J. M. Whitcutt. *S Afr J Med Sci* 37:39-46, 1972.

Chromosome studies were conducted on eight different clones of hamster cells transformed *in vitro* by simian adenovirus SA7, on a group of Sa7 infected, but as yet, untransformed cells, and uninfected controls to determine the mode of origin of the unmatched chromosomes present in SA7 transformed cells. All transformed clones contained a more or less constant fraction of polyploid cells, the cultures approximated to a diploid mode. Despite this, however, the transformed karyotypes had an irregular chromosome distribution with the appearance of extra metacentric or subtelocentric markers. Occurrence of the metacentric was associated with loss of a group 7-9 chromosome in both male and female clones, whereas the unmatched subtelocentric correlated with loss of a sex chromosome in female, but not male, clones. Changes in chromosome distribution similar to those found in transformed cells could be detected in SA7 infected cultures at the first cell division after virus inoculation. These observations were consistent with SA7 virus induced chromosome breaks followed by rejoinings and translocations to produce the abnormal markers.

1365 ULTRASTRUCTURAL STUDIES OF THE ENVELOPMENT AND RELEASE OF GUINEA PIG HERPES-LIKE VIRUS IN CULTURED CELLS. *(E.)* Fong, C. K. Y. (Yale U. Sch. Med., New Haven, Conn.), R. B. Tenser, G. D. Hsiung and P. A. Gross. *Virology* 52(2):468-477, 1973.

The process of envelopment and release of guinea pig herpes-like virus was examined in both infected guinea pig kidney and thymus tissue culture cells by electron microscopy. The majority of the nucleocapsids were enveloped by budding into nuclear vacuoles; some were enveloped by budding from the inner nuclear membrane. Budding into cytoplasmic vacuoles was also seen. Many enveloped virus particles inside the nuclear vacuoles were pear shaped with a tail-like structure. Approximately 23% of pear-shaped virus particles were seen in the infected thymus fibroblastic cells, but only 6% were found in the infected epithelial cells. The envelopes of all nuclear enveloped virus particles appeared as smooth membranes, while the majority of particles exhibiting fuzzy and thick dense envelopes were seen in the cytoplasm or extracellular space; it is possible that additional host cell material was acquired during their passage through the cytoplasmic channels, although direct evidence was not obtained. The average diameter of the cytoplasmic or extracellular enveloped virus particles was approximately 167 nm, and the average diameter of the nuclear enveloped virus particles was about 146 nm. Data also showed that mature nuclear virus particles were first released into perinuclear cisterna and then traveled through cytoplasmic channels to the extracellular space.

1366 STUDY OF THE CELLULAR PROLIFERATION KINETICS OF FRIEND LEUKEMIA. *(E.)* Smadja-Joffe, F. (Inst. Cancer, Immunogenetics, Villejuif, France), C. Jasmin, E. P. Malaise and C. Bournoutian. *Int J Cancer* 11(2):300-313, 1973.

The proliferation kinetics of Friend leukemia cells have been studied *in vivo* in infected female DBA$_2$ mice. Proliferation was primarily observed in the splenic erythroblasts. Growth was biphasic with an initial rapid phase lasting from the 7th to the 21st day after injection of the viral suspension followed by a second slower phase which lasted until death at about the 30th day. The livers of infected mice began to increase in weight at a later stage following leukemic cell invasion. The peripheral blood showed early erythroblast invasion with a greatly increased lymphocytic population. The peripheral erythroblastic component did not become large until the late stages of disease. The peripheral reticulocyte count gradually increased to a maximum at the terminal phase of the disease. The mean duration of the mitotic cycle, as determined from a curve of labeled mitotic figures following ^{3}H-thymidine injection, was 7.6 hr for splenic erythroblasts. Comparison of this value with the mathematically calculated experimental doubling time (144 hr) suggested a mean cell loss of 94%. Repeated hourly ^{3}H-thymidine injections into infected mice resulted in labeling of 90-97% of the leukemic spleen and liver cells, but in less than 7% of peripheral leukemic cells. These results indicated that, although practically all splenic and hepatic erythroblasts were actively growing, the peripheral erythroblast population was unable to do so.

1367 RELATIONSHIP BETWEEN THE SENSITIVITY OF EBV-CARRYING LYMPHOBLASTOID LINES TO SUPERINFECTION AND THE INDUCIBILITY OF THE RESIDENT VIRAL GENOME. *(E.)* Klein, G. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden) and L. Dombos. *Int J Cancer* 11(2):327-337, 1973.

Several human Epstein-Barr virus (EBV) carrying producer and non-producer lymphoblastoid cell lines have been studied with respect to their sensitivity to superinfection with EBV concentrates and to the inducibility of viral antigens by S-bromodeoxyuridine (BUDR) and S-iododeoxyuridine (IUDR). Although there were major differences between cell lines with regard to both sensitivity to superinfection and to inducibility of early antigen (EA) positive cells by BUDR and IUDR, a correlation did exist between these two parameters in 23 virus-receptor-positive lines. No such correlation was found in 10 virus-receptor-negative lines which were resistant to superinfection but differed in their sensitivity to BUDR and IUDR activation. These findings suggested that the intracellular restrictive mechanism that limits superinfection in the receptor-positive, resistant lines could also restrict the function of the internally derived activated genome. This same mechanism probably did not necessarily affect spontaneous genome activation since some lines which were resistant to both superinfection and activation were also spontaneous producers.

1368 THE DIFFERENTIAL EFFECT OF ACTINOMYCIN D IN NORMAL AND VIRUS-TRANSFORMED CELLS. *(E.)* Williams, J. G. (Imperial Cancer Res. Fund Labs., London, England) and I. A. Macpherson. *J Cell Biol* 57(4):148-158, 1973.

Baby hamster kidney cell cultures, normal and transformed by polyoma or Rous sarcoma virus (Bryan), were infected with fowl plaque virus (FPV) and treated with actinomycin D (AMD). While FPV replication in normal cells was sensitive to AMD, both tumor virus-transformed cell lines were less AMD sensitive in their ability to support FPV replication. In related experiments, it was found that pretreatment of normal and transformed cells with AMD before FPV infection did not alter the pattern of differential sensitivity of FPV replication to AMD in normal and transformed cells. RNA synthesis, in all RNA classes, of the two transformed cell lines was two or three times more resistant to AMD than was RNA synthesis in untransformed lines.

1369 *IN VITRO* TRANSFORMATION OF CANINE EMBRYO CELLS BY MURINE SARCOMA VIRUS (KIRSTEN). *(E.)* Duh, F. G. (Microbiol. Associates, Inc., Bethesda, Md.), M. L. Vernon and J. S. Rhim. *Proc Soc Exp Biol Med* 142(4):1277-1280, 1973.

The results of experiments showing that canine embryo cells can be transformed morphologically *in vitro* by the Kirsten strain of murine sarcoma virus (Ki-MSV) and that these morphologically altered cells contain both infectious virus and group-specific (gs) antigen are reported. Cell-free supernatant fluids from the transformed cultures were infectious for rat, guinea pig, and canine embryo cells. RNA-dependent DNA polymerase activity was demonstrated in the transformed cells. Electron microscope observation showed that the transfered cells produced type C virus particles. The present results indicate that members of the murine sarcoma-leukemia virus complex, particularly Ki-MSV, exhibit a broader host range than was previously believed.

1370 DECREASED FORMATION OF SYNCYTIAL PLAQUES IN THE XC-TEST WITH A DOUBLE-CLONED HYPOTETRAPLOID XC-CELL LINE AND EVIDENCE OF BUDDING C-TYPE PARTICLES IN THIS AND ORDINARY HYPODIPLOID XC-CELL LINES. *(E.)* Ebbesen, P. (Inst. Med. Microbiol., U. Copenhagen, Denmark), O. Jorgensen and J. Visfeldt. *Eur J Cancer* 9(1):31-35, 1973.

An apparent connection between XC-cell karyotype and efficiency of syncytium formation in the XC-test for murine leukemia virus (MuLV) is reported. Two uncloned hypodiploid XC-cell lines, a double-cloned hypodiploid subline and a double-cloned subline consisting of a mixture of hypodiploid and hypotetraploid cells, were found equally efficient in syncytium formation upon contact with MuLV. However, a double-cloned subline apparently composed only of hypotetraploid cells showed a one log reduction in plaque count in the XC-test. The ultrastructure of all hypodiploid and hypotetraploid cells was identical. Both immature and mature C-type particles were observed within the cytoplasm and budding from the cell membrane of both these sublines. A-type particles coated with electron-dense material were also seen within the cytoplasm.

1371 CHROMOSOMES OF SA7 TRANSFORMED HAMSTER CELLS. I. PRELIMINARY STUDIES. *(E.)*

Bey, E. M. (Poliomyelitis Res. Fdn., Johannesburg, S. Africa) and J. M. Whitcutt. *S Afr J Med Sci* 37:33-38, 1972.

The characteristics of the chromosome changes induced by simian adenovirus SA7 during *in vivo* and *in vitro* transformation of hamster kidney and skin cells were studied. All nine SA7 transformed cell lines investigated showed numerical and structural chromosomal abnormalities. Cultures derived from *in vivo* transformed tumor cells were characterized by a high modal number; whereas, cultures transformed *in vitro* and either maintained *in vitro* or *in vivo* in weanling hamsters displayed near-diploidy or pseudo-diploidy. Although loss or gain of chromosomes appeared to be random with respect to grouping, large, unmatched chromosomes, usually occurring singly, were present in a high percentage of karyotypes from all specimens. They were of two types: 1) a near metacentric or metacentric longer than no. 5 and sometimes difficult to distinguish from a Y or a contracted X chromosome and 2) a subtelocentric longer than no. 1 with a secondary constriction, coiling deficiency or other defect and sometimes confused with distorted or incompletely coiled X chromosomes. These two markers were rarely found together within the same metaphase plate.

1372 STUDIES ON BURKITT'S LYMPHOMA. I. A PRELIMINARY REPORT ON THE PRESENCE OF MYCOPLASMA-LIKE ORGANISMS IN SPECIMENS EXAMINED BY ELECTRON MICROSCOPY. *(E.)*

Spence, I. M. (Poliomyelitis Res. Fdn., Johannesburg, S. Africa). *S Afr J Med Sci* 37:27-32, 1972.

Mycoplasma-like organisms have been observed in electron micrographs of biopsy and necropsy specimens taken from four male and female Burkitt's lymphoma patients. Cell suspensions prepared from these specimens produced fatal bronchopneumonia following s.c. inoculation into male *cercopithecus aethiops* monkeys and caused cytopathic effects in several passages of a hamster adult kidney cell line. The infected tissue culture material contained structures resembling elementary bodies within intra-cellular vacuoles in addition to mature extra-cellular forms. Whether or not these organisms play a role in the etiology of Burkitt's lymphoma is unknown.

1373 MECHANISM OF SHOPE FIBROMA VIRUS-INDUCED SUPPRESSION OF HOST DEOXYRIBONUCLEIC ACID SYNTHESIS. *(E.)*

Chan, J. C. (M. D. Anderson Hosp. Tumor Inst., Houston, Texas) and M. E. Hodes. *Infect Immun* 7(4):532-538, 1973.

The effects of treatment with live or inactivated Shope fibroma virus on host cell deoxyribonucleic acid (DNA) synthesis were determined. HeLa cell or LLC-RK_1 cell monolayers were infected with virus at a multiplicity of infection of approximately 1 or 5 plaque-forming units per cell. Cells were fed with medium and incubated. Uninfected controls were treated with an equal volume of medium. The incorporation of ^{3}H-thymidine into nuclear DNA was suppressed by both active and inactivated virus, although live virus was more effective. During the early phase of infection, stimulation of host nuclear DNA synthesis of up to 240% of control value was observed in cells infected with active virus. Inhibition of DNA synthesis began at about the 8th hr and was maximal by 12 hr postinfection. Virus inactivated by ultraviolet-irradiation or heat treatment did not induce viral DNA synthesis but was, nevertheless, able to suppress host DNA synthesis.

1374 BIOCHEMICAL AND MORPHOLOGICAL STUDIES OF SIMIAN SARCOMA VIRUS, TYPE 1. *(E.)*

Harewood, K. R. (J. L. Smith Mem. Cancer Res., Pfizer, Inc., Maywood, N.J.), J. G. Vidrine, D. L. Larson, J. S. Wolff III, G. Schidlovsky and S. A. Mayyasi. *Biochim Biophys Acta* 308(2): 252-259, 1973.

The RNA of Simian Sarcoma Virus, Type 1 (SSV-1) as well as its morphological and biochemical properties were examined. A normal primary human-embryonic cell culture (HE) was infected with SSV-1 and subsequently studied by electron microscopy. Radioactively labeled RNA from the virus was characterized by rate zonal centrifugation and acrylamide-agarose gel electrophoresis. Morphological characterization revealed that the intracellular development of SSV-1 is typical of that of C-type viruses. The extraction of a high molecular wt (60-70S) RNA component from this virus and the subsequent demonstration that these component is complexed with the DNA product of the reverse transcriptase reaction show that this virus resembles other RNA tumor viruses biochemically. The detection of a 4-10S RNA component in the RNA of SSV-1 is also characteristic of C-type viruses. The significance of the 28S and 18S RNA components has not been determined. The 4-10S radioactive peak seen when the product of the reverse transcriptase assay was examined on glycerol gradients could represent DNA that has been detached from its high MW template, along with DNA resulting from polymerase activity on other RNA templates. The sensitivity of the SSV-1 high molecular wt RNA to formamide suggests a subunit structure for the viral high molecular wt RNA, a property which has been demonstrated for the high molecular wt RNA of a number of C-type RNA viruses.

1375 HOST-GENE CONTROL OF TYPE-C RNA TUMOR VIRUS EXPRESSION AND TUMORIGENESIS IN INBRED MICE.

(E.) Meier, H. (Jackson Lab., Bar Harbor, Me.), B. A. Taylor, M. Cherry and R. J. Huebner. *Proc Natl Acad Sci* 70(5):1450-1455, 1973.

Genetic hybridization experiments involving a high leukemia incidence mouse strain (AKR/J) and a low incidence strain (C57L/J), their reciprocal F_1 hybrids, and the F_2 and backcross generations of this cross were conducted to determine whether or not a relationship exists between endogenous viral expression and tumorigenesis. A highly significant ($P < 0.001$), predictable association was found between the expression of complete infectious virus, or gs antigen, as determined by complement fixation or the COMuL test, and tumorigenesis in later life. Most tumors were thymic leukemia or reticulum cell sarcoma (RCS). Of 124 tumors occurring in parental C57L/J mice, 110 developed in gs-positive animals. All gs- and virus-positive AKR/J mice and all but one of the F_1 hybrids developed tumors. All 56 gs-positive F_2 progeny developed tumors. It was thus concluded that the endogenous C-type RNA viral genome was a major determinant of at least leukemias and RCS. In addition, a possible minor effect of the H-2 histocompatibility locus on leukemogenesis was found in F_2 mice.

1376 ATTEMPTS TO PRODUCE TUMORS IN MICE WITH CELO VIRUS.

(E.) Mancini, L. O. (Dept. Animal Path., U. Rhode Island, Kingston) and V. J. Yates. *Arch Gesamte Virusforsch* 40:174-175, 1973.

Outbred white Swiss pregnant mice were randomly selected for use in tests to determine the oncogenicity of chicken-embryo-lethal-orphan (CELO) virus, an avian adenovirus. Eight of 20 mice injected intrathoracically with 0.1 ml of CELO virus ($ELD_{50}=10^{8.2}/0.1$ ml) survived the ten month observation period. Those that survived were stunted and underweight; however, none of the 20 mice revealed tumorous growths at autopsy. Those animals that were injected s.c. or intracranially with CELO virus all appeared perfectly normal with no indication of tumor induction. Eighteen of 21 inoculated s.c. survived the ten month observation period and 20 of 22 inoculated intracranially were alive at the end of the test period. Neoplasms were not found in any of the control animals which were injected with phosphate buffered saline in a manner similar to the test animals. The inability to induce tumors in mice with CELO virus is consistent with reports for other adenoviruses, with the exception of human adenovirus type 12.

1377 LOCALIZATION OF ENDOGENOUS C-TYPE VIRUS IN THE GLOMERULAR BASEMENT MEMBRANE OF AGED AKR MICE.

(E.) Batzing, B. L. (Carcinogenesis Program, Biol. Div., Oak Ridge Natl. Lab., Tenn.) and M. G. Hanna, Jr. *J Immunol* 110(4):1189-1193, 1973.

The localization of intact endogenous C-type virus within the basement membrane of aged AKR mice showing evidence of chronic glomerulonephritis is reported. One year old male AKR/Cum mice were used; the specific antiserum employed was obtained from rats with a transplanted Moloney sarcoma virus tumor. This serum reacts with Friend-Moloney-Rauscher and Gross virus envelope and cell-surface antigens and against the group-specific antigen of the murine leukemia virus (MuLV). Histologic examination of kidneys from 53 1-yr old, male mice revealed a high incidence of glomerulonephritis. Fourteen animals had more severe glomerular lesions than the others and were selected for additional studies. In electron microscopic studies of nephritic kidney of these animals, the majority of sections showed occasional C-type virus; 10 to 20%, however, had extensive localization of endogenous C-type virus particles within the amorphous material of the basement membrane. This observation suggests a binding of specific antibody to virus envelope antigens of the intact virus with subsequent virus inactivation and entrapment of the antibody-coated virus in the glomerular basement membrane.

1378 MOLECULAR RELATEDNESS OF MAMMALIAN RNA TUMOR VIRUSES AS DETERMINED BY DNA·RNA HYBRIDIZATION.

(E.) Haapala, D. K. (Natl. Cancer Inst., Bethesda, Md.) and P. J. Fischinger. *Science* 180(4089):972-974, 1973.

Hybridization experiments utilizing viral RNA and ^3H-labeled DNA synthesized by reverse transcriptase have shown that six mammalian C-type viruses-including two feline leukemia viruses, three murine leukemia viruses, and the human "candidate" virus RD-114, can be distinguished from one another. Hydroxyapatite column chromatography of heterologous and homologous DNA·RNA hybrids indicated that, whereas the two feline viruses and the three murine viruses each had closely related nucleotide sequences within their respective groups, little similarity existed between feline, murine or human C-type virus nucleic acids. Decreased thermal stability of the heterologous DNA·RNA hybrids confirmed these results. These studies indicated that molecular hybridization techniques can be used to form a taxonomic scheme for the classification of mammalian RNA tumor viruses.

1379 SEGREGATION OF LOCI FOR C-TYPE VIRUS. INDUCTION IN STRAINS OF MICE WITH HIGH AND

LOW INCIDENCE OF LEUKEMIA. *(E.)* Stephenson, J. R. (Natl. Cancer Inst., Bethesda, Md.) and S. A. Aaronson. *Science* 180(4088):865-866, 1973.

The biologic properties of viruses activated from a series of second generation embryos obtained from backcrosses involving the noninducible Swiss NIH strain with (NIH Swiss x Balb/c)F_1 and (NIH Swiss x C58)F_1 hybrids were examined. C58 mice have a high incidence and Balb/c have a low incidence of leukemia. Treatment of 51 and 55 NIH Swiss x (NIH Swiss x C58)F_1 backcross embryo cultures with Iododeoxyuridine (IdU) resulted in induction of C-type virus as detected by radioimmunoassay and the XC-plaque assay. These results fit best with those expected for three or four independently segregating dominant alleles for virus inducibility in C58 cells. Study of the biologic properties of virus activated from NIH x (NIH x Balb/c)F_1 backcross embryos showed that they were identical to those of virus induced from cells of the parental Balb/c strain and differed from those of virus induced from NIH x (NIH x C58)F_1 embryos. These results indicated that the inducibility loci detected may represent C-type viral structural information and that regulatory factors necessary for their activation were either present in all cell strains or closely linked to the viral structural loci.

1380 DETERMINATION OF NATURAL HOST TAXONOMY OF RNA TUMOR VIRUSES BY MOLECULAR HYBRIDIZATION: APPLICATION TO RD-114, A CANDIDATE HUMAN VIRUS.

(E.) Ruprecht, R. M. (Inst. Cancer Res., Columbia U., New York, N.Y.) N. C. Goodman and S. Spiegelman. *Proc Natl Acad Sci USA* 70(5):1437-1441, 1973.

Molecular hybridization studies have been conducted using purified human or cat DNA and ^3H-labeled DNA synthesized in a reverse transcriptase reaction with 70S RNA from RD-114, the candidate human oncogenic C-type virus. Whereas 78% of the RD-114 DNA was involved in hybridization to DNA purified from normal feline cells, no detectable annealing occurred with human or avian DNAs. The RD-114 DNA, which did not anneal to normal feline DNA, hybridized with DNA from cat mastocytomas but not with DNA from normal human tissues. These findings suggested that RD-114 is of feline, not human, origin.

1381 CELLULAR LOCALIZATION OF AN EPSTEIN-BARR VIRUS (EBV)-ASSOCIATED COMPLEMENT-FIXING ANTIGEN IN PRODUCER AND NON-PRODUCER LYMPHOBLASTOID CELL LINES.

(E.) Reedman, B. M. (Karolinska Inst., Stockholm, Sweden) and G. Klein. *Int J. Cancer* 11(3):499-520, 1973.

Anti-complement immunofluorescence (ACIF) was used to study the complement-fixing antigens of human lymphoblastoid cell lines. These cell lines carry the Epstein-Barr virus (EVB) genome although only producer cultures synthetize EBV-specific antigens (virus capsid antigen, VCA and early antigen, EA) detectable by direct and indirect immunofluorescence, usually in less than 5% of the cells. The ACIF test revealed an antigen localized in the nucleus of the lymphoblastoid cells. In contrast to EA and VCA, this antigen was present in over 90% of the cells of both producer and nonproducer cultures. The antigen was shown to be specific for EBV by comparing the reactions of 52 sera in the ACIF test. Sera giving the nuclear reaction contained antibodies to VCA, EA or antigens detectable by complement fixation tests on cell extracts, but sera without EBV antibodies failed to give the reaction. Weak, equivocal or discordant reactions occurred with six sera with low titres in VCA, EA or complement fixation tests. Cell lines derived by transformation of human and primate lymphocytes by EBV gave the nuclear reaction. Control cells with no known association with EBV were non-reactive. These included fetal lymphocytes transformed by phytohemagglutinin, cell lines derived from breast cancer, glioma, normal glia, pleuritis maligna and myeloma, and two marmoset lymphoid lines carrying Herpesvirus saimiri (HVS). In preliminary experiments, the ACIF test was used as a tool to trace the EBV genome at the cellular level. Cells from two Burkitt lymphoma biopsies, one tested after biopsy and one after passaging in nude mice, contained an EBV-specific antigen. Three clones of cells derived from hybrids of mouse somatic cells and a human lymphoblastoid cell line also contained such an antigen, but the number of reactive cells varied from clone to clone. A fourth clone was non-reactive.

1382 POLYSACCHARIDES OF SV 40-TRANSFORMED GREEN MONKEY KIDNEY CELLS.

(E.) Makita, A. (Tohoku U. Sch. Med., Sendai, Japan) and H. Shimojo. *Biochim Biophys Acta* 304(2):571-574, 1973.

Glycoproteins and mucopolysaccharides in cultured green monkey kidney cells (GMK cells) and their counterpart transformed with SV 40 virus (GMK-SV cells) were studied. GMK cells, strain C14, and GMK-SV cells, Strain T22, were cultured until they formed a confluent layer. Total polysaccharides from each cell culture were separated into glycoproteins and mucopolysaccharides by precipitation with quarternary ammonium salts. Individual mucopolysaccharide class was determined by electrophoresis on a cellulose acetate sheet followed by colorimetry of metachromatic stains with Alcian blue. Glycopeptides were recovered from the cetylpyridinium chloride (CPC)-soluble fraction. Mucopolysaccharides were isolated from a CPC- in soluble fraction. A quantitative survey of mucopolysaccharide composition showed a relative increase in hyaluronic acid and chondroitinase-resistant mucopolysaccharides, of which a major portion was most probably heparan sulfate, and a relative decrease of chondroitin sulfates in the transformed cells. Because heparan sulfate is associated with the cell surface, the present results may demonstrate the changes in the cell surface occurring on viral transformation.

1383 RNA-DIRECTED DNA POLYMERASE AND VIRUS-INDUCED LEUKEMIA IN MICE.

(E.) Wu,

A. M. (Litton Bionetics, Inc., Bethesda, Md.), R. C. Y. Ting and R. C. Gallo. *Proc Nat Acad Sci, USA* 70(5):1298-1302, 1973.

The inhibitory effect of rifamycin SV derivatives on murine leukemogenesis as measured by the degree of splenomegaly was examined. Four- to 6-week old NIH Swiss mice of either sex were injected i.v. through the tail vein with 0.2 ml Rauscher leukemia virus (RLV) (about 5×10^5 plaque-forming units/ml), at dilutions ranging from 100- to 5000-fold. At various times afterwards the mice were killed and their spleens weighed. No obvious splenomegaly occurred for the first two weeks. At the fifth and eighth weeks after inoculation, spleen size increased as a function of virus concentration. It was found that rifamycin antibiotics prevent the leukemogenic activity of RLV, and that the effect of leukemogenesis correlates with the magnitude of the inhibition of RNA-directed DNA polymerase of the virus. The inhibition of enzyme activity is due to a relatively tight binding between the enzymes and the inhibitors, yet the binding can be reversed by nonionic detergents. It is suggested that RNA-directed DNA polymerase is essential for induction of leukemia by exogenous virus. These findings correlate with the previous observation that the same derivatives block viral transformation *in vitro*.

1384 SUGAR EFFECTS ON MURINE SARCOMA VIRUS TRANSFORMATION. *(E.)* Hatanaka, M. (Flow Labs., Inc., Rockville, Md.). *Proc Natl Acad Sci* 70(5):1364-1367, 1973.

The effects of different carbohydrates on sugar transport and transformation of secondary cultures of growing NIH Swiss mouse embryo fibroblasts by the Harvey strain of murine sarcoma virus (MSV) were studied. MSV-induced focus formation was decreased when cells were grown in medium containing 0.1% D-galactose, L-arabinose, D-mannose, L-xylose, α-methyl D-glucose, D-glucose, 2-deoxy-D-fucose, L-fucose or β-D-thioglucose. D-Mannose and 2-deoxy-D-glucose also delayed the onset of and reduced tumor incidence in MSV-infected newborn mice. L-Mannose, L-glucose, D-inulin and D-mannoheptulose had no effect on focus formation. The uptake of isotopically labeled D-glucose, D-mannose and D-galactose into normal, uninfected cells was stimulated by D-xylose, L-xylose, L-arabinose and, to a lesser extent, by D-galactose when compared to the extent of incorporation in the presence of D-glucose or D-mannose. A similar stimulation was also observed in MSV-infected cells; however, sugar transport in infected cells was consistently greater than that in stimulated uninfected cells. These results suggested the presence of a second mechanism for the enhancement of sugar uptake in MSV-infected cells which is not present in uninfected cells.

1385 ONCOGENIC TRANSFORMATION OF HAMSTER EMBRYO CELLS *IN VITRO* BY ADENOVIRUS TYPE 5. *(E.)* Williams, J. F. (MRC Virology Unit, U. Glasgow, Scotland). *Nature* 243:162-163, 1973.

To determine if adenovirus type 5 can transform permissive cells, secondary cultures of hamster embryo fibroblasts (HEF) were infected with one of the temperature sensitive (ts) mutants, ts1, ts4, or ts14, at the restrictive temperature which prevents cell killing caused by virus replication. For transformation, the infected cells were incubated at 38.5 C for two days, then subcultured by seeding at a density of either 4×10^5 or 10^6 cells per 50 mm dish. These subcultures were then cultivated at 38 C for up to 6 wk. Transformed foci first appeared approximately 4 wk after infection. The morphology of these transformed HEF cells was similar to that of rat embryo cells transformed by adenovirus type 5. A number of single foci were picked from cultures infected by each mutant and six lines established. Cells of all six lines retained the transformed morphology throughout many passages *in vitro*. The transformed cells could be superinfected by wild type virus at either temperature and by various ts mutants at the permissive temperature of 31 C. After five passages *in vitro*, cells of two of these lines were inoculated i.p. into 1-day old hamsters. Approximately 6 wk later, some of these animals had developed i.p. tumors, which were found to be transplantable and have been transplanted for up to six consecutive passages. In a few animals metastases to either lungs or liver were observed. The results indicate that the adenovirus type 5, until now considered to be a non-oncogenic adenovirus, is capable of oncogenic transformation of cells *in vitro*.

1386 STUDIES OF THE EPSTEIN-BARR VIRUS-HOST RELATIONSHIP: AUTOCHTHONOUS AND ALLOGENEIC LYMPHOCYTE STIMULATION BY LYMPHOBLAST CELL LINES IN MIXED CELL CULTURE. *(E.)* Bausher, J. C. (Coll. Med., U. Florida, Gainesville) and R. T. Smith. *Clin Immunol Immunopathol* 1(2):270-281, 1973.

The capacity of 21 human lymphoblast cell lines established from both normal individuals and those with infectious mononucleosis (IM) to stimulate DNA synthesis in peripheral blood leukocyte populations of autochthonous or allogeneic origin was compared in one-way mixed lymphocyte cultures. Strong stimulation in the autochthonous combination was found in 20 of 21 pairs, and weak but significant stimulation was observed in the exceptional pair. Allogeneic pairs gave additional increments of stimulation over that of the autochthonous pair in 21 of 31 pairs tested. The observed stimulation was shown not to be due to a secreted blastogenic agent. The data support but do not prove that Epstein-Barr Virus (EBV)-related antigens are responsible for the observed stimulation. This implies the existence of an EBV-immune cell population recognizing specific antigens in the target cell membrane.

1387 PURIFICATION OF ONCORNAVIRUSES BY AGGLUTINATION WITH CONCANAVALIN A. *(E.)* Stewart, M. L. (Albert Einstein Coll. Med., Bronx, N.Y.), D. F. Summers, R. Soeiro, B. N. Fields and J. V. Maizel, Jr. *Proc Natl Acad Sci* 70(5):1308-1312, 1973.

Selective agglutination with concanavalin A (con A) has been used to purify Friend leukemia virus from culture medium or plasma. Treatment of supernatant medium from infected cultures with 800 μg/ml con A at 25 C for one hr followed by low speed centrifugation and resolubilization of the virus-con A pellet in α-methyl mannoside resulted in recovery of over 64% of the total infectivity of the medium and a 2.3-fold increase in infectivity with respect to nucleic acid content. Sucrose gradient centrifugation of uridine prelabeled con A agglutinated virus produced a sharp band of infectivity between 1.15 and 1.17 gm/cm^3. Con A did not bind to infectious particles with a density of 1.17 to 1.19 gm/cm^3. Con A agglutination had no effect on Friend virus specific infectivity or morphology. The ability of con A to agglutinate selectively ^{3}H-labeled virus which banded at a density of 1.16 gm/cm^3 further indicated the specificity of the technique.

1388 TRANSFER RIBONUCLEIC ACID SYNTHETASE ACTIVITY ASSOCIATED WITH AVIAN MYELOBLASTOSIS VIRUS. *(E.)* Erikson, E. (U. Colorado Med. Ctr., Denver) and R. L. Erikson. *J Virol* 9(2):231-233, 1972.

Avian myeloblastosis virus (AMV) preparations have been examined for tRNA synthetase activity. AMV protein extracts were found to contain several synthetase activities as judged from the ability to utilize ^{3}H-labeled arginine, tryptophan, cystine and lysine in an *in vitro* aminoacylation assay. Pre-treatment of AMV with the nonionic detergent, NP-40, was necessary to release the tRNA synthetase activities, indicating that these enzymes were membrane-protected. Enzyme activity sedimented in sucrose density gradients at the same rate as did ^{32}P-labeled AMV. The role, if any, of tRNA synthetase in AMV replication is unknown.

1389 VIRUS-INDUCED TRANSFORMATION WITHOUT CELL DIVISION. *(E.)* Bader, J. P. (Natl. Cancer Inst., Bethesda, Md.). *Science* 180(4090):1069-1071, 1973.

Cultured chick embryo cells infected with the Bryan "high titer" strain of Rous sarcoma virus (RSV) and then exposed to vinblastine sulfate to prevent cell division were assayed for their ability to incorporate ^{3}H-deoxyglucose and to synthesize hyaluronic acid, two biochemical processes characteristic of RSV-transformed cells. Infected, vinblastine treated cells showed both an increased capacity to incorporate deoxyglucose and to synthesize hyaluronic acid (polyacrylamide-agarose gel assay). These results, along with an earlier demonstration of morphological changes developing in infected nondividing cells, demonstrated that the phenotypic development of the malignant state can occur without the intervention of cell divisions after infection by RSV.

1390 A TEMPERATURE-SENSITIVE MUTANT OF SIMIAN VIRUS 40 AFFECTING TRANSFORMING ABILITY. *(E.)* Kimura, G. (Salk Inst., San Diego, Calif.) and R. Dulbecco. *Virology* 52(2):529-534, 1973.

In productive infection of monkey BSC-1 cells, ts 640, a temperature-sensitive (ts) mutant of simian virus 40 (SV40), was defective in its ability to synthesize infectious viral DNA and infectious viral particles at the restrictive temperature of 40°, but not at the permissive temperature of 33°. Growth of the mutant after infection with viral DNA was also temperature-sensitive, indicating that the block to multiplication at 40° is not due to failure in adsorption, penetration or uncoating. Results of mixed infections with other ts mutants showed that the gene identified in this mutant is different from those two cistrons which had been suggested to specify virion proteins. In nonproductive infection of rat 3Y1 cells, ts 640 was temperature-sensitive in its transforming ability at 40° but not at 33°, providing evidence that cell transformation by SV40 is under the control of a viral gene function. The transformed cells obtained at 33° appeared not to revert to normal when shifted to 40°, as judged by their colonial morphology under the standard culture conditions, indicating that the viral gene function under study may be required for the initiation but not for the maintenance of transformation.

1391 MEASUREMENT OF ENDOGENOUS LEUKOSIS VIRUS NUCLEOTIDE SEQUENCES IN THE DNA OF NORMAL AVIAN EMBRYOS BY RNA-DNA HYBRIDIZATION. *(E.)* Neiman, P. E. (U. Washington Sch. Med., Seattle). *Virology* 53(1):196-203, 1973.

Rous associated virus type 0 (RAV-0) is a subgroup E avian leukosis virus selected as an example of "endogenous" leukosis viruses which are associated with normal avian embryo cells. DNA sequences complementary to RAV-0 60-70S RNA genome were detected and quantitated in DNA from normal avian embryos by the technique of RNA-DNA hybridization with DNA excess. DNA from "leukosis free" white leghorn chicken embryos, as well as from line 7 chicken embryos, contained sequences related to at least 70%, if not all, of RAV-0 RNA sequences. A tentative estimate of gene frequency is low, in the range of two to four copies of RAV-0 per cell genome. A number of factors limit the precision of the frequency estimate: 1) complete hybridization was not achieved; 2) the slight departure of the first third of the experimental points from the expected curve on which the frequency estimate was based; and 3) the frequency estimate is based on comparison with hybridization kinetics of complementary RNA which differs in base ratio and possibly in secondary structure from that of viral RNA. Embryos of ring-necked pheasant contain only about 10% of the RAV-0 genome. These findings are not affected by the presence or absence of viral group specific (*gs*) antigen in chicken or pheasant embryos. Embryos of Japanese quail, a species which lacks a known leukosis virus and does not synthesize avian *gs* antigen, contained DNA sequences specifically related to no more than 4% of the RAV-0 genome. The data document the specificity and sensitivity of this hybridization technique and provide additional firm support for the proposition that normal avian cells carry endogenous avian leukosis virus in the form of DNA proviruses.

1392 THE RELATIONSHIP OF VISNA, MAEDI AND RNA TUMOR VIRUSES AS STUDIED BY MOLECULAR HYBRIDIZATION.

(E.) Harter, D. H. (Coll. Physicians, Surgeons, Columbia U., New York, N.Y.), R. Axel, A. Burny, S. Gulati, J. Scholm and S. Spiegelman. *Virology* 52(1):287-291, 1973.

The relationship of visna and maedi viruses was investigated in experiments designed to determine if nucleic acid sequence homology could be demonstrated by molecular hybridization. Studies were also made to determine if hybridization could be demonstrated between visna and maedi virus nucleic acids and the nucleic acids of representative mammalian RNA tumor viruses. The DNA product of the endogenous visna virus RNA-directed polymerase reaction annealed to maedi virus 60-70S RNA. Similarly, the DNA product of the endogenous maedi virus polymerase reaction hybridized to visna virus 60-70S RNA. The DNA products of endogenous visna and maedi virus polymerase reactions failed to anneal significantly either to Rauscher murine leukemia or mouse mammary tumor virus 60-70S RNAs. The results indicate that visna virus and maedi virus have homologous nucleic acid sequences.

1393 RELATION OF CYTOHISTOPATHOLOGY OF GENITAL HERPESVIRUS TO CERVICAL ANAPLASIA.

(E.) Naib, Z. M. (Emory U. Sch. Med., Atlanta Georgia), A. J. Nahmias, W. E. Josey and S. A. Zaki. *Cancer Res* 33(6):1452-1463, 1973.

Results from previous studies had established an association between genital herpes infection and cervical "anaplasia" (i.e., dysplasia, *in situ* or invasive carcinoma). The present study was conducted on 674 patients with cytological evidence of herpes infection to determine, in greater detail, the histological and cytological structural changes associated with genital herpetic disease. The first cytological evidence of herpes infection in Papincolaustained specimens was nuclear and cytoplasmic hypertrophy, especially of the basal, parabasal and reserve cells. The nucleoli enlarged and became granular, and, simultaneously, the cytoplasmic ground substance became opaque, staining dark purple of bluegreen. This was followed by disappearance of the nucleoli and the appearance of nuclear B-bodies which displaced the chromatin peripherally. Infected cells finally became multinucleated, began to degenerate, and intranuclear acidophilic A-inclusion bodies appeared. Bizarrly shaped, degenerated cells rapidly exfoliated, resulting in epithelial ulceration. Forty-five of 126 patients who also had cervical punch biopsies showed histological evidence of herpes infection which had the same characteristics seen in the cytological specimens. Herpetic cells were seen primarily at the edges of the ulcers which were often localized at or near the squamocolumnar junction. The 81 histological specimens which revealed no evidence of herpes infection showed chronic nonspecific cervicitis, granulation tissue and nonspecific ulceration.

1394 INVESTIGATIONS OF THE MECHANISM OF DECREASED TUMORIGENICITY OF CELLS GROWN IN BrdU.

(E.) Rothschild, H. (Harvard. Med. Sch., Boston, Mass.) and P. H. Black. *J Cell Physiol* 81(2):217-224, 1973.

The tumorigenicity of SV40-transformed hamster kidney cells made thymidine kinase negative (dTK$^-$) by repeated passage *in vitro* in the presence of 5-bromodeoxyuridine (BrdU) was studied. Whereas inoculation of weanling hamsters with dTK$^+$ cells resulted in 95% tumor growth by 8 wk, only 3% (2 of 78 animals) showed comparable tumor growth 8 wk following inoculation with dTK$^-$ cells. However, by 16 wk, approximately half the animals inoculated with dTK$^-$ cells had tumors containing dTK$^+$ cells, as determined from the ability to phosphorylate ^{14}C-thymidine *in vitro*. Eighty-five percent of animals inoculated with dTK$^+$ cells developed tumors > 1 cm in mean diameter 16-20 wk after inoculation as compared with 58% of animals inoculated with dTK$^-$ cells. It was concluded that loss of tumorigenicity of SV40 transformed hamster cells can occur when BrdU is incorporated into cellular DNA, or when there is a genetic modification of dTK activity.

1395 THE GENETICS OF HERPESVIRUS.

(E.) Subak-Sharpe, J. H. (Inst. Virol., U. Glasgow, Scotland). *Cancer Res* 33(6):1385-1392, 1973.

Genetic studies using temperature sensitive (ts) mutants of herpes simplex virus (HSV) types 1 and 2 were conducted. Complementation and recombination studies with syncytium (*syn*) and nonsyncytium (*syn+*) forming varieties of HSV-1 enabled construction of a linkage map of nine ts mutants and the *syn* locus. Since no good independent marker like the *syn* locus of HSV-1 was available in HSV-2 and since no 3-point crosses could be accomplished, a linkage map for the HSV-2 ts mutants could not be contructed. The ability of ts mutants of HSV-1 and HSV-2 to complement one another established the ability of HSV-1 and HSV-2 to interact nongenetically. In addition, recombinants were isolated which were immunologically intermediate between HSV-1 and HSV-2. Some of these recombinants continued to segregate for the *syn* and/or ts markers for at least three progeny generations. These observations show that genetic interactions between HSV-1 and HSV-2 are also possible.

1396 SIMIAN VIRUS 40 DNA INTEGRATION WITHIN THE GENOME OF VIRUS-TRANSFORMED MAMMALIAN CELLS. *(E.)* Gelb, L. D. (Natl. Inst. Hlth., Bethesda, Md.) and M. A. Martin. *Virology* 51(2): 351-357, 1973.

The location of integrated SV40 DNA in a series of virus-free transformed murine cells was analyzed by characterizing the reiteration frequency of adjacent host cell DNA sequences. Cellular DNA was mechanically sheared to fragments approximately 8 X 10^6 or 7 X 10^5 daltons in size, separated into reiterated and nonreiterated fractions, and then examined for the presence of SV40 DNA. SV40 DNA sequences were associated with the nonreiterated fraction of mammalian DNA in each of the cell lines examined. In addition, the pattern of viral DNA distribution between the repetitive and unique cellular DNA fractions varied from cell line to cell line suggesting that integration of SV40 DNA may occur at different sites in each cell line examined.

1397 THE TRANSCRIPTION AND STATE OF HERPES SIMPLEX VIRUS DNA IN PRODUCTIVE INFECTION AND IN HUMAN CERVICAL CANCER TISSUE. *(E.)* Roizman, B. (Dept. Microbiol., Biophys., U. Chicago, Ill.) and N. Frenkel. *Cancer Res* 33(6):1402-1416, 1973.

The transcriptional program of herpes simplex viruses 1 and 2 (HSV-1 and HSV-2) in productive infection in human epidermoid (HEp-2) cells and the transcription and the state of HSV-2 DNA in a human cervical tumor were examined. In productive infection, a total of 48% of HSV-1 DNA is transcribed. Analysis of the transcripts shows two kinds of controls. "Off-on" control of transcription is evident from the fact that early, before the onset of viral DNA synthesis, the transcripts arise from 44% of the DNA whereas late in infection the transcripts present in the infected cell arise from 48% of the DNA. The extent of transcription of the HSV-1 DNA early in infection is not affected by cycloheximide, an inhibitor of protein synthesis. Control of RNA abundance is evident from the observation that the transcripts present both early and late in infection form two classes differing in molar ratios. The abundant RNA is complementary to 14 and 19% of viral DNA early and late in infection, respectively, whereas the scarce RNA is complementary to 30 and 28%, respectively, of the DNA at the same time intervals. Several lines of evidence suggest that the abundant RNA specifies structural proteins of the virus. A cervical tumor free of virus or viral antigen was analyzed by DNA-RNA hybridization techniques for the presence of viral RNA transcripts. The cervical tumor contained viral RNA transcripts complementary to 5% of HSV-2 DNA; preliminary studies show that they correspond to both early and late transcripts. Analysis of the cervical DNA for the presence of HSV-2 DNA sequences led to three conclusions: 1) only a fragment representing 40% of HSV-2 DNA was present; 2) the fragment was present at concentrations of 1 mole/mole of cell DNA; and 3) at least parts of this fragment are covalently linked to host DNA.

1398 HUMAN EMBRYONIC LUNG CELLS TRANSFORMED BY HERPES SIMPLEX VIRUS. *(E.)* Munk, K. (German Cancer Res. Ctr., Heidelberg, W. Germany) and G. Darai. *Cancer Res.* 33(6):1535-1538, 1973.

Epithelioid cells isolated from primary human embryonic lung cultures were abortively infected by two strains of herpes simplex virus (HSV). The cells were exposed to HSV at 37 C and then transferred to 42 C. After 48 hr, when the medium still contained a high percentage of abortively infected cells, cells floating in the medium were transferred and maintained in culture at 42 C for 15 to 20 days. Cells floating in these cultures were transferred and maintained at 37 C. These latter cultures showed syncytia formation without actual virus production, indicating that the cells were transformed. The cells contained virus-associated protein not detected in mock-infected cells. Immunofluorescence studies of these transformed cells revealed cytoplasmic HSV fluorescence characteristic of that seen in other cells during early infection. The abortively infected cells could be maintained to higher passage numbers than could uninfected embryonic lung cultures.

1399 ENDOGENOUS TYPE C VIRUS FROM A CAT CELL CLONE WITH PROPERTIES DISTINCT FROM PREVIOUSLY DESCRIBED FELINE TYPE C VIRUS. *(E.)* Livingston, D. M. (Natl. Cancer Inst., Bethesda, Md.) and G. J. Todaro. *Virology* 53(1):142-151, 1973.

A type C virus has been isolated from a continuous line of feline kidney fibroblasts (CCC). The virus particle contains a reverse transcriptase and group-specific protein which cross react immunologically with the analogous proteins of RD-114 but not with the previously described feline leukemia-sarcoma viruses. A cloned line of non-virus-producing CCC cells isolated prior to the "spontaneous" activation of the CCC virus contains a type C virus which is produced after iododeoxyuridine treatment. The reverse transcriptase and gs protein of the induced virus also share antigenic properties with the same proteins of RD-114 but not the feline leukemia-sarcoma viruses. Induction from a virus-free single cell clone establishes that CCC virus is an endogenous feline type C virus. Virus-negative CCC cells and feline embryo cells are resistant to exogenous infection by either the induced or the spontaneously appearing CCC virus; however, human and other primate cells readily support their replication. Thus, certain primate, but not feline cells, appear to be permissive hosts for growth of an endogenous type C virus of the cat. The findings show that there is an infectious type C viral genome in somatic cat cells. While the data indicate that there is significant relatedness between 2 virus-specific proteins of the endogenous feline CCC virus and the RD-114 virus, they do not entirely establish the species of origin of the latter.

1400 COMPARISON OF OLIGONUCLEOTIDES PRODUCED BY RNASE T1 DIGESTION OF 7 S RNA FROM

AVIAN AND MURINE ONCORNAVIRUSES AND FROM UNINFECTED CELLS. *(E.)* Erikson, E. (U. Colorado Med. Sch., Denver), R. L. Erikson, B. Henry and N. R. Pace. *Virology* 53(1):40-46, 1973.

The 7 S RNA molecules isolated from Rous sarcoma virus (RSV), avian myeloblastosis virus (AMV), mouse sarcoma virus (MSV), and L cell virion (LCV) were treated with RNase T1, and the oligonucleotides produced were fractionated by two-dimensional electrophoresis. The 7 S RNAs from RSV and AMV yield identical fingerprints. Thirty-five oligonucleotides are observed in yields greater than 0.5 mole per mole RNA, and the molecules consist of about 280 nucleotides. LCV and MSV 7 S RNAs show a difference of two oligonucleotides of a total of 37. Comparison of avian and murine 7 S RNAs also shows a large number of similarities, including the 3´ terminus $(U_4C_2)X_{OH}$. The results indicate that large portions of the sequence of 7 S oncornavirus RNAs have been conserved among viruses that replicate in different hosts. Analysis of 7 S RNA from uninfected chick fibroblasts demonstrates that they contain the same molecule as that found in AMV and RSV. The function of the 7 S RNA examined in this study is not known; based on the number of similarities observed in the 7 S RNA from several sources, they probably serve the same function in each case.

1401 GENETIC RECOMBINANTS AND HETEROZYGOTES DERIVED FROM ENDOGENOUS AND EXOGENOUS AVIAN RNA TUMOR VIRUSES. *(E.)* Weiss, R. A. (U. Southern California Sch. Med., Los Angeles), W. S. Mason and P. K. Vogt. *Virology* 52(2):535-552, 1973.

A selective technique based on infective centers is described for isolating host range recombinants of avian RNA tumor viruses. The term recombinant is used to indicate any virus which carries genes derived from two parents, without distinguishing between reassortment of independent genome segments and recombination within one segment. This method was exploited to obtain recombinants between the genome of chick helper factor (*chf*) and nondefective strains of Rous sarcoma virus (RSV), including temperature-sensitive mutants. Recombinants of *chf* with the defective Bryan strain RSV could not be isolated. A majority of nondefective RSV particles selected as carrying the *chf* host range also carried the parental RSV host range; i.e., were of dual host range, but the progeny segregated into parental or recombinant genotypes. These observations suggested that the particles were at least partially diploid or polyploid and represented unstable "heterozygotes." Genotypic mixing was not evident in *chf*-negative cells which contain viral DNA but not viral RNA, suggesting that genetic reassortment occurs among RNA molecules. A model is proposed in which reassortment of independent genome segments may be converted into stable recombinants following provirus formation in the next replicative cycle.

1402 INCORPORATION OF 5-IODO-2´-DEOXYURIDINE (IUdR) INTO SV40 DNA. *(E.)* Buettner, W. (Hygiene Inst., U. Freiburg, West Germany) and H. Werchau. *Virology* 52(2):553-561, 1973.

The effect of various amounts of 5-iodo-2´-deoxyuridine (IUdR) on Simian Virus 40 (SV40) infected African green monkey kidney (AGMK) cells was investigated in respect to: (1) degree of IUdR incorporation into viral DNA, (2) yield of viral DNA, (3) viral coat protein synthesis, (4) infectivity of IUdR-substituted SV40 DNA. Rather than the absolute concentration it is the ratio of IUdR to thymidine (TdR) in the culture medium which determines the effect of the drug, at least in the range between 2.8 x 10^{-5}M and 2.8 x 10^{4}M. The degree of IUdR incorporation into viral DNA was linearly dependent upon the relative amount of IUdR in the TdR plus IUdR precursor mixture added to the culture medium. The amount of viral DNA synthesized decreased exponentially with increasing degrees of IUdR substitution. Coat protein formation as determined by immunofluorescence microscopy behaved similarly. At equal concentrations of IUdR and TdR, when considerable amounts of viral DNA and capsid proteins were still formed, complete virions were assembled. The infectivity of the IUdR substituted viral DNA was greatly reduced. Illumination studies of the IUdR containing SV40 DNA showed that "visible" light induced single strand breaks into this DNA at neutral pH. The lack of production of infectious SV40 virus in cultures treated with IUdR can be attributed to two major effects: (1) suppression of viral DNA synthesis, (2) reduced infectivity of the IUdR-substituted viral DNA that is produced.

1403 TRANSFORMATION OF HAMSTER EMBRYO FIBROBLASTS BY HERPES SIMPLEX VIRUSES TYPE 1 AND TYPE 2. *(E.)* Rapp, F. (Coll. Med., Pennsylvania St. U., Hershey) and R. Duff. *Cancer Res* 33(6):1527-1534, 1973.

Hamster embryo fibroblast cells can be transformed after infection by ultraviolet light-irradiated herpes simplex viruses type 1 (HSV-1) or type 2 (HSV-2). Two of 12 strains of HSV-1 and 7 of 15 strains of HSV-2 induced cell transformation. Transformed cells from two strains of HSV-2 and one strain of HSV-1 have been tested for oncogenicity in newborn Syrian hamsters. One cell line transformed by HSV-2 strain 333 was found to be oncogenic. The remaining two cell lines were not oncogenic. Herpesvirus antigens were found in the cytoplasm and on the surface of the transformed cells. Neutralizing antibodies were produced in hamsters developing tumors after the injection of the HSV-2-transformed cells. Weanling hamsters that were preimmunized by injection of HSV-1 or HSV-2 did not develop transplantation immunity to the HSV-2 transformed cells as a result of this preimmunization. HSV-2 transformed cells metastasized after injection into hamsters. The number of metastases was not inhibited by HSV-1 or HSV-2 immunization but was actually enhanced. Immunization of hamsters with simian virus 40 inhibited metastases but did not reduce the rate of primary tumor development. The fact that HSV-2 transformed cells do not contain the levels of virus-specific transplantation antigens that are found in most cell lines transformed by other DNA viruses coupled with failure to stimulate the immune response of the host animal is probably responsible for the high rate of metastases that is found in HSV-2 tumor-bearing animals.

1404 APPEARANCE OF VIRUS-SPECIFIC RNA, VIRUS PARTICLES, AND CELL SURFACE CHANGES IN CELLS RAPIDLY TRANSFORMED BY THE MURINE SARCOMA VIRUS.

(E.) Salzberg, S. (Saint Louis U. Sch. Med., Mo.), M. S. Robin and M. Green. *Virology* 53(1):186-195, 1973.

The time course of viral RNA synthesis, virus maturation, and cell surface alteration was studied in a clonal line of mouse 3T6 cells infected and transformed by the Harvey strain of the murine sarcoma-leukemia virus complex, H-MSV(MLV). The majority of cells (80%) were infected and gave rise to transformed foci after exposure to 2-3 focus-forming units of virus/cell for 60 min. Most cells became spindle shaped by 24 hr, and virtually all cells assumed a round shape, presumably the final stage of morphological transformation, within 3-4 days. The transcription of virus-specific RNA, as measured by hybridization of RNA from infected cells with the [^{3}H]DNA product of the virion DNA polymerase, was detected at 7 hr after infection, and reached maximal levels at 25 hr. Virus replication in newly infected cells was measured by the appearance in the culture medium of (1) [^{3}H]uridine labeled particles banding at a density of 1.14-1.16 g/ml in sucrose density gradients, (2) particles with RNA and DNA-directed DNA polymerase activity banding at a similar density, and (3) focus forming units. Of the three methods, the most sensitive was the assay for polymerase, which detected the formation of extracellular virus at 14-15 hr after infection. Maximal virus production occurred at about 63 hr. Surface alterations, as revealed by agglutination of cells by concanavalin A, (con A) were detected 22 hr after infection and reached maximal levels at 63 hr. Acquisition of agglutinability by con A appears to parallel the fraction of 3T6 cells with a rounded morphology and the increase in virus production.

1405 CHARACTERIZATION OF TWO CONDITIONAL EARLY MUTANTS OF ROUS SARCOMA VIRUS.

(E.) Linial, M. (U. Southern California Sch. Med., Los Angeles) and W. S. Mason. *Virology* 53(1):258-273, 1973.

Some of the biological and physical properties of two independently isolated mutants of Rous sarcoma virus have been investigated. Both *ts* 335 and *ts* 337 are unable either to transform or replicate at 41 C. Both viral replication and cell transformation, however, are initiated after a short time at 35 C. Once initiation has occurred, replication and transformation can be maintained after a shift to 41 C. The lesions in the mutants occur very early after infection (prior to 2 hr); however, the lesions appear to be expressed after adsorption to and penetration into the host cells. Both mutants contain temperature sensitive structural components, and *ts* 337 is also antigenically different from the wild type. The virions contain a polymerase activity which appears to be temperature sensitive in both the endogenous reaction and with poly rA·d(pT)$_{10}$ as the exogenous template. Upon coinfection of chick fibroblasts with *ts* 335 or *ts* 337 and Rous-associated virus (RAV), genetically stable wild-type sarcoma virus is produced at a high frequency. Therefore, the information for the function(s) which is mutant in these viruses is also present in RAV. Results suggest that the lack of a DNA polymerase activity at the nonpermissive temperature prevents initiation of cell transformation and virol replication but that once initiation occurs, replication and transformation can be maintained.

1406 THE NUCLEIC ACID OF AN SV40-LIKE VIRUS ISOLATED FROM A PATIENT WITH PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY.

(E.) Sack, G. H., Jr. (Johns Hopkins U. Sch. Med., Baltimore, Md.), O. Narayan, K. J. Danna, L. P. Weiner and D. Nathans. *Virology* 51(2):345-350, 1973.

The nucleic acid of an SV40-like virus isolated from a patient with progressive multifocal leukoencephalopathy has been compared with the DNA of a laboratory strain of SV40. DNA of this isolate (designated PML-2) is a covalently closed circular duplex with the same sedimentation rate as SV40 DNA. Denatured PML-2 DNA hybridizes with denatured SV40 DNA at about the same rate as homologous DNA. When digested with *Hemophilus influenzae* restriction endonuclease, PML-2 DNA yielded 11 fragments separable by electrophoresis, nine of which co-migrated with fragments of SV40 DNA, and two of which did not. It is concluded that the PML-2 virus is a variant of SV40.

1407 INTEGRATION OF VIRAL INTO CHROMOSOMAL DEOXYRIBONUCLEIC ACID IN AN INDUCIBLE LINE OF POLYOMA-TRANSFORMED CELLS.

(E.) Manor, H. (Weizmann Inst. Sci., Rehovot, Israel), M. Fogel and L. Sachs. *Virology* 53(1):174-185, 1973.

RNA-DNA hybridization has shown that in polyoma-transformed rat cells which can be induced to synthesize infectious virus (LPT cells), polyoma DNA sequences are associated with chromosomal DNA. RNA complementary to the viral DNA (cRNA) was synthesized *in vitro*, using purified viral DNA as a template and *Escherichia coli* RNA polymerase. High-molecular-weight chromosomal DNA was fractionated from linear and supercoiled viral DNA molecules by centrifugation of whole cells through alkaline glycerol gradients. Hybridization carried out between the cRNA and fractionated chromosomal DNA showed that the amount of RNA hybridized to the LPT DNA was two to three times larger than the amount hybridized to DNA from normal rat cells. cRNA was also hybridized, under the same conditions, with mixtures containing a constant amount of normal cell DNA and varying quantities of purified viral DNA. These assays have established that a linear relationship exists between the amount of cRNA specifically hybridized with a given sample of DNA and the quantity of viral DNA in the sample. Using this relationship, it is estimated that LPT chromosomal DNA contains 6-9 genome-equivalents of polyoma DNA per cell. This quantity represents 18-29% of the amount of polyoma DNA found in the cells, as determined by hybridization of cRNA with unfractionated LPT DNA. To exclude the possibility that the chromosomally associated viral DNA is an artifact due to incomplete removal of the extrachromosomal viral DNA, control experiments were performed in which the cells were superinfected with polyoma virus (m.o.i.-500; 3 hr infection). In these experiments, less than 1% of the viral DNA introduced into the cells by the superinfecting virus were found by the same techniques to be associated with chromosomal DNA. Other experiments show that LPT cells do not contain significant amounts of complex viral DNA molecules which sediment in the vicinity of chromosomal DNA. It is therefore suggested that viral and chromosomal DNA are bound to each other by bonds which cannot be disrupted by alkali treatment.

1408 HOST CELL DNA SYNTHESIS AS A POSSIBLE FACTOR IN THE ENHANCEMENT OF REPLICATION OF HUMAN ADENOVIRUSES IN SIMIAN CELLS BY SV40. *(E.)* Jerkofsky, M. (Coll. Med., Pennsylvania St. U., Hershey) and F. Rapp. *Virology* 51(2):466-473, 1973.

Temperature-sensitive mutants of SV40, ts-701 and ts-702, have been isolated after treatment of wild-type virus with nitrous acid and nitrosoguanidine, respectively. Although neither mutant produces infectious virus at the nonpermissive temperature, both can induce SV40 T and V antigens, virus DNA synthesis, and the stimulation of host cell DNA synthesis. Both are late mutants and appear to be in the same SV40 complementation group. Both mutants can also complement the replication of human adenovirus type 7 at the elevated temperature, as can another late mutant, pm-301, and an early mutant, tsA7. Therefore, the adenovirus complementation step appears to be prior to SV40 virus DNA synthesis. Since a BSC-1 clone in which SV40 infection stimulates host cell DNA synthesis allows the complementation of adenovirus type 7 by SV40 while parental BSC-1 cells neither support complementation of adenovirus type 7 by SV40 nor are stimulated to replicate their DNA after SV40 infection, the stimulation of host cell DNA synthesis appears to be critical to adenovirus enhancement. This stimulation appears to be specific since stimulation of DNA synthesis after nutrient deprivation does not induce the replication of human adenoviruses in simian cells.

1409 EFFECT OF HERPESVIRUS TYPE 2 AND HORMONAL IMBALANCE ON THE UTERINE CERVIX OF THE MOUSE. *(E.)* Muñoz, N. (World Hlth. Organization, Lyon, France). *Cancer Res* 33(6):1504-1508, 1973.

BALB/c mice, two to three months old, were treated with different combinations of hormones (estrogens, progesterone, and Enovid) and herpesvirus type 2 (HSV-2). Characteristic cytological changes of herpesvirus infection were observed in 40% of 140 mice treated with the virus; these mice had previously been immunized with HSV-2 inactivated with ultraviolet light. No significant differences were observed in the frequency of precancerous and cancerous lesions of the cervix and/or vagina between the group treated with estrogens plus HSV-2 (47.2%) and that treated with estrogens alone (41.2%), or between the group treated with Enovid plus HSV-2 (22.6%) and that treated with Enovid alone (31.2%). No carcinomas were observed in either the group treated with progesterone alone or the two control groups, but one carcinoma was found in the group treated with progesterone and HSV-2, and two carcinomas were found in the group treated with HSV-2 alone. These three carcinomas are of special interest since spontaneous cervical carcinoma in mice has not been described, and progesterone has been found to exert a protective effect against chemical carcinogens in the mouse cervix. These results suggest that, at least in these three animals, HSV-2 may have a role in tumor induction.

1410 EVIDENCE FOR RNA TUMOR VIRUSES IN HUMAN LYMPHOMAS INCLUDING BURKITT'S DISEASE. *(E.)* Spiegelman, S. (Coll. Physicians, Surgeons, Columbia U., New York, N.Y.), D. Kufe, R. Hehlmann and W. P. Peters. *Cancer Res* 33(6):1515-1526, 1973.

Human lymphomas (including Burkitt's disease) contain an RNA related in sequence to that of the Rauscher leukemia virus. The viral-related RNA found in these lymphomas is a 70 S component encapsulated with RNA-instructed DNA polymerase in a particle possessing a density characteristic of the RNA tumor viruses. Furthermore, the DNA synthesized by the lymphoma particles hybridizes specifically to the RNA of the Rauscher leukemia virus. The particles identified in 81% of human lymphomas and in 87% of Burkitt's tumors thus contain four features characteristic of a known oncogenic RNA agent. The relation of these RNA particles to the DNA-containing Epstein-Barr virus, an etiological candidate for Burkitt's lymphoma, remains to be established. Both or neither may be necessary, or one may be the true causative agent. Relevant to the ultimate resolution of these issues is the demonstration reported here that the presence of Epstein-Barr virus information in non-neoplastic cells, such as in infectious mononucleosis, is not accompanied by the appearance of RNA particles.

1411 RNA-DIRECTED DNA SYNTHESIS BY VIRIONS OF ROUS SARCOMA VIRUS: FURTHER CHARACTERIZATION OF THE TEMPLATES AND THE EXTENT OF THEIR TRANSCRIPTION. *(E.)* Garapin, A. C. (Dept. Microbiol., U. California, San Francisco), H. E. Varmus, A. J. Faras, W. E. Levinson and J. M. Bishop. *Virology* 52(1):264-274, 1973.

Four species of RNA found in Rous sarcoma virus have been tested for their roles as templates for RNA-directed DNA synthesis by detergent-activated virions of RSV: the high molecular-weight subunits of the 70S RNA complex, 4S and 5S RNAs contained in the

70S complex, and 7S RNA extracted from purified virions. All three forms of low molecular-weight RNA are at least partially transcribed, but the principal templates for DNA synthesis are the high molecular-weight subunits of 70S RNA. DNA synthesized by virions of RSV contains nucleotide sequences representative of most or all of the viral genome, but the majority of these sequences are present in very small proportions. These results further substantiate the occurrence of preferential transcription from a limited region of the viral genome as defined previously for double-stranded enzymatic product by measurement of reassociation kinetics. By contrast, single-stranded DNA synthesized by virions of RSV in the presence of actinomycin D is a complete and relatively uniform copy of the entire viral genome. The latter observation facilitates both the study of nucleic acid homologies among RNA tumor virus genomes and the execution of competition hybridizations to determine the portion of a viral genome present in various populations of cellular RNA.

1412 GROWTH RATE AND ^{59}Fe UPTAKE IN THE PROGENY OF RAUSCHER LEUKEMIA VIRUS-INFECTED SPLENIC COLONY FORMING UNITS FOLLOWING TRANSPLANTATION. *(E.)* Okunewick, J. P. (Allegheny Gen. Hosp., Pittsburgh, Pa.), A. M. Markoe, P. Erhard and E. L. Phillips. *J Lab Clin Med* 81(4):489-496, 1973.

1413 STUDIES OF ROUS SARCOMA VIRUS: EFFECTS OF NUCLEOSIDE ANALOGUES ON VIRUS SYNTHESIS. *(E.)* Brdar, B. (Rockefeller U., New York, N.Y.), D. B. Rifkin and E. Reich. *J Biol Chem* 248(7):2397-2408, 1973.

1414 THE SENSITIVITY OF DJUNGARIAN HAMSTERS TO ROUS VIRUS AND SIMIAN VIRUS 40. *(Rus.)* Senina, E. T. (USSR Acad. Med. Sci., Moscow) and E. E. Pogosyants. *Vopr Onkol* 19(2):46-49, 1973.

1415 ON THE RELATIONSHIP BETWEEN MICE SARCOMA AND ECTROMELIA VIRUSES. *(Rus.)* Yakovleva, L. S. (USSR Acad. Med. Sci., Moscow) and Z. I. Merekalova. *Vopr Onkol* 19(2):42-45, 1973.

1416 AN ANALYSIS OF THE T AND B CELLS OF MAREK'S DISEASE LYMPHOMAS OF THE CHICKEN. *(E.)* Hudson, L. (Middlesex Hosp. Med. Sch., London, England) and L. N. Payne. *Nature [New Biol]* 241(106):53-54, 1973.

1417 STUDIES OF SV40 DNA: V. CONVERSION OF CIRCULAR TO LINEAR SV40 DNA BY RESTRICTION ENDONUCLEASE FROM *ESCHERICHIA COLI B.* *(E.)* Adler, S. P. (Johns Hopkins Sch. Med., Baltimore, Md.) and D. Nathans. *Biochim Biophys Acta* 299(2):177-188, 1973.

1418 THE ISOLATION AND CHARACTERIZATION OF PLASMA MEMBRANE FROM CULTURED CELLS. V. THE CHEMICAL COMPOSITION OF PLASMA MEMBRANES ISOLATED FROM CHICKEN TUMORS INITIATED WITH VIRUS-TRANSFORMED CELLS. *(E.)* Perdue, J. F. (McArdle Lab. Cancer Res., U. Wisconsin, Madison), D. Warner and K. Miller. *Biochim Biophys Acta* 298(4):817-826, 1973.

1419 RADIOPROTECTIVE EFFECT OF RAUSCHER LEUKEMIA VIRUS IN THE SJL/J MOUSE. III. RELATIONSHIP OF HEMATOLOGIC FACTORS AND SPLENOMEGALY TO THE DEVELOPMENT OF RADIORESISTANCE. *(E.)* Markoe, A. M. (Allegheny Gen. Hosp., Pittsburgh, Pa.) and J. P. Okunewick. *Radiat Res* 53(3):428-434, 1973.

1420 CYCLIC-3',5'-AMP-DEPENDENT AND INDEPENDENT PROTEIN KINASE LEVELS IN NORMAL AND FELINE SARCOMA VIRUS TRANSFORMED CELLS. *(E.)* Troy, F. A. (Sch. Med., Comparative Oncol. Lab., U. California, Davis), I. K. Vijay and T. G. Kawakami. *Biochem Biophys Res Commun* 52(1):150-158, 1973.

1421 INHIBITION OF DNA POLYMERASE OF AVIAN MYELOBLASTOSIS VIRUS BY AN ALKALOID EXTRACT FROM *NARCISSUS TAZETTA L.* *(E.)* Papas, T. A. (Natl. Cancer Inst., Bethesda, Md.), L. Sandhaus, M. A. Chirigos and E. Furusawa. *Biochem Biophys Res Commun* 52(1):88-92, 1973.

1422 LEUKEMIA VIRUS ACTIVATION DURING HOMOGRAFT REJECTION. *(E.)* Hirsch, M. S. (Massachusetts Gen. Hosp., Boston), D. A. Ellis, P. H. Black, A. P. Monaco and M. L. Wood. *Science* (4085):500-502, 1973.

1423 LACK OF INFLUENCE OF MITOCHONDRIAL GENETICAL INFORMATION ON THE MULTIPLICATION OF HERPES SIMPLEX VIRUS IN HELA CELLS. *(E.)* Radsak, K. (Hyg. Inst., U. Marburg, West Germany). *Biochem Biophys Res Commun* 51(4):849-857, 1973.

1424 INHIBITION OF RNA SLOW VIRUSES BY THIOSEMICARBAZONES. *(E.)* Haase, A. T. (VA Hosp., San Francisco, Ca.) and W. Levinson. *Biochem Biophys Res Commun* 51(4):875-880, 1973.

1425 ELECTRON MICROSCOPIC OBSERVATIONS OF NEOCARZINOSTATIN TREATED BURKITT LYMPHOMA CELLS. *(E.)* Shamoto, M. (Mercy Hosp., Pittsburgh, Pa.), Y. Kawai and A. Katoh. *Experientia* 29(2):197-198, 1973.

1426 THE FINDING OF VIRAL PARTICLES IN SPONTANEOUS MAMMARY ADENOCARCINOMA IN RATS. *(E.)* Domina, F. (U. Messina, Tumor Res. Ctr., Italy), A. Gimbo and A. Licata. *Experientia* 29(2):200-201, 1973.

1427 ASSESSMENT OF THE RADIOPROTECTIVE CAPACITY OF SPLEEN CELLS TRANSPLANTED FROM MICE WITH RAUSCHER VIRAL LEUKEMIA. *(E.)* Okunewick, J. P. (Allegheny Gen. Hosp., Pittsburgh, Pa.) and P. Erhard. *Radiat Res* 53(2):241-247, 1973.

1428 PLATELET KINETICS AND ADENOSINE TRIPHOSPHATASE ACTIVITY IN AKR MICE. *(E.)* Brodsky, I. (Hahnemann Med. Coll., Hosp., Philadelphia, Pa.) and N. V. Dimitrov. *J Natl Cancer Inst* 50(4):997-1001, 1973.

1429 MIXED VIRAL INFECTIONS WITH ONCOGENIC AND NON-ONCOGENIC VIRUSES IN THE HAMSTER: NOTE I. EXACERBATION OF THE EVOLUTION OF THE TUMOURS IN CONCOMITANT INFECTION OF NEWBORN HAMSTERS WITH SV_{40} VIRUS AND HERPES VIRUS. *(E.)* Nastac, E. (Stefan S. Nicolau Inst. Virol., Bucharest, Romania), M. Hozoc and E. Ursu. *Rev Roum Virol* 9(3):223-231, 1972.

1430 VIRUS ISOLATION FROM HUMAN LARYNGEAL PAPILLOMA. *(E.)* Nastac, E. (Stefan S. Nicolau Inst. Virol., Bucharest, Romania), M. Hozoc, E. Ciufecu, M. Stoian, G. Serban and V. Toma. *Rev Roum Virol* 9(3):233-237, 1972.

1431 ELECTRON MICROSCOPIC EVIDENCE OF THE SUBVIRAL ONCOGENIC PARTICLES (VIROSOMES) ISOLATED FROM THE MITOCHONDRIA OF ROUS SARCOMA CELLS. *(E.)* Kara, J. (Inst. Exp. Biol., Genetics, Czechoslovak Acad. Sci., Prague) and O. Mach. *Folia Biol* 19(1):78-80, 1972.

1432 THE SUBUNITS OF MURINE SARCOMA-LEUKAEMIA VIRUS RNA. *(E.)* McCain, B. (Baylor Coll. Med., Houston, Tex.), N. Biswal and M. Benyesh-Melnick. *J Gen Virol* 18(1):69-74, 1973.

1433 INDUCTION OF ANGIOGENIC SARCOMAS BY SOME MAMMALIAN ADENOVIRUSES. *(E.)* Levenbuk. I. S. (L. A. Tarasevich State Res. Inst. Standardization, Control Med. Biol. Preparations, Moscow, USSR). *Biull Eksp Biol Med* 74(10):1292-1295, 1972.

1434 TUMOUR CELL SURFACE SPECIALIZATION IN THE UPTAKE OF NUTRIENTS EVIDENCED BY CINEMICROGRAPHY AS A PHENOTYPIC CONDITION FOR DENSITY INDEPENDENT GROWTH. *(E.)* Vesely, P. (Inst. Exp. Biol. Genetics, Czechoslovak Acad. Sci., Prague). *Folia Biol* 18(6):395-401, 1972.

1435 AETIOLOGY OF BURKITT'S LYMPHOMA. *(E.)* Williams, E. H. (Internatl. Agency Cancer Res., Arua, Uganda). *Lancet* (7812):1123, 1973.

See also:

* (Rev): 1208, 1211, 1212, 1215, 1217, 1219, 1227
* (Chem): 1280
* (Immun): 1469, 1485, 1486, 1487, 1488, 1489, 1490, 1504, 1508, 1509, 1524, 1530, 1557

1436 ABSENCE OF CELL-MEDIATED IMMUNOLOGIC TOLERANCE TO LEUKEMIA VIRUS IN CARRIER MICE.

(E.) Proffitt, M. R. (Massachusetts Gen. Hosp., Boston), M. S. Hirsch and P. H. Black. *J Immunol* 110(4):1183-1188, 1973.

The hypothesis that chronic virus-carrier states may be a result of virus-specific "split" or partial tolerance involving only the cell-mediated component of the immune response was investigated and shown to be false. A MuLV-M (Moloney murine leukemia virus) carrier-colony was established by injecting neonatal C3H/HeJ mice with a stock pool of MuLV-M strain LTV. In the experiments discussed here, male mice 8 to 12 weeks old were used, before the onset of detectable leukemia. Control mice were of the same strain, sex, and age. Normal mice were immunized once by i.p. and foodpad injections of 0.05 ml of the concentrated MuLV-M diluted 1:10 in phosphate-buffered saline. Control mice were injected with DTMK buffer (0.002 M dithiothreitol; 0.04 M Tris HCl at pH 7.8; 0.15 M $MgAc_2$; 0.06 M KCl) similarly diluted. At various times after the immunization, one mouse each was sacrificed from the immune, carrier, and control groups. The spleen and the popliteal, brachial, axillary, and mediastinal lymph nodes were harvested from each mouse and the lymphocytes prepared for future assay. Results of these experiments indicate that 7 of 9 MuLV-M carrier mice had significant cell-mediated cytotoxic reactivity to syngeneic MuLV-M-infected cells *in vitro*. This indicates that in virus-carrier states, at least with lymphocytic choriomeningitis and murine leukemia virus, neither humoral nor cell-mediated immune tolerance is present. It is suggested that the term immunologic tolerance should no longer be used to describe virus-carrier states. The degree or responsiveness and/or blocking due to antibody or antigen-antibody complexes may prove to be important in these carrier systems.

1437 EFFECTS OF CARRAGEENAN AND HIGH SERUM DILUTION ON SYNERGISTIC CYTOTOXICITY TO TUMOR CELLS.

(E.) Pollack, S. (U. Washington Med. Sch., Seattle) and K. Nelson. *J Immunol* 110(5):1440-1443, 1973.

The theory that synergistic cytotoxicity is actually conventional antibody-dependent killing of target cells with complement produced by the lymphoid cells was tested by use of the complement inhibitor carrageenan and of high serum dilutions. Target tumor cells were obtained from a transplantable Moloney sarcoma induced and carried in A/Sn mice. Sera were obtained by bleeding from the retroorbital sinus of BALB/c mice in which tumors had been induced by intramuscular injection of 0.05 ml of a 1:7 dilution of stock Moloney sarcoma virus. The cytotoxicity produced by treatment with serum from tumor-bearing animals and normal rabbit serum (NRS) as a source of complement is completely abolished in the presence of 200 μg/ml carrageenan. This concentration of carrageenan had no apparent deleterious effect on the target cells. In another experiment, the sera were diluted far beyond the range where complement-dependent lysis can be shown to occur. Synergistic cytotoxicity was observed at dilutions of 1:1280 and 1:2560 in two different sera. In neither case has an end point been reached. Complement-dependent cytotoxicity, however, was not detectable beyond a dilution of 1:320. There was a range of low dilutions in each serum in which synergistic cytotoxicity did not occur. As a result of the evidence from these two lines of experiments, it is concluded that conventional complement-dependent cytotoxicity does not play a role in synergistic cytotoxicity to tumor cells.

1438 A RABBIT ANTISERUM TO A THYMUS EXTRACT SPECIFIC FOR MOUSE THYMUS-DERIVED CELLS.

(E.) Sarkar, S. (Salk Inst., San Diego, Calif.), R. Hyman, T. Masuda and L. A. Herzenberg. *J Immunol* 110(5):1222-1232, 1973.

A heteroantiserum has been prepared in rabbits against an EDTA eluate of female BALB/c thymus cells. The globulin fraction of unabsorbed serum precipitated macromolecules of various molecular weights which were eluted from several AKR and BALB/c lymphomas. Unabsorbed heteroantiserum was cytotoxic to both Thy-1 (θ)-positive lymphomas and normal thymus cells, but was also toxic to 10-15% of bone marrow cells. The serum was made highly specific for mouse thymus-derived cells by absorption with Sepharose 4B immunoabsorbent coupled with C57BL serum and bone marrow cells. Indirect immunofluorescence of thymus cells from C57BL mice assessed by rapid-flow fluorescence showed a single, sharp distribution; the activity of thymus-derived cells was specifically removed from primed spleen cells by absorbed serum plus complement when tested in an *in vivo* transfer system. The antiserum did not discriminate between the different Thy-1 (θ) alleles.

1439 LONG-TERM SPONTANEOUS TUMOR INCIDENCE IN NEONATALLY THYMECTOMIZED MICE.

(E.) Sanford, B. H. (Massachusetts Gen. Hosp., Boston), H. I. Kohn, J. J. Daly and S. F. Soo. *J Immunol* 110(5):1437-1439, 1973.

The long-term incidence of spontaneous tumors after neonatal thymectomy in mice was investigated. BALB/c mice were thymectomized within 48 to 72 hr after birth. Sham-thymectomies involved surgical exposure of the thymus under similar conditions. Littermates of the same sex were paired and after weaning, pairs of thymectomized and sham-thymectomized mice were maintained together in cages for 2 for the duration of their lives. Forty-six pairs of mice were entered into the experiment, along with 13 extra non-paired mice from the same litters. In 17 cases one or both members of a pair were found dead in condition unsuitable for pathologic study. As a result, at the end of the study, there was gross and histologic data on 58 mice from 29 intact pairs and an additional 19 non-paired subjects. Survival time did not differ significantly between the 2 groups; neither did the overall tumor incidence, which was approximately 80%. On the other hand, the results do suggest some differences at more subtle levels. There was, for instance, a decreased incidence of lung and mammary tumors and a greater variety of types of tumors in the thymectomized group. The data provide an example of how overall spontaneous tumor incidence may remain the same while the distribution of tumor types and behavior of particular types of tumors may be altered by neonatal thymectomy.

1440 RABBIT ANTISERUM TO BRAIN-ASSOCIATED THYMUS ANTIGENS OF MOUSE AND RAT. I. DEMONSTRATION OF ANTIBODIES CROSS-REACTING TO T CELLS OF BOTH SPECIES. *(E.)* Peter, H. H. (Scripps Clin. Res. Fdn., La Jolla, Calif.), J. Clagett, J. D. Feldman and W. O. Weigel. *J Immunol* 110(4):1077-1084, 1973.

Antisera which, upon appropriate absorptions, are T cell-specific, were prepared following immunization of rabbits with either mouse (CBA/ST) or rat (le) brain homogenates. Results from *in vitro* complement-dependent cytotoxicity and direct immunofluorescence tests before and after absorption of anti-mouse brain (AMB) antisera with mouse or rat thymus or brain showed that mouse and rat T cell theta (θ) alloantigens were cross-reacting. AMB also reacted with thymocytes from Bn, ACI and Buffalo strain rats, but not with guinea pig thymus or bone marrow cells. Further evidence for cross-reactivity and for the specificity of anti-brain anti-sera for T cells was obtained from experiments in which AMB, in the presence of complement, abolished cell-mediated immunity *in vitro*, but did not affect antibody-forming cells.

1441 GROWTH OF SYNGENEIC TUMOURS IN UNIMMUNIZED NEWBORN AND ADULT HOSTS. *(E.)* Forni, G. (U. Torino, Sch. Med., Italy) and P. M. Comoglio. *Br J Cancer* 27(2):120-127, 1973.

The post-natal development of "natural" resistance of Balb/c mice to the challenge of syngeneic tumors was studied using s.c. injections of various doses of live plasmacytoma or adenocarcinoma cells into untreated animals of increasing age, from neonatal to 12 wk. The minimum number of neoplastic cells necessary to induce tumor growth increased in parallel with the age of the animals. Whole body x-irradiation reduced the natural resistance of adult mice to tumor challenge to a level identical with that of the neonates. The rate of tumor growth following inoculation of a given number of tumor cells was slower in untreated adult mice than in neonates.

1442 A RADIOIMMUNOASSAY FOR MTV-ANTIGENS. *(E.)* Verstraeten, A. A. (Netherlands Cancer Inst., Amsterdam), Ph.C. Hageman and H. G. Kwa. *Eur J Cancer* 9(2):155-157, 1973.

A quick, sensitive radioimmunoassay has been developed to detect mammary tumor virus (MTV)-specific antigens. The assay measures the degree of inhibition of binding of known ^{125}I-labeled MTV antigens to rabbit anti-MTV antiserum preabsorbed to a sample of suspect plasma, milk or tumor extract. The assay detects two separate MTV antigens, one thought to be viral coat antigen and the other, viral nucleoid antigen. The assay is specific as no reaction occurs with plasma and milk samples from non-tumor-bearing Balb/c mice or with Rauscher leukemia virus extract. The assay is 10 to 300 times more sensitive in detecting the two MTV antigens than is the micro-Ouchterlony double-diffusion technique.

1443 THE PRESENCE OF J-CHAINS IN THE INTRACELLULAR FLUID OF IgM-SECRETING MURINE TUMOR CELLS. *(E.)* Raam, S. V. (Dept. Microbiol., U. Georgia, Athens) and F. P. Inman. *J Immunol* 110(4):1044-1050, 1973.

Intracellular fluids prepared by centrifuging lysed cells from IgM secreting HPC 76 or IgA secreting MOPC 315 mouse tumors were analyzed by polyacrylamide gel electrophoresis for the presence of J-chain. Intracellular fluid from both tumors, when electrophoresed, showed a rapidly migrating band which corresponded in position to J-chain marker derived from sulfonated IgM and sulfonated IgA. Intracellular J-chains were also demonstrated by selective precipitation with anti-J-chain antiserum prepared in rabbits. In addition, J-chain was identified in extracellular fluid (i.e., dialyzed incubation medium) of both HPC 76 and MOPC 315 tumor cells by serologic and electrophoretic methods.

1444 RABBIT ANTISERUM TO BRAIN-ASSOCIATED THYMUS ANTIGENS OF MOUSE AND RAT. II. ANALYSIS OF SPECIES-SPECIFIC AND CROSS-REACTING ANTIBODIES. *(E.)* Clagett, J. (Scripps Clin. Res. Fdn., LaJolla, Calif.), H.-H. Peter, J. D. Feldman and W. O. Weigle. *J Immunol* 110(4):1085-1089, 1973.

A detailed immunologic analysis was conducted on species-specific and the cross-reacting T cell-specific antibodies in anti-mouse brain (AMB) and anti-rat brain (ARB) antibodies prepared from sera of rabbits immunized with mouse or rat brain homogenates. In addition to the cross-reacting T cell specific anti-θ anti-bodies previously reported, ARB and AMB contained non-cross-reacting species-specific markers (designated 'R' and 'M', resp.) which were detected in the complement-dependent cytotoxicity test and which were not absorbed by mouse or rat thymus or brain, resp. In addition, the presence of a shared antigenic determinant (designated 'H') was indicated by experiments in which absorption of AMB with thymus or brain tissues from other inbred mouse strains regardless of their θ phenotype (or of analogous experiments with ARB) abolished cytotoxicity for θC3H and θAKR mouse and for Lewis rat target cells. The tissue distribution of the species-specific M and R markers and of the shared and cross-reacting H and θ antigens was identical.

1445 CELL SURFACE ANTIGEN MOVEMENT: INDUCTION IN HEPATOMA CELLS BY ANTITUMOR ANTIBODY.

(E.) Leonard, E. J. (Natl. Cancer Inst., Bethesda, Md.). *J. Immunol* 110(4):1167-1169, 1973.

The movement of tumor-specific antigenic sites on the surface of an ascitic variety of diethylnitrosamine-induced guinea pig hepatoma cells has been studied *in vitro*. Tumor cells were removed from animals, equilibrated in the cold with rabbit antibody against tumor-specific antigen, exposed to fluorescein-conjugated anti-rabbit serum, and incubated at 37 C in culture medium. Cells initially showed a peripheral, ring-shaped fluorescence which gradually changed over a 3 hr period to multiple, small unevenly distributed aggregates. The number and intensity of staining of the aggregates appeared to decrease 1-4 hr later. These findings were interpreted as coalescence of the fluorescein-conjugated molecules in the plane of the cell membrane, followed by extrusion of the aggregates into the culture medium. Movement of the fluorescein conjugate was not seen in non-viable cells nor was it observed to any significant extent in cells incubated in phosphate-buffered saline instead of culture medium. Successive application of rabbit antitumor antibody and fluorescein conjugate was required to trigger the aggregation response.

1446 MACROPHAGE NONIMMUNOLOGIC RECOGNITION: TARGET CELL FACTORS RELATED TO CONTACT INHIBITION.

(E.) Hibbs, J. B., Jr. (VA Hosp., Salt Lake City, Utah). *Science* 180(4088):868-870, 1973.

Studies were conducted on the effects of activated macrophages (obtained from *Toxoplasma gondii* or BCG infected C3H/He or Balb/c mice) on cultured Balb/c 3T3, Balb/c 3T12 and SV40-transformed 3T3 (SV-3T3) cells. 3T3 cells are contact-inhibited and non-tumorigenic whereas 3T12 cells are non-contact-inhibited and tumorigenic. Cytotoxicity tests showed that syngeneic and allogeneic activated macrophages destroyed 3T12 and SV3T3 fibroblasts but had no effect on 3T3 cells. Also, activated macrophages were not cytotoxic to secondary mouse kidney cells but were cytotoxic to EMT-6 mouse mammary adenocarcinoma cells. Non-activated macrophages had no effect on either contact-inhibited or non-contact-inhibited cells. These results suggested that the abnormal growth characteristic important in non-immunologic macrophage recognition was a target cell surface membrane modification associated with loss of contact inhibition.

1447 STRUCTURAL PROPERTIES OF THE LIGAND BINDING SITES OF MURINE MYELOMA PROTEINS.

(E.) Hsia, J. C. (Dept. Pharmacol., U. Toronto, Canada) and J. R. Little. *FEBS Letters* 31(1):80-84, 1973.

The mobility of two spin labeled dinitrophenol (DNP) ligands which are approximately the same size, but different in their configuration, has been studied by electron spin resonance using two immunospecifically purified mouse A-myeloma proteins (protein-315 and protein-460). The two myeloma proteins each bound the DNP hydrazone derivative of 1-oxyl-2,2,5,5-tetramethyl-3-pyrrolidone (I) and N-[1-oxyl-2,2,5,5-tetramethyl-3-methyl aminopyrrolidinyl]2,4-dinitrobenzene (II) with nearly identical affinities. Whereas the protein-315 binding site immobilized both I and II, the protein-460 binding site could only immobilize I. These results indicated that the active site of the higher affinity protein-315 was more flexible than that of protein-460. Although it was not clear whether the CNP portion of each ligand had a similar or identical steric relationship with the active site of protein-460, it was found that the spin label portion located at a fixed distance from the CNP group was making extensive contact, in the case of I, or very little contact, in the case of II, with the active site.

1448 THE TRANSPLANTATION KINETICS OF TUMOUR CELLS.

(E.) Porter, E. H. (Belvidere Hosp., Glasgow, Scotland), H. B. Hewitt and E. R. Blake. *Br J Cancer* 27(1):55-62, 1973.

The *in vitro* growth characteristics of serial dilutions of cells from five spontaneously arising mouse tumors have been analyzed statistically to demonstrate how assay results can be tested for consistency with "single-cell" transplantation kinetics. "Single-cell" transplantation kinetics occur when three criteria are met: 1) Some of the tumor cells are clonogenic; 2) An inoculum produces a take if, and only if, it contains at least one clonogenic cell; and 3) Any non-clonogens in the inoculum have no influence on whether or not a tumor takes. Single cell transplantation kinetics is the simplest type and is exemplified by such experimental tumors as CBA 'NT' carcinoma and WHT squamous cell carcinoma 'D'. Other tumors (e.g. WHT bone sarcoma and CBA sarcoma 'F') consistently transplant with anomalous kinetics (i.e., other than single-cell). The mechanism by which these anomalous tumors depart from single-cell behavior is unknown.

1449 A COMMON TUMOUR SPECIFIC ANTIGEN. I. RESTRICTION *IN VIVO* TO MALIGNANT NEOPLASTIC TISSUE.

(E.) Dickinson, J. P. (Newcastle Gen. Hosp., England), E. A. Caspary and E. J. Field. *Br J Cancer* 27(2):99-105, 1973.

The antigenic activity of basic protein extracts of various human tissues toward sensitized lymphocytes from cancer patients was studied using the macrophage electrophoretic migration method. The antigenic properties of basic proteins isolated from human malignant colon, stomach, bronchus and breast tumor tissues differed from those of basic proteins prepared from normal tissue, hyperplastic tissue and normal tissue from tumor-bearing organs. These tumor-type antigenic molecules were at least 10^4

times less numerous on normal than on tumor cells and were absent also from hyperplastic tissues. Chronic lymphatic leukemia cells possessed a cancer antigen with the same qualitative properties as that obtained from solid tumors. This antigenic activity was not shared by normal lymphocytes.

1450 LOCAL IMMUNOGLOBULIN PRODUCTION IN BREAST CANCER. *(E.)* Roberts, M. M. (Dept. Clin. Surg. U. Edinburgh, Scotland), E. M. Bass, I. W. J. Wallace and A. Stevenson. *Br J Cancer* 27(4):269-275, 1973.

IgA, IgG and IgM levels were estimated by the radial immunodiffusion technique in protein extracts of 55 malignant (stage I and II) and 20 benign human tumors of the breast, and in 17 normal tissues from a cancer bearing breast. IgA and IgG levels were significantly lower in cancer tissues than in either benign tumor or normal breast tissues. IgM, which was detected in only 19 of the 55 malignant tumors, 6 of the 17 normal tissues and 4 of the 20 benign tumors, showed higher levels in the malignant tumors than in the benign tumors or normal tissues. The tissue IgM fraction was higher in 6 cancers than in the serum from the same patient. This was never the case for benign or normal tissues. Total immunoglobulin and IgG levels were significantly higher in malignant tumors with a marked plasma cell or round cell infiltrate than in tumors with a minimal infiltrate. The menstrual status of the patient had no effect on these observations.

1451 FOETAL ANTIGENS AND CANCER. *(E.)* Castro, J. E. (Clin. Res. Ctr., Harrow, England), E. M. Lance, P. B. Medawar, J. Zanelli and R. Hunt. *Nature* 243(5404):225-225, 1973.

The growth of fetal tissues was studied following implantation under the kidney capsule of adult CBA or C57 male mice. Implants of both syngeneic and allogeneic fetal tissues grew poorly in normal adult mice. Moderate lymphocytic infiltration was observed in each case. In contrast, adult syngeneic tissue homogenates grew well under the same conditions with no lymphocytic infiltration. Fetal implants grew extremely well in thymectomized, lethally irradiated, bone marrow reconstituted ("deprived") syngeneic mice and in normal mice which had received a previous implant of syngeneic fetal tissue. Unexpectedly, previous treatment of adult mice with syngeneic fetal tissue shortened the latent period of 20-methylcholanthrene induced tumors. Previous treatment with fetal implants also enhanced the growth of a transplantable syngeneic tumor in normal adult Balb/c mice. These results suggested that syngeneic fetal antigens may provoke a transplantation reaction in immunocompetent adult mice.

1452 ANTIGEN-INDUCED DEPRESSION OF DNA SYNTHESIS IN MOUSE SPLEEN. *(E.)* Zatz, M. M. (Albert Einstein Coll. Med., Bronx, N.Y.) and A. L. Goldstein. *J Immunol* 110(5):1312-1317, 1973.

The effect of prior i.v. administration of 5 x 10^8 sheep erythrocytes (SRBC), 0.2 ml of a standard suspension of *S. typhi*-agglutinable H antigen, or 200 µg keyhole limpet hemocyanin on incorporation of injected ^{125}I-iododeoxyuridine (^{125}I-IUDR) into spleen cell DNA of CBA/J mice was studied. All three antigens reduced ^{125}I-IUDR incorporation to values ranging from 15 to 54% of that in the saline injected controls. Maximal depression of ^{125}I-IUDR incorporation occurred when antigen injection preceeded ^{125}I-IUDR injection by six hours. Antigen-induced depression of ^{125}I-IUDR incorporation into spleen cell DNA was not affected by adrenalectomy, but was abolished four weeks after thymectomy of adult animals. Antigen induced depression of ^{125}I-IUDR incorporation was not affected by two thymus-independent antigens, PPS II and PVP. These results suggested that antigen-induced depression of ^{125}I-IUDR incorporation into spleen cell DNA was most likely occurring in a nonspecific cell population but was mediated by the release of inhibitory factors from surrounding antigen-active T-dependent cells.

1453 RECEPTOR MOBILITY AND RECEPTOR-CYTOPLASMIC INTERACTIONS IN LYMPHOCYTES. *(E.)* Edelman, G. M. (Rockefeller U., New York, N.Y.), I. Yahara and J. L. Wang. *Proc Natl Acad Sci* 70(5):1442-1446, 1973.

Experimental findings from the literature have been correlated to formulate a hypothesis concerning cell membrane receptor-cytoplasmic interactions. Based primarily on studies of the inhibition by concanavalin A (conA) of lymphocyte surface receptor mobility, it has been proposed that binding of multivalent lectins alters the interaction of an assembly of colchicine-binding proteins with lectin receptors and other receptors, and reciprocally that the state of the colchicine-binding assembly alters the mobility and distribution of surface receptors on the cell membrane. Support for this hypothesis has come from experiments in which colchicine, colcemid, vinblastine and vincristine were found to reverse partially the effect of con A on receptor mobility and in which colchicine and vinblastine inhibited the agglutination of fibroblasts with neutrophils by con A. The hypothesis requires that some of the cell surface receptors are anchored on, in, or under the lymphocyte plasma membrane. The bulk of evidence favors a cytoplasmic origin for these receptors which may be related to actin-like proteins or microtubules. Constantly changing mobility of these receptors would provide a mechanism whereby external stimuli may be transmitted to the interior of the cell to elicit such responses as contact inhibition, mitosis or cellular motility.

1454 THE DEVELOPMENT AND SPECIFIC SUPPRESSION OF CONCOMITANT IMMUNITY IN TWO SYNGENEIC TUMOUR-HOST SYSTEMS. *(E.)* Chandradasa, K. D. (Sub-Dept. Immunology, U. Liverpool, England). *Int J Cancer* 11(3):648-662, 1973.

Immunological resistance, concomitant with tumour growth, against two methyl-cholanthrene-induced murine sarcomas MC23 and MC6 differing widely in their immunogenicities, was studied in inbred C57BL and BALB/c strains of mice which are syngeneic with the respective tumors. The tumors were induced in C57BL male, BALB/c female and male mice by s.c. injection of 5 mg of 20 methylcholanthrene in 0.1 ml olive oil. Tumors formed within five months. It was found that, initially, immune responses to specific transplantation antigens resulted in a peak of antitumor resistance at 10-14 days after the implantation of the tumors. In the second phase of tumor growth a rapid decline of the resistance was observed. The peak at 10-14 days was followed by a greater than 50% loss of resistance to reinoculation with the tumor cells, 1 wk later. Further decline of resistance in the MC6-BALB/c system was followed by the acceptance and progressive growth of over-threshold doses of tumor cells. A comparable observation could not be made in the immunogenically weaker MC23-C57BL system where further loss of resistance was gradual. The above changes in the development and decline of anti-tumor resistance were broadly correlated with the progress of the immunoblast reaction of the paracortical areas of regional lymph nodes. Changes of anti-tumor resistance in the MC6-BALB/c system were found to be associated with corresponding changes in the availability and/or effectiveness of specifically cytotoxic lymphoid cells, in the lymphoid organs as well as a change in the quality of the serum factors from weakly inhibitory to mildly enhancing, in the tumor. Specifically cytotoxic lymphoid cells could not be demonstrated in the MC23-C57BL system by the lymphocyte neutralization technique employed, possibly due to the involvement of *in vivo* blocking phenomena. Accordingly, sera of these mice proved to be tumor-enhancing throughout the period of tumor growth. It is suggested that the progressive growth of the tumors is facilitated by the specific suppression of cell-mediated immunity by the "efferent" as well as by the "afferent" mechanisms and is brought about by a factor or factors--possibly antibody and/or antigen-antibody complexes--present in the serum of the tumor-bearing mice.

1455 ISOLATION AND PARTIAL CHEMICAL CHARACTERIZATION OF CELL-SURFACE GLYCOPEPTIDES FROM AS-30D RAT HEPATOMA WHICH POSSESS BINDING SITES FOR WHEAT GERM AGGLUTININ AND CONCANAVALIN A. *(E.)* Smith, D. F. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), G. Neri and E. F. Walborg, Jr. *Biochemistry* 12(11):2111-2117, 1973.

Studies were conducted to determine if a correlation existed between the wheat germ agglutinin (WGA)- and concanavalin A (Con A)-binding activities of glycopeptides isolated from tumor cell surface and the cyto-agglutination properties of the intact tumor cell. AS-30D rat ascites hepatoma cells, which are agglutinable by WGA, but only weakly agglutinable by high concentrations of Con A, were used. Treatment of the intact cells with papain rendered them highly agglutinable by both lectins and released a glycopeptide fraction from the cell surface. The cell-surface glycopeptide fraction inhibited the cytoagglutination of AS-30D cells by WGA, indicating that this fraction contained components that possessed the minimum requirements of cell-surface binding sites for WGA. The cell-surface glycopeptide fraction exhibited both WGA- and Con A-hemagglutination inhibitory activity. Partial resolution of the glycopeptides by gel filtration and ion-exchange chromatography resulted in the isolation of a sialoglycopeptide fraction, which represented 12% of the weight of the papain-labile, cell-surface glycopeptides and possessed the major portion of the WGA-binding activity. This glycopeptide fraction exhibited specific hemagglutination inhibitory activities which reflected the cytoagglutination properties of the intact AS-30D cell. Purification of Con A-binding activity from the papain-labile, cell-surface glycopeptides resulted in the isolation of a sialoglycopeptide fraction which possessed only Con A-hemagglutination inhibitory activity. These findings suggest that plasma membrane glycoproteins may play an important role in the cytoagglutination of AS-30D tumor cells by WGA. The necessity to investigate the phytoagglutination-binding properties of protease-labile, cell-surface glycopeptides from other tumor cells with differing cytoagglutination properties and from normal cells is emphasized.

1456 AUTO-IMMUNIZATION WITH ACUTE LEUKEMIA CELLS: DEMONSTRATION OF INCREASED LYMPHOCYTE RESPONSIVENESS. *(E.)* Gutterman, J. U. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), G. Mavligit, K. B. McCredie, E. J. Freireich and E. M. Hersh. *Int J Cancer* 11(3):521-526, 1973.

Factors which will be important in active immunotherapy programs for acute leukemia were examined. Ten patients with adult acute leukemia were autoimmunized with 10^8 and/or 10^6 non-irradiated leukemia cells during remission. Six of nine receiving 10^8 cells demonstrated a 100% or greater increase in *in vivo* lymphocyte blastogenic response to their own leukemia cells measured 7 days after immunization. In four responders, increased blastogenesis was noted only in autologous serum, but not in allogeneic serum. Only one of four demonstrated increased lymphocyte blastogenic response in autologous serum after immunization with 10^6 cells. The increased immune response was always accompanied by significantly increased blastogenic response to phytohemagglutinin (PHA) and/or Streptolysin-O (SLO). Three patients studied 28-42 days following immunization showed a persistent increase in lymphocyte blastogenic response to leukemia cells, but the response to PHA and SLO had returned to pre-immunization levels. The study suggests that 10^8 non-irradiated leukemia cells are more effective than 10^6 cells in increasing the cell-mediated immune response of patients to their own leukemia cells. It also suggests that autoimmunization may change the serum of leukemia patients in some way to account for the increased lymphocyte reactivity *in vitro*.

1457 DEPRESSION OF COMPLEMENT ACTIVITY IN THREE STRAINS OF MICE AFTER TUMOR TRANSFER. *(E.)*

Drake, W. P. (Baltimore Cancer Res. Ctr., Md.), S. M. LeGendre and M. R. Mardiney, Jr. *Int J Cancer* 11(3):719-724, 1973.

Both immune adherence and hemolytic complement activity were measured as a function of time after either i.p. or s.c. injection of six transplantable tumors carried in three strains of mice. BW10232, a spontaneous adenocarcinoma, and C1498, a spontaneous myeloid leukemia, were carried in C57BL/6 mice. Harding Passey, a spontaneous melanoma, and S180, a spontaneous pleomorphic-cell sarcoma, were carried in BALB/c mice. C3HBA, a spontaneous adenocarcinoma, and 6C3HED, an induced lymphosarcoma, were carried in C3H/He mice. There was a lack of correlation between complement activity measured by these assays in accord with other studies on human cancer patients. The data indicate that host complement activity was depressed in all cases following tumor transfer and suggest that the serotherapy of murine tumors using passively administered anti-tumor serum may be compromised by host complement insufficient to mediate tumor cytolysis *in vivo*.

1458 ESTABLISHED CELL LINE OF URINARY BLADDER CARCINOMA (T24) CONTAINING TUMOR-SPECIFIC ANTIGEN.

(E.) Bubenik, J. (Inst. Exp. Biol., Genetics, Czechoslovak Acad. Sci., Prague), M. Baresova, V. Viklicky, J. Jakoubkova, H. Sainerova and J. Donner. *Int J Cancer* 11(3):765-773, 1973.

A cell line derived from human urinary bladder carcinoma, designated T24, was established *in vitro* and immunological tests for bladder tumor-specific antigen (TSA) performed. The growth of T24 cells in tissue culture was characterized by a disorderly pattern of growth in one or more layers and by mixed epithelioid-fibroblastoid morphology. The generation time of T24 cells was 19 hr. The cells had a hypotetraploid stemline with marker chromosomes. The malignant character of the line was verified by inoculation of cell suspension into hamster cheek pouch. The presence of TSA in T24 cells was demonstrated by a micromodification of the cytotoxicity test with autochthonous leukocytes and serum from the donor of the tumor. The TSA was also well detectable by the reaction with allogeneic leukocytes from tumor-bearing patients and persisted during a 30-month culture period. The permanent presence of TSA in this carcinoma cell line suggests that the TSA is a genetically determined characteristic of T24 cells.

1459 CELL SURFACE ANTIGENIC CHANGES INDUCED IN NORMAL ADULT RAT LIVER CELLS BY CARCINOGEN TREATMENT *IN VITRO*.

(E.) Iype, P. T. (Paterson Lab., Manchester England), R. W. Baldwin and D. Glaves. *Br J Cancer* 27(2):128-133, 1973.

Antigenic changes in normal rat liver cell lines treated with the chemical carcinogen N-methyl-N-nitrosourea (MNU) were examined. These cells elicited antibody production (detected by membrane immunofluorescence test *in vitro*) when injected into a highly inbred strain of rats from which the liver cells were originally isolated. In contrast, the control cells which were untreated did not evoke humoral antibodies. Antisera raised against the MNU-treated cells reacted not only with the immunizing cells, but also with 3'-methyl-4-dimethylaminoazobenzene and 3-methylcholanthrene-treated cells. However, this antiserum failed to react with cells treated with either aflatoxin or N-acetoxy-2-acetyl-aminofluorene. Embryonic antigens were found to be absent from the normal adult liver cell line. Preliminary results indicated that none could be detected on carcinogen-treated cells either. Two important points arise from these experiments: 1) three different carcinogens induce an apparently similar antigenic change; 2) the change(s) could be detected as early as 4 wk after carcinogen treatment, at which time no other indication of transformation was observed.

1460 THE FREQUENCY OF M-COMPONENTS IN SERA OF PATIENTS WITH SOLID MALIGNANT NEOPLASMS.

(E.) Talerman, A. (Inst. Radiotherap., Rotterdam, Holland) and W. G. Haije. *Br J Cancer* 27(4):276-282, 1973.

The frequency of M-components, monoclonal immunoglobulins, was studied by agar gel electrophoresis in sera from 807 patients, 467 (57%) females and 340 (43%) males with histologically proven solid malignant neoplasms. M-components were found in the sera of 40 male and 20 female patients. Apart from two known cases of multiple myeloma and one case of Waldenstrom's macroglobulinemia, none of the patients were found to be suffering from these diseases. The frequency of M-components increased with age; this was more evident in males. Twenty-two of 60 patients with M-components did not exhibit abnormalities on immunoelectrophoresis. Of the 35 remaining patients, 27 had an abnormal component of the IgG class, 6 of the IgA and 2 of the IgM class. M-components were found in the sera of patients with a wide variety of neoplasms. There appeared to be no evidence of an increased frequency of M-components in the sera of patients with solid malignant neoplasms compared with normal adult population.

1461 IMMUNOLOGICAL STUDIES IN ACUTE MYELOID LEUKAEMIA: PHA RESPONSIVENESS AND SERUM INHIBITORY FACTORS.

(E.) Walker, J. S. (St. Mary's Hosp., Manchester, England), D. Davis, P. Davies, C. B. Freeman and R. Harris. *Br J Cancer* 27(3):203-211, 1973.

The effect of serum from patients with acute myeloid leukemia (AML) on the lymphocyte response to phytohemagglutinin (PHA) was examined. Sera from 16 of 20 patients with AML at some stage of the disease inhibited the *in vitro* PHA transformation of normal lymphocytes assessed by measuring the rate of DNA synthesis after 67-70 hr; 42% of pretreatment sera were inhibitory. Inhibitory activity was overcome at PHA concentrations 2-3 times greater than the concentration which allowed maximum discrimination between normal human serum and leukemia sera. PHA transformation of washed lymphocytes obtained from AML patients before treatment and when receiving induction or consolidation (cytoreductive) chemotherapy was reduced only when cultures contained a high proportion of primitive cells. Even in primitive cell contaminated cultures significant responses to PHA could be measured if conditions were modified to prevent increasing acidity. Reports of reduced *in vitro* immunological reactions in pretreatment and poor prognosis patients may therefore be due to the presence of primitive cells in culture, and in treated patients to the failure of chemotherapy to reduce the circulating primitive cell count. Serum inhibitory factors may have a significant immunosuppressive effect *in vivo*, but the accurate assessment of the role of immune mechanisms in AML should attempt the measurement of specific immunity.

1462 INFLUENCE OF CULTIVATION, TRYPSINIZATION AND AGGREGATION ON THE TRANSPLANTABILITY OF MELANOMA B16 CELLS. *(E.)* Hagmar, B. (Dept. I Path., U. Goteborg, Sweden) and K. Norrby. *Int J Cancer* 11(3):663-675, 1973.

The usefulness of tumor cells grown *in vitro* for transplantation studies was examined in Melanoma B16 grown in monolayer culture. Three types of suspensions were prepared from the cell cultures: two virtually monocellular suspensions, one produced by mechanical procedures and Versene (CV), the other with the addition of trypsinization (CTV); a third suspension containing cell aggregates (CVA) was produced by centrifuging a CV suspension. For comparison, a well-dissociated suspension of solid tumor origin was prepared by a trypsin-Versene procedure (STV). All suspensions were characterized with regard to dissociation grade, cell volumes and cellular electrophoretic mobility and were transplanted in graded cell doses s.c., i.p., and i.v. While inoculum-doses needed for progressive growth were lower i.p. than s.c. the transplantabilities were similar in these sites for all suspensions, indicating similar viabilities and absence of major dilution errors. The growth patterns for i.v. infused cells exhibited significant differences between the suspensions. CV cells gave rise to lung tumors almost exclusively, while CVA animals, in addition to a large total lung tumor volume, had some extrapulmonary takes. CTV had the greatest number of extrapulmonary tumors, but in combination with small lung tumors, STV cell recipients were intermediate with respect to pulmonary as well as extrapulmonary tumors. These observations were collated with data regarding aggregation, cell volumes and electrophoretic mobility. It was concluded that (1) cultivated tumor cells can have similar transplantation characteristics to the tumor of origin; (2) such cells may be useful in tumor dissemination studies; (3) trypsinization as well as the presence of cell aggregates affects the growth pattern of i.v. infused tumor cells by yielding on increased extrapulmonary tumor crop.

1463 CARCINOEMBRYONIC ANTIGEN (CEA): RADIOIMMUNOASSAY USING HIGHLY PURIFIED CEA AND ^{125}I CEA. *(E.)* Smith, H. J. (Hlth. Central, Inc., Minneapolis, Minn.), P. H. Figard, P. J. O'Neill and M. Gökcen. *Res Commun Chem Path Pharm* 5(3):573-583, 1973.

Carcinoembryonic antigen (CEA) was purified from metastatic colon cancer nodules from a blood group O patient by Sephadex G-200 and Sepharose 4B column chromatography followed by electrophoresis and affinity chromatography. With this procedure, 385 g of tissue yielded 10 mg purified CEA which was free of blood group antigen. Purified CEA and I-labeled CEA were used to develop a radioimmunoassay which employed monospecific anti-CEA antiserum. The assay could detect 0.5-32 ng CEA/ml serum or plasma and was highly reproducible. Whereas normal controls had circulating plasma CEA levels of 0-1.3 ng/ml, levels in most patients with chronic myelogenous leukemia, ovarian cancer, cancer of the cecum, breast cancer, malignant melanoma and cancer of the colon were elevated above 1.3 ng/ml.

1464 ALLERGY AND CANCER. *(E.)* Shapiro, S. (Boston Collaborative Drug Surveillance Program, Waltham, Mass.) and Fedullo, A. J. (Temple U. Sch. Med., Philadelphia, Pa.). *Lancet* (7811):1055-1056, 1973.

Two letters to the editor criticize the sample selection techniques described in a previous article in *Lancet* (Meers, p. 884, April 21, 1973) in which the suggestion was made that allergy confers protection against the risk of cancer. One author discusses previously published findings relating to cancer and allergy in 605 cancer patients and 3310 controls, in which no evidence of association either positive or negative, between cancer and allergy, was found. The overall allergy rate in the cancer patients was 7.1% and in the controls 6.9%. The second author points out several erroneous approaches in the Meers paper and suggests that the only valid manner to test the hypothesis is a well-controlled prospective study without preselection of the target populations.

1465 CARCINOEMBRYONIC ANTIGENIC ACTIVITY OF TISSUE EXTRACTS: A QUANTITATIVE STUDY OF MALIGNANT AND BENIGN NEOPLASMS, CIRRHOTIC LIVER, NORMAL ADULT AND FETAL ORGANS. *(E.)* Khoo, S. K. (Walter, Eliza Hall Inst. Med. Res., Melbourne, Australia), N. L. Warner, J. T. Lie and I. R. Mackay. *Int J Cancer* 11(3):681-687, 1973.

A quantitative study of carcinoembryonic antigenic activity (CEA) in extracts of 44 tumors, 42 fetal and 36 non-malignant adult tissues from various organs was made. The CEA content detected by microradioimmunoassay, using a nonspecific goat antiserum, was related to the wet tissue weight. A variety of malignant neoplasms, including carcinomas, sarcoma and Hodgkin's disease, contained CEA (range 17 to 2727 ng); apart from gastrointestinal carcinomas which had the highest content of CEA (230-2727 ng/g) only carcinomas of the lung contained moderate amounts of CEA (63-950 ng/g). The amount of CEA in extracts of fetal and normal adult organs was mostly low or not detectable except in the gastrointestinal tract and pancreas where it ranged from 65 to 200 ng/g in the fetus, and 28 to 86 ng/g in the adult. Differences in mean content of CEA between normal and cirrhotic livers were not significant. The wide distribution of CEA activity in cancerous as well as non-cancerous tissues raises the question of cancer-specificity of CEA. There may be a family of CEA-like molecules which differ slightly in structure according to their tissue of origin but share common antigenic determinants.

1466 INHIBITION OF HEPATOMA-IMMUNE LYMPH-NODE CELL CYTOTOXICITY BY TUMOUR-BEARER SERUM AND SOLUBILIZED HEPATOMA ANTIGEN.

(E.) Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), M. R. Price and R. A. Robins. *Int J Cancer* 11(3):527-535, 1973.

Sera from Wistar rats bearing transplanted aminoazo-dye-induced hepatomas inhibited the cytotoxicity of lymph-node cells from tumor-immune rats for plated hepatoma cells, when the sera were incubated with the effector cells, rather than target cells. In contrast, sera from hepatoma-immune rats, which have previously been shown to block lymphocyte cytotoxicity at the level of the target cell, failed to inhibit lymph-node cell cytotoxicity when pre-incubated with the effector cells. The possibility that antigenic determinants could be responsible for lymphocyte inhibition is supported by the demonstration that papain-solubilized hepatoma-specific antigen preparations inhibited lymph-node cell cytotoxicity. The relative contribution of serum factors to humoral inhibition of lymphocytotoxicity undoubtedly varies at various stages of tumor growth depending upon the height of the tumor-immune response and the extent of tumor-cell turnover leading to membrane antigen release.

1467 MITOGENIC ACTIVITY OF A SEPHAROSE-CONCANAVALIN A COMPLEX ON RABBIT SPLEEN CELLS.

(E.) Ono, M. (Fac. Pharmaceutical Sci., U. Tokyo, Japan), H. Maruta and D. Mizuno. *J Biochem* 73(2):235-243, 1973.

The possibility that concanavalin A (Con A), a plant hemagglutinin, could induce DNA synthesis without entering rabbit spleen cells was examined. Con A was converted to an insoluble derivative by coupling with Sepharose particles using the cyanogen bromide method. Induction of DNA synthesis *in vitro* by this Sepharose-Con A complex was measured in terms of incorporation of tritiated thymidine into acid insoluble materials in rabbit spleen cells. The Sepharose-Con A complex showed mitogenic activity, but its activity was approximately 14% of that of free Con A. The rates of incorporation of tritiated thymidine on addition of the complex and of free Con A were the same, but the complex resulted in highest maximum incorporation. α-Methyl D-mannoside (α-MM) had no protective effect on the coupling reaction of the Sepharose-Con A complex at molar ratios of α-MM to Con A of up to 10^4. The interaction between the Sepharose-Con A complex and spleen cells did not cause agglutination. It is concluded that the initial information of mitogenic activity is transmitted to rabbit spleen cells without the entrance of Con A into the cells, and consequently, that Con A transmits the information for induction of DNA synthesis by contact with the surface of spleen cells.

1468 SELECTIVE ACTIVATION OF T LYMPHOCYTES. ITS POSSIBLE APPLICATION IN THE IMMUNOMANIPULATION OF TUMORS.

(E.) Miller, J. F. A. P. (Inst. Immunol., Basel, Switzerland). *Biomed* 18(2):81-85, 1973.

X-irradiated (CBA x C57BL)F$_1$ or (CBA x Balb/c)F$_1$ mice injected i.v. with thymus cells from CBA mice showed intense cell proliferation in the white pulp of the spleen and cortex of the lymph nodes after three days. This was immediately followed at 3.5 days by the appearance in the thoracic duct of large numbers of lymphoblasts, 95% of which had incorporated ^{3}H-thymidine *in vivo* as determined by autoradiography. These cells had all the characteristics of T-cells, being lysed by anti-theta serum plus complement and resistant to anti-kappa serum. Fewer than one cell in 200 bound antibody-antigen complexes, and the activated cells could specifically reject allogeneic skin grafts. These activated thoracic duct lymphocytes (TDL) thus appeared to have their immunological activities directed primarily, if not exclusively, against the antigens which initially provoked their formation. Intraperitoneal injection of Balb/c activated CBA TDL along with allogeneic DBA/2 mastocytoma cells into NTx CBA mice completely suppressed growth of the tumor in nine of 20 X-irradiated mice. TDL were also able to lyse specifically ^{51}Cr-labeled mastocytoma cells *in vitro*. The ability to stimulate selectively a T-cell population without affecting B-cells may be of value in the field of tumor immunology.

1469 CELL SURFACE ANTIGEN ON QUAIL CELLS INFECTED WITH HERPESVIRUS OF TURKEY OR MAREK'S DISEASE VIRUS.

(E.) Ishikawa, T. (Res. Inst. Microbial Dis., Osaka U., Japan), M. Naito, S. Osafune and S. Kato. *Biken J* 15(4):215-222, 1972.

The fluorescent antibody technique has been used to demonstrate a virus-induced cell surface antigen (CSA) of Japanese quail fibroblasts infected with Marek's disease virus (MDV) or cell-free herpes virus of the turkey (HVT). Although the CSA of HVT reacted strongly with anti-MDV serum, the CSA of MDV reacted only weakly with anti-HVT serum. The CSA of MDV- or HVT-infected cells did not react with antisera against Epstein-Barr virus, cowpox virus, vaccinia virus or chikungunya virus. Results of studies using specific blocking serum on the fluorescent antibody reaction confirmed the specificity of the CSA of both viruses. Formation of HVT CSA was inhibited by puromycin but not by cytosine arabinoside, suggesting that CSA production was not dependent on viral DNA replication but was induced as an early antigen.

1470 MEASUREMENT OF TUMOR IMMUNITY *IN VITRO* WITH ^{125}I-IODODEOXYURIDINE-LABELED TARGET CELLS. *(E.)* Seeger, R. C. (Dept. Zool., University Coll. London, England) and J. T. Owen. *Transplantation* 15(4):404-408, 1973.

The use of ^{125}I-iododeoxyuridine (^{125}IUDR)-labeled target cells to measure *in vitro* tumor immunity is reported. In the cytotoxicity assay, an *in vitro* line of Moloney sarcoma-leukemia virus-induced sarcoma target cells was prelabeled with ^{125}IUDR at a minimally toxic concentration. These target cells were then exposed to lymph node cells sensitized *in vivo* to a syngeneic tumor. The target cell monolayer was then washed, embedded in plastic and radioactivity was measured in an automatic γ counter. Target culture growth was also assayed by ^{125}IUDR labeling of cells preexposed to sensitized lymph node cells. Results showed that lymph node cells from sensitized mice decreased survival and inhibited growth of target cells more effectively than lymph node cells from tumor-free control mice. The ^{125}IUDR method offers advantages over ^{3}H-thymidine labeling since ^{125}IUDR-labeled target cells do not have to be processed for liquid scintillation counting. The problem of spontaneous release of isotope from ^{51}Cr-labeled target cells is also not encountered with ^{125}IUDR. The advantages of ^{125}IUDR over ^{99m}Tc labeling include a longer half life and the ability to use small amounts of isotope.

1471 STUDIES ON DIFFERENT STAGES IN HOST-TUMOR RELATIONSHIP. 1. DIFFERENCES IN THE BEHAVIOR *IN VITRO* OF ASCITES CELLS AND LYMPHOCYTES DERIVED FROM MICE IN EARLY AND LATER STAGES OF TUMOR GROWTH. *(E.)* Pikovski, M. A. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel). *Isr J Med Sci* 9(2):136-144, 1973.

It had previously been shown that spleen and lymph nodes from mice bearing ascites tumors underwent two-stage changes: an increase in size and wt during the first few days of tumor growth followed by a gradual depletion of cells. The events taking place in these stages were further studied *in vitro*. Incubation together for two hr of lymphocytes and MCIA ascites tumor cells from RIII mice resulted in the former cells developing pseudopodia and attaching to or penetrating the tumor cells. The lymphocytes then either moved away or remained adherent to the tumor cells. This phenomenon was observed both with lymphocytes from spleens or lymph nodes of mice with three- to five-day tumor growth ("early stage") and with lymphocytes from spleens or lymph nodes of mice with 12- to 14-day tumor growth ("later stage"). Staining of tumor cell bound lymphocytes with trypan blue, however, showed that the early stage lymphocytes were alive and usually adherent to dead tumor cells. The opposite was seen for tumor cell-bound later stage lymphocytes and tumor cells. Lymphocytes from spleens of nontumor-bearing mice also adhered to early and late stage tumor cells; however, neither lymphocytes nor tumor cell death occurred.

1472 SEROLOGICALLY DEMONSTRABLE CELL SURFACE SPECIFICITIES ON MOUSE NEUROBLASTOMA C1300. *(E.)* Schachner, M. (Sloan-Kettering Inst. Cancer Res. New York, N.Y.). *Nature [New Biol]* 243(125):117-119, 1973.

The surface membrane constituents of cells of mouse neuroblastoma C1300, a transplantable tumor of sympathetic nerve cells, have been examined. The antigens demonstrable on the surface of these cells, which are of sympathetic neuronal origin, are similar to those of normal brain tissue. The major mouse histocompatibility antigen, H-2, was demonstrated on C1300 by the direct cytotoxic test. The results of absorption experiments demonstrate both that the alloantigen, Thy-1, is present on cells of neuronal origin and that it is probably not confined to cells of the central nervous system. Whereas the tumor grown *in vivo* shows concordance with normal brain except for the presence of Gross cell surface antigen in the tumor; the tissue culture line NB41A grown *in vitro* is deficient in two of the antigenic specificies, Thy-1 and NZB autoantigen.

1473 IMMUNITY TO A TA3 TUMOR SUBLINE THAT GROWS IN ALLOGENEIC HOSTS ELICITED BY STRAIN-SPECIFIC TA3 TUMOR CELLS. *(E.)* Lippman, M. M. (Natl. Cancer Inst., Bethesda, Md.), J. M. Venditti, I. Kline and D. L. Elam. *Cancer Res* 33(4):679-684, 1973.

The injection of live mouse strain A/He-specific TA3K1 tumor cells into C57BL6 X DBA/2 F_1 mice rendered these mice immune to subsequent challenge with 10^4 to 10^6 non-strain-specific TA3Ha cells. In the absence of immunization, the i.p. injection of these levels of TA3Ha cells kills 100% of the mice in 11 to 18 days. Defined experimental conditions yielded three distinct alternative results: immunity, prolongation of survival time, or reduced survival time. A high incidence of successful immunization was achieved when 10^2 or 10^6 TA3K1 cells were used, while reduced survival time was the major consequence of the injection of 10^1 or 10^3 to 10^5 TA3K1 cells. One experimental procedure gave a highly significant increase in survival time without immunity. The alternative results reflect differences in sensitizing inoculum levels, tumor challenge, and host variability. The ability of TA3K1 cells to induce a response capable of eliminating up to 10^6 TA3Ha cells demonstrates that ineffective immunogenicity, rather than immune resistance on the part of TA3Ha cells, is the major factor in the permissive growth of these cells in histoincompatible animals. The results indicate the quantitative considerations that would be involved in a therapeutic approach in order to determine the appropriate balance between tumor load and cellular immunogen, which is necessary to produce rejection instead of inhancement.

1474 ANTIBODIES AGAINST AVIAN GS ANTIGEN IN CHICKENS INFECTED NATURALLY AND EXPERIMENTALLY WITH AVIAN RNA TUMOR VIRUSES.

(E.) Ruoslahti, E. (Dept. Serology, U. Helsinki, Finland), A. Vaheri, T. Estola and K. Sandelin. *Int J Cancer* 11(3): 595-603, 1973.

Using a radioimmunoassay (RIA), antibodies against one of the major group specific (gs) antigens of avian RNA tumor viruses were detected in chickens with a naturally acquired leukosis virus infection or bearing Rous sarcoma virus (RSV)-induced tumors. The antibodies were absent in leukosis-free animals, but appeared a few weeks after inoculation with infectious RSV. Out of 11 chickens with a natural or experimental infection, nine had demonstrable anti-gs antibodies. Titers as high as 1:10,000 were observed in chicken sera, but these titers were lower than those in the sera of tumor-bearing hamsters and guinea-pigs. The RIA was about 10,000 times more sensitive in the detection of anti-gs antibodies than the COFAL test. The chicken antibodies could be inhibited with the purified gs antigen and appeared to be of the IgG type. There would seem to be no complete tolerance in chickens at least to one of the major avian gs antigens. Gs-a was also detected in normal chicken embryos and in organs of normal adult chickens. It is suggested that production of antibodies against gs antigens can be explained by the presence of these antigens in amounts too low to maintain tolerance in adult animals.

1475 *BACILLUS CALMETTE-GUERIN* VACCINATION AND SKIN TUMOR PROMOTION WITH CROTON OIL IN MICE.

(E.) Schinitsky, M. R. (U. Wisconsin Ctr. Hlth. Services, Madison), L. R. Hyman, A. A. Blazkovec and P. M. Burkholder. *Cancer Res* 33(4): 659-663, 1973.

The mechanism that may be responsible for an observed decrease in induction and growth rate of croton oil promoted cutaneous papillomas and squamous cell carcinomas in mice following their exposure to *Bacillus Calmette-Guérin* (BCG) was explored. Female Charles River mice, 6 to 8 wk old, were painted once with 7,12-dimethylbenz(a)anthracene (tumor initiation), followed by repeated applications of croton oil (tumor promotion). The i.v. injection of *Bacillus Calmette-Guérin* (BCG) 3 wk prior to the initiation of croton oil treatment reduced the incidence and rate of appearance of both benign and malignant skin tumors. After 50 applications of croton oil, (2/week for 25 weeks), it was possible to demonstrate that the BCG-treated mice without tumors had a smaller spleen index and showed a greater cell-mediated immunological responsiveness to purified protein derivative than did the BCG-treated mice with the greatest number of tumors in the same group. Tubercle organisms were isolated from spleen cultures of the BCG-treated mice, regardless of their capacity to respond to *in vivo* and *in vitro* tests with purified protein derivative as antigen.

1476 STUDIES OF THE EFFECTS OF *BACILLUS CALMETTE-GUERIN* ON MOLONEY SARCOMA VIRUS-INDUCED TUMORS IN NORMAL AND IMMUNOSUPPRESSED MICE.

(E.) Houchens, D. P. (Natl. Cancer Inst., Bethesda, Md.), A. I. Goldberg, M. R. Gaston, M. Kende and A. Goldin. *Cancer Res* 33(4):685-690, 1973.

The actions of *Bacillus Calmette-Guérin* (BCG) on the growth pattern of Moloney sarcoma virus-induced tumor and on hormonal antibody production in immunocompetent mice and in mice immunosuppressed with cyclophosphamide (CY) were examined. BALB/c male mice, 6 to 8 wk old and weighing 20 to 25g, were used in all experiments. Pretreatment with BCG reduced the maximum tumor size and increased the number of survivors of BALB/c mice that were immunosuppressed with CY and inoculated with MSV, compared with immunosuppressed mice that were not pretreated with BCG but which were given MSV. Dose of BCG and time of administration had little effect on this phenomenon. Varying the time that CY was given in relation to MSV inoculation had a significant effect on tumor growth. BCG without immunosuppression did not markedly affect the maximum tumor size, compared with animals that received only MSV. BCG pretreatment could not overcome the immunosuppression as measured by the production of humoral antibodies to sheep red blood cells. Two mechanisms for the effects seen in this system possibly could occur:
1) the observation that pretreatment with BCG did not enhance the humoral response in mice given CY suggests that CY, in addition to producing a depression of general humoral response, may also inhibit blocking antibody; and 2) BCG may replicate more readily in immunosuppressed mice than it does in immunocompetent mice.

1477 GENETIC CONTROL OF MuLV-gs EXPRESSION IN CROSSES BETWEEN HIGH AND LOW LEUKEMIA INCIDENCE STRAINS.

(E.) Hilgers, J. (Netherlands Cancer Inst., Amsterdam) and J. Galesloot. *Int J Cancer* 11(3):780-793, 1973.

A genetic analysis of the host control for the expression of the group-specific antigen of murine leukemia virus (MuLV-gs) using crosses between high and low leukemia incidence mouse strain and a newly developed test for MuLV-gs antigen, based on disappearance rate (DR) of injected rat anti-MuLV antibody in mice in combination with a previously described quantitative immunofluorescence adsorption (IFA) technique for the antigen in spleens is presented. The following groups could be distinguished: (1) low leukemia incidence strains, mice which were MuLV-gs negative in their spleens and sera; (2) low leukemia incidence strains, of which young adult mice were generally MuLV-gs negative, whereas old mice were occasionally positive, levels of the antigen being invariably low in the spleens of positive animals; (3) one low leukemia strain (NZB, a mouse strain with auto-immune disease), of which young adult mice were generally MuLV-gs negative, whereas all older mice were highly positive, levels in spleens reaching those in spleens of high leukemia strain mice; and (4) high leukemia strains, mice of which were always MuLV-gs positive, levels of the antigen in spleens and sera being invariably high, even in young adult mice. Spleens and sera of adult animals of the F_1 hybrid between a high (AKR) and a low (C57BL) leukemia incidence strain contain intermediate amounts of MuLV-gs. In this heterozygote, the antigen was generally found at an earlier age than in all low leukemia incidence strains, but at a later age than in the high leukemia incidence strain. Typing for MuLV-gs in spleens and sera of normal segregants between mice of high and low leukemia strains revealed that: (1) one semidominant host gene, presumably Fv-1, controls MuLV-gs levels as shown in backcrosses to the high incidence strain, and (2) two additional dominant genes are involved in all-or-none expression of MuLV-gs as shown in backcrosses to the low incidence strain and in F_2 generations.

1478 TEMPERATURE-DEPENDENT MOBILITY OF CONCANAVALIN A SITES ON TUMOUR CELL SURFACES.

(E.) Nicholson, G. L. (Salk Inst. Biol. Studies, San Diego, Calif.). *Nature* [*New Biol*] 243(128): 218-220, 1973.

The temperature-dependent variations in the cell surface distribution of concanavalin A (con A) binding sites were studied in normal and virus-transformed established mouse cell lines. Balb/c 3T3, SV40-transformed 3T3 (SV3T3), and 3T12 cells either labeled at 0 C with fluorescein-conjugated con A, or incubated at 0 C with con A followed by fluorescein-conjugated anti-con A, showed a uniform, "ring-shaped" distribution of fluorescence about the cell periphery. However, when SV3T3 or 3T12 cells, the two noncontact-inhibited lines, were labeled with con A at 0 C, incubated at 22 C or 37 C, and labeled at 0 C with fluorescein-conjugated anti-con A, the distribution of fluorescence over the cell surface was patchy. 3T3 cells, a contact-inhibited line, similarly grown and treated continued to show the uniform distribution of fluorescence. Formaldehyde fixation at high concentrations after con A labeling at 0 C prevented the SV3T3 and 3T12 cells from showing "patchy" fluorescence. The data suggest that the clustering of lectin binding sites on transformed cells is a consequence of a difference in fluidity of the transformed cell membranes which allows distributional perturbations induced by the polyvalent lectin molecules.

1479 RADIOIMMUNOASSAY OF RAT α_1-FETOPROTEIN.

(E.) Sell, S. (U. California San Diego Med. Sch., La Jolla). *Cancer Res* 33(5):1010-1015, 1973.

A competitive binding radioimmunoassay for rat α_1-fetoprotein (α_1F) has been developed. For standard assays, 0.1 ml of unknown is added to 0.1 ml of labeled rat α_1F and 0.8 ml of a dilution of goat anti-α_1F is added (usually 1:8192). After 1 hr at room temperature, 1.5 ml of Na_2SO_4 (30 g/100 ml) are added and, after 1 hr more, the mixtures are centrifuged at 3100 rpm at 25 C for 1 hr. The supernatant is removed to a counting tube. Both the precipitate and supernatant are counted for 1 min on an automatic γ scintillation counter. This assay is approximately 20,000 times more sensitive than double diffusion in agar for α_1F detection, the threshold being approximately 5 ng. Further purification of apparently pure (by immunodiffusion and immunization) radiolabeled α_1F is required for the specific assay. An assay for a previously undetected contaminant(s) is used to check the purity of rat α_1F isolated by isoelectric focusing in order to obtain the purified unlabeled α_1F needed to establish the standard inhibition curve.

1480 DEPRESSION OF ANTILYMPHOMA ALLOGRAFT REACTIVITY BY TUMOR-ASSOCIATED FACTORS. *(E.)*

Bonmassar, E. (U. Perugia Sch. Med., Italy), A. Bonmassar, A. Goldin and G. Cudkowicz. *Cancer Res* 33(5):1054-1061, 1973.

Relatively small numbers of viable or killed tumor cells (10^2 to 10^5) of five transplantable mouse leukemias and one carcinoma were inoculated i.v. or i.p. into syngeneic or allogeneic adult mice. The inoculations depressed the allograft reactivity of the recipients as judged from reduced resistance to the growth of a second transplant of 10^6 to 10^7 *H-2*-incompatible strain-specific lymphoma cells. A single injection of the most effective conditioning cells caused long-lasting depression of antilymphoma reactions (up to 3 months) regardless of whether the *H-2* antigens of challenging and conditioning tumors were different or alike. The active factor was extractable from cells in suspension and separable by filtration through Millipore membranes. Of 338 mice pretreated with tumor material under a variety of conditions and schedules, 326 were rendered susceptible to allogeneic lymphoma grafts, whereas only 19 of 219 untreated control mice failed to reject the tumors. Depression of antilymphoma reactions was not caused by cells of normal spleen and of two other leukemias and one carcinoma. However, lactic dehydrogenase-elevating virus, a contaminant of all tumors used, and Moloney sarcoma virus did cause nonspecific depression, thus imitating the tumors. Depressive factors capable of breaking the major histocompatibility barrier of the species (possibly viruses, viral products, or tumor cell products) were associated with as few as 100 neoplastic cells. If this association would also occur during oncogenesis, it could favor the escape of antigenic tumors from host surveillance.

1481 QUANTITATIVE EFFECTS OF NUTRITIONAL PROTEIN AND CALORIE DEFICIENCY UPON IMMUNE RESPONSES TO TUMORS IN MICE.

(E.) Jose, D. G. (Variety Club Heart Hosp., Minneapolis, Minn.) and R. A. Good. *Cancer Res* 33(4):807-812, 1973.

The effects of protein calorie deficiency on host immune responses to tumors in mice were examined. Measurement of specific cellular and humoral immune responses to tumors in allogenic, syngenic, and autochthonous mouse systems showed both responses to be depressed by nutritional protein or calorie restriction, but humoral responses were affected at a higher nutritional level than are cellular responses. A range of moderate protein deficiency was found where cellular immune responses were enhanced due to lack of blocking serum antibody; animals on this dietary protein level showed marked inhibition of tumor growth. Changes in lymphoid cell numbers and kinetics of the cellular response were found that may reflect mechanisms to conserve nutritional reserves.

1482 ULTRASTRUCTURAL STUDIES OF HISTIOCYTE-TUMOR CELL INTERACTIONS DURING TUMOR REGRESSION AFTER INTRALESIONAL INJECTION OF *MYCOBACTERIUM BOVIS.*

(E.) Snodgrass, M. J. (Carcinogenesis Program, Oak Ridge Natl. Lab., Tenn.) and M. G. Hanna, Jr. *Cancer Res* 33(4):701-716, 1973.

The site of a transplanted syngeneic hepatocarcinoma and its draining lymph nodes in inbred guinea pigs were examined ultrastructurally in the presence and absence of *Mycobacterium bovis* strain *Bacillus Calmette-Guerin* (BCG). BCG was injected into the growing intradermal tumor 7 days after transplantation, at which time it had metastasized to the first regional lymph node. Critical morphological similarities were found among activated histiocytes in the various reactive regions: infiltrating at the tumor site, autochthonous to the draining lymph node, migrating from the tumor site to the subcapsular marginal sinus of the draining lymph node, and infiltrating into the medullary sinuses of the lymph node. This observation indicates that these are all forms of a specific class of cell within the macrophage-histiocyte series. The cytopathic effect of these activated histiocytes on tumor cells is mediated by cell surface contact; sizeable areas of fusion of the cellular membranes of histiocytes and hepatocarcinoma cells were observed. Thus, destruction of target cells by nonphagocytic mechanisms may be a phenomenon related to activated but not specifically sensitized cells of the macrophage-histiocyte series.

1483 INCREASED IMMUNOSENSITIVITY IN NITROGEN MUSTARD-RESISTANT YOSHIDA SARCOMA.

(E.) Tsukagoshi, S. (Cancer Inst., Tokyo, Japan) and Y. Hashimoto. *Cancer Res* 33(5):1038-1042, 1973.

Yoshida sarcoma cells acquired resistance to nitrogen mustard on repeated *in vitro* contact with the agent. When female, Donryu rats, 6 to 7 wk old and weighing 120 to 140 g, were inoculated s.c. with these highly resistant Yoshida sarcoma cells (resistance index, 2500) and not treated, initial tumor growth was followed by regression and all of the animals survived. Inoculation was made into the right hind footpad, a site favorable for the experimental induction of lymph node and organ metastases. Among these animals, the size of the right foot enlarged, accompanying lymph node metastasis, but the tumors began to regress starting about 2 weeks after inoculation and disappeared completely after 1 month. With antiserum or peritoneal lymphoid cells obtained from rats immunized with either drug-sensitive or -resistant Yoshida sarcoma cells, it was found that the immunosensitivity of drug-resistant Yoshida sarcoma cells increased in comparison with the cells of the original sensitive line. It is suggested that increased immunosensitivity could account for the observation of greater chemotherapeutic response to 6-mercaptopurine for rats inoculated i.p. with resistant Yoshida sarcoma cells as compared with those inoculated with the original sensitive line.

1484 EARLY APPEARANCE OF SERUM α-FETOPROTEIN AS A FUNCTION OF DOSAGE OF VARIOUS HEPATOCARCINOGENS.

(E.) Kroes, R. (Natl. Cancer Inst., Bethesda, Md.), G. M. Williams and J. H. Weisburger. *Cancer Res* 33(3):613-617, 1973.

The effect of different doses of 3'-methyl-4-dimethylaminoazobenzene (3'-MOAB) on the early appearance of α-fetoprotein (AFP) in the serum of rats was investigated. AFP was induced after 3 weeks of ingestion of diets with 600 ppm 3'MDAB and appeared after only 2 weeks when higher dosages (900 and 1200 ppm) were used. Lower levels of 300 ppm 3'-MDAB gave only a transient appearance of AFP, starting at the 5th week and detectable for 3 more weeks, while 150 ppm did not induce AFP production. Immunosuppression with anti-rat lymphocyte globulin extended for 1 week the time during which positive AFP titers were maintained upon cessation of 3'-MDAB (600 ppm) intake. A transient appearance of AFP was found when rats were given the carcinogens 2'-methyl-4-dimethylaminoazobenzene (600 ppm), aflatoxin B_1 (4 ppm), *N*-2-fluorenylacetamide (200 and 300 ppm), and *N*-hydroxy-*N*-2-fluorenylacetamide (213 and 320 ppm). Lower dosages of aflatoxin (0.2 and 2 ppm), *N*-2-fluorenylacetamide (150 ppm), *N*-hydroxy-*N*-2-fluorenylacetamide (160 ppm), and diethylnitrosamine (40 ppm) did not induce detectable AFP levels in serum, nor did 2'-methyl-4-dimethylaminoazobenzene (600 ppm) and CCl_4 (50 mg i.p. twice a week). It would appear that high levels of liver carcinogens are required to induce the early appearance, within 2 to 5 weeks, of detectable AFP in serum. The histological study of the liver revealed that the alterations evoked by the different compounds were quite severe in those rats showing the early appearance of AFP although no definite cancers were shown at that time. It may well be that the carcinogenic compounds found to be active in early AFP-induction were so because of their carcinogenic action in combination with the induction of extensive parenchymal and bile duct cell proliferation.

1485 VIRAL ETIOLOGY OF IMMUNOLOGICALLY INDUCED LYMPHOMAS.

(E.) Cornelius, E. A. (Yale U. Sch. Med., New Haven, Conn.). *Transplant Proc* 5(1):957-960, 1973.

Experiments were conducted on F_1 hybrids of two mouse strains (S and B) known to harbor tumorigenic virus in an attempt to elucidate the etiology of graft-versus-host reaction (GVHR)-induced lymphomas. Inoculation of $(SB)F_1$ male and female mice with either viable or frozen and thawed S spleen cells resulted in the widespread development of reticulum cell sarcoma in all animals. No tumors developed over a five-month period in $(SB)F_1$ mice given S liver cells, $(SB)F_1$ mice given viable $(SB)F_1$ spleen cells or in normal $(SB)F_1$ controls. Tumor growth was also observed in $(SB)F_1$ and B, but not in S, $(NZBxS)F_1$, or NZB, mice inoculated with viable or frozen and thawed tumor cells transplanted into syngeneic $(SB)F_1$ mice. Tumors also developed in newborn B and $(BA)F_1$ mice which received cell-free preparations from tumors arising in $(SB)F_1$ mice injected with viable S spleen cells. These results provided strong evidence for a viral etiology of GVHR-induced tumors.

1486 DEPRESSED CELL-MEDIATED IMMUNITY IN NEWBORN RABBITS BEARING FIBROMA VIRUS-INDUCED TUMORS. *(E.)* Tompkins, W. A. F. (Coll. Vet. Med., U. Illinois, Urbana), R. M. Schultz and G. V. S. V. Rama Rao. *Infect Immun* 7(1):613-619, 1973.

Cell-mediated immunity to fibroma tumors in adult and newborn New Zealand rabbits was examined. The adult rabbits (2-4 months old) were given intradermal inoculation at two sites on the shaved back with 2.5 x 10^5 PFU of fibroma virus. Newborn rabbits (1-3 days old) were inocualted s.c. with the same amount of virus. Inoculation of the adult rabbits resulted in tumors that grew for 9 to 12 days to a diameter of 2.0 to 2.5; this was followed by complete regression after 18 to 21 days. In contrast, tumors induced in newborn rabbits grew to a much greater size, ranging from 5 to 7 cm in diameter by 3 to 4 wk after virus inoculation. In the latter case, some animals died, others survived with a gradual regression of the tumors over an additional period of 5 to 6 wk. Virus neutralization studies demonstrated antibodies to fibroma virus in the serum of both adult and newborn tumor-bearing rabbits. Newborn rabbits with progressively growing tumors failed to elicit a delayed cutaneous hypersensitivity reaction to fibroma antigens, whereas adult rabbits showed a strong reaction as early as seven days after tumor induction. Macrophage migration inhibition tests revealed that the lymphocytes from newborn rabbits with progressively growing tumors were only weakly reactive to fibroma antigens, as compared to lymphocytes from adult tumor-bearing rabbits. Newborn rabbits that survived and regressed the tumors demonstrated strong cell-mediated immunity by skin testing and migration inhibition. It is suggested that in newborn rabbits the macrophage or reticuloendothelial system is immature resulting in reduction in the number of macrophases available for recruitment into the antigen inoculation site and thus offering one possible explanation for failure to passively transfer delayed hypersensitivity. A similar theory would explain the failure to demonstrate a delayed hypersensitivity reaction in newborn rabbits with progressively growing tumors.

1487 PURIFICATION AND PROPERTIES OF THE 'A' ANTIGEN ASSOCIATED WITH MAREK'S DISEASE VIRUS INFECTIONS. *(E.)* Ross, L. J. N. (Houghton Poultry Res. Sta., Huntingdon, England), P. M. Biggs and A. A. Newton. *J Gen Virol* 18(3):291-304, 1973.

The production, purification and properties of the A antigen induced by Marek's disease virus in tissue culture were investigated. Although the virus multiplied on passage in chick kidney, chick embryo and duck embryo cells, A antigen could be detected by immunofluorescence much more easily in chick kidney and duck embryo. A antigen produced by duck embryo cells cross reacted in immunodiffusion tests with antigen produced in duck embryo cells infected with turkey herpes virus, but not with antigen produced in cells infected with herpes simplex or pseudorabies virus. A antigen could be purified 20-fold by electrophoresis in 5% polyacrylamide gels with a 45% recovery. Similar purification was obtained by chromatography on DEAE-Sephadex A50 and Sephadex G200; however, recovery was only 20%. A antigen was ether resistant, sensitive to trypsin digestion, had an isoelectric point in 2% Ampholine +2M urea ranging from pI 4.5 to pI 5.5, and was stained by the periodic acid Schiff reaction. Electrophoresis in 7.5% acrylamide SDS gels revealed a major band corresponding to a molecular wt of 90,000 and a minor band at 50,000. Gel filtration on Sephadex suggested a molecular wt for A antigen of 80,000 ± 10,000. Immunological and autoradiographic studies using ^{3}H-glucosamine labeled material suggested that the A antigen is a glycoprotein.

1488 IMMUNOHISTOCHEMICAL DEMONSTRATION OF AVIAN LEUKOSIS VIRUS ANTIGENS IN PARAFFIN EMBEDDED TISSUE. *(E.)* Di Stefano, H. S. (Upstate Med. Ctr., Syracuse, N.Y.), A. A. Marucci and R. M. Dougherty. *Proc Soc Exp Biol Med* 142(4):1111-1113, 1973.

An immunological procedure was applied to the detection of avian leukosis virus (ALV) antigen in paraffin-embedded tissues of the intact host. Antiviral antisera were obtained from chickens infected either with RAV-1, which belongs to ALV serotype A, or with RAV-6 which is serotype B. Pancreas, which is a particularly rich source of ALV in infected birds, was obtained from chickens infected with the F-42 strain of ALV, which belongs to serotype A. Pancreases of uninfected birds from a flock known to be free of ALV infections were used as controls. The specificity of the procedure was confirmed by exposing sections of pancreas infected with type A virus to chicken antiserum directed against the type B ALV antigen. Under these conditions no specific staining developed when the immunohistochemical reaction was carried out. Sections of pancreas from uninfected control birds were also negative with either type A or B antiviral serum.

1489 IMMUNE RESPONSE TO GROSS VIRUS-INDUCED LYMPHOMA: COMPARISON OF TWO *IN VITRO* ASSAYS OF CELL-MEDIATED IMMUNITY. *(E.)* Wright, P. W. (Natl. Cancer Inst., Bethesda, Md.), M. O. de Landazuri and R. B. Herberman. *J Natl Cancer Inst* 50(4):947-954, 1973.

Male rats were injected s.c. with 10^8 cells of the Gross virus-induced W/Fu rat lymphoma (C58NT)D. Spleen, lymph node and blood cells were prepared from injected rats and tested in microcytotoxicity assays (MCA) and in ^{51}Cr-release cytotoxicity assays (CRA) with tissue culture target cells. Significant cytotoxicity was found in both assays ten days after inoculation with lymphoma material. After 40 days blood lymphocytes were cytotoxic in CRA only when lymphocytes had been preincubated for 24 hr at 37 C before exposure to target cells. MCA showed cytotoxicity for cells without preincubation at 40 days. With preincubation, CRA showed cytotoxicity for rat lymphocytes up to 180 days postinoculation. With MCA, positive results were seen for 120 days. Rats, inoculated when newborn with Gross leukemia virus and tested at 60 days of age also showed cytotoxic lymphoid cells in both MCA and CRA.

1490 CELL-MEDIATED IMMUNE REACTION AGAINST TUMORS INDUCED BY ONCORNAVIRUSES: NATURE OF THE EFFECTOR CELLS IN TUMOR-CELL CYTOLYSIS.

(E.) Leclerc, J. C. (St. Louis Hosp., Paris, France), E. Gomard, F. Plata and J. P. Levy. *Int J Cancer* 11(2):426-432, 1973.

Spleen cells were prepared from BALB/c and C57B1/6 mice bearing murine sarcoma virus (MSV)-induced tumors. The cytotoxicity of these cells for ^{51}Cr-labelled tumor target cells was tested. In the presence of complement, cytotoxicity of spleen cells was completely inhibited by exposure of cells to anti-θ-C3H immune sera prepared by injecting AKR mice with C3H/He thymus cells. Anti-mouse immunoglobulin sera did not affect the cytotoxicity of lymphoid cells. Attempts to render normal BALB/c spleen cells cytotoxic for tumor cells by preincubating spleen cells with heat-inactivated hyperimmune anti-H_2b sera or with sera from MSV-infected mice were not successful.

1491 REACTION OF NORMAL HUMAN LYMPHOCYTES AND CHRONIC LYMPHOCYTIC LEUKEMIA CELLS WITH AN ANTITHYMOCYTE ANTISERUM.

(E.) Aisenberg, A. C. (Massachusetts Gen. Hosp., Boston), K. J. Bloch, J. C. Long and R. B. Colvin. *Blood* 41(3):417-423, 1973.

The identification of human T-cells (thymus-derived or -dependent) using an antithymocyte antiserum was examined. An antiserum against human fetal thymus was prepared in rabbits and absorbed with liver cells from the thymus donor and lymphocytes from a patient with chronic lymphocytic leukemia (CLL). The antiserum reacted with 80 - 95% of the peripheral small lymphocytes from 12 normal controls but with only 1 - 11% of cells from 12 patients with CLL. The antithymocyte antiserum demonstrates a cell surface antigen present on the majority of normal human lymphocytes, but absent from the neoplastic CLL cells. This finding is consistent with the view that the CCL lymphocyte is devoid of T-cell antigen and with other evidence that this lymphocyte closely resembles the B-cell (bone marrow or bursal equivalent-derived or -dependent) of experimental animals. It is suggested that leukemic cells will prove useful in preparing an antihuman thymocyte antiserum that will react exclusively with T-cells by removing B-cell reactivity.

1492 LACK OF ASSOCIATION BETWEEN CARCINOMA OF THE BREAST AND HL-A SPECIFICITIES.

(E.) Martz, E. (Dept. Pathol., Harvard Med. Sch., Boston, Mass.) and B. Benacerrof. *Tissue Antigens* 3(1): 30-38, 1973.

Forty-eight patients with carcinoma of the breast and 122 matched controls from the Boston area were typed for 24 HL-A specificities by cytotoxicity tests using human isoantisera. Computer analysis of the results failed to indicate a significant correlation between breast carcinoma and changes in the frequency of any of the HL-A specificities or pairs of specificities. These findings failed to confirm previously published results which suggested an association between breast cancer and HL-A7.

1493 SPONTANEOUS TUMORS OF AGING WILD HOUSE MICE. INCIDENCE, PATHOLOGY, AND C-TYPE VIRUS EXPRESSION.

(E.) Gardner, M. B. (Southern California Sch. Med., Los Angeles), B. E. Henderson, R. W. Rongey, J. D. Estes and R. J. Huebner. *J Natl Cancer Inst* 50(3):719-734, 1973.

Of 532 wild mice trapped over a two year period in urban Los Angeles County and held without treatment until old age, 293 were eventually killed and autopsied because of illness, suspected tumor, or death. Of these, 28 (9.5%) mice (15 females, 13 males) contained spontaneous tumors which consisted in decreasing order of frequency of lymphomas, pulmonary adenomas, fibrosarcomas, and granulocytic neoplasms. Tumor incidence increased with aging with a sharp rise in the rate of lymphomas, pulmonary adenomas and fibrosarcomas occurring after two years. Granulocytic neoplasms were observed as early as one year. Of 13 lymphomas which could be classified histologically, three were well-differentiated lymphocytic leukemias, three were poorly-differentiated lymphocytic leukemias, and seven were type-B reticulum cell sarcomas. CF antigen and C-type particles were detected in less than 20% of the spleen extracts of freshly trapped mice or from sick or dead non-tumor-bearing mice about 2.5 to 3 years of age. However, 70 to 100% of mice with spontaneous lymphomas, regardless of histologic type, showed a high CF antigen titer and presence of C-type particles in the spleen and tumor. Mice with spontaneous fibrosarcomas had a 20% incidence of detectable CF antigen in tumor and spleen with no observable C-type particles. No CF antigen or C-type particles were detected in mice with pulmonary adenomas. No mice contained detectable antibodies against polyoma or other indigenous murine viral antigens.

1494 A STUDY OF THE EFFECT OF ACID MUCOPOLYSACCHARIDES ON THE GROWTH OF EXPERIMENTAL TUMORS IN MICE.

(E.) Shohat, B. (Beilinson Hosp., Petah Tikva, Israel), D. Allalouf and A. Ber. *Pathol Biol* 21(2): 117-122, 1973.

The effect of i.p. injections of hyaluronic acid, chondroitin sulfate and heparin on the growth of solid sarcoma S-180, mammary adenocarcinoma E0771, ascites sarcoma S-180, Ehrlich ascites carcinoma, L1210 mouse leukemia and Y7A4 Moloney-induced lymphosarcoma in mice was examined. The effect of preincubation of the cancer cells in solutions of acid mucopolysaccharides (AMPS) prior to inoculation was also studied. The strains of mice used for the various types of tumors were inbred C57 BL for E0771 and Y7A4, hybrid BDF1 for L1210, and random-bred Swiss mice for solid and ascitic sarcoma S-180 and for the Ehrlich ascites carcinoma. Experimental groups comprised 6-8 animals each. Solutions of buffered saline were injected i.p. in a volume of 0.1 ml daily, starting 24 hr after tumor implantation. Ten injections were given for sarcoma S-180, adenocarcinoma E0771 and Y7A4 Moloney lymphosarcoma; six injections were given for the other types of tumors. Control mice received buffered saline. There was a trend for increased tumor weights, total volumes or total packed-cell volumes in treated mice as compared to the untreated. Sarcoma S-180 cells pretreated with hyaluronic acid and chondroitin sulfate developed into tumors significantly larger than those developing from untreated cells. Contrary to a recently advanced hypothesis, a number of experiments carried out with mice immune to sarcoma S-180 showed that pretreatment of sarcoma S-180 cells with AMPS did not affect the resistance of the mice to this tumor. The variabilities in the responsiveness of various tumors in these studies indicate that one should be careful in making generalizations in this kind of biological response.

1495 THE PROTON RELAXATION ENHANCEMENT PROPERTIES OF CONCANAVALIN A.

(E.) Barber, B. H. (Dept. Med. Cell Biol., U. Toronto, Canada) and J. P. Carver. *J Biol Chem* 248(9):3353-3355, 1973.

The measurement of the proton relaxation rate ($1/T_1$) enhancement (ε_1*) for the concanavalin A (Con A)-Mn^{2+}complex as a function of time following the addition of Mn^{2+} indicates a pH-dependent rate of structural rearrangement at the bound Mn^{2+} site. The addition of excess Ca^{2+} interrupts this kinetic process and results in a reduced time-independent value of ε_1*. The further addition of α-methyl-D-glucoside to the Con A-Mn^{2+}-Ca^{2+} complex also alters the observable ε_1*. Using the recently published 2 A resolution crystal structure of Con A, the results are interpreted as arising from the gradual folding of the loop of residues 12 to 22 over the initial Mn^{2+} binding site (formed by residues 8, 10, and 24) to close the Mn^{2+} off from free access by water molecules. Measurement of ε_1* may thus be useful in probing the interaction of con A with cell surface components.

1496 TRANSPLANTATION, IMMUNOSUPPRESSION, AND ONCOGENESIS.

(E.) Anonymous. *JAMA* 224(8):1183-1184, 1973.

The relationship between the immunosuppression regimen for kidney transplant patients and the appearance of virus particles with the typical structure and size of the oncogenic papovavirus in the urine of these patients is briefly discussed. Five of the patients had received a cadaver kidney, two live donor kidneys, and one patient had received first a cadaver kidney, then on second transplantation a live donor kidney. Antilymphocyte serum preparations given to these patients showed no sign of virus nor did other patients or hospital personnel. The source of the virus is unclear but it is theorized that the virus found in these transplant patients may have been a latent human virus activated by immunological interference. The form of immunosuppression may also have an influence upon development of such a virus. It will be necessary to follow up these patients for many years to determine if malignant transformation will develop in their genitourinary tracts.

1497 A CROSS-REACTING EMBRYONIC ANTIGEN IN THE MEMBRANE OF RAT SARCOMA CELLS WHICH IS IMMUNOGENIC IN SYNGENEIC HOST.

(E.) Thomson, D. M. P. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England) and P. Alexander. *Br J Cancer* 27(1):35-47, 1973.

Indirect immunofluorescence, immunodiffusion and precipitation-inhibition assays were used to test for tumor-associated macromolecules on the membrane surfaces of methylcholanthrene-induced MC-1 sarcoma cells maintained in inbred male hooded rats. Serum of rats hyperimmunized with syngeneic MC-1 cells contained antibodies to two apparently different sarcoma membrane antigens. One antigen, which was unique to the MC-1 tumor and could not be detected on the surface of syngeneic embryo cells, had a molecular wt of 40,000-50,000 and resembled tumor-specific transplantation antigen. The other, oncoembryonic antigen (OEA-I), was present on 16 other sarcomas induced by various chemical carcinogens and by interferon, as well as in 9-11 day-old embryos. Two other onco-embryonic components, OEA-II and OEA-III, were also detected on MC-1 sarcoma cells, but these were not antigenic in hooded rats.

1498 CELLULAR ANALYSIS OF IMMUNOSUPPRESSION TO SYNTHETIC POLYPEPTIDE IMMUNOGENS INDUCED BY A MURINE LEUKEMIA VIRUS.

(E.) Shearer, G. M. (Weizmann Inst. Sci. Rehovot, Israel), E. Mozes, N. Haran-Ghera and Z. Bentwich. *J Immunol* 110(3):736-742, 1973.

Adult SJL/J mice were injected i.p. with a leukemogenic virus (that causes lymphatic leukemia in SJL/J mice) prepared from irradiated syngeneic donors. One month later, the animals were either immunized with the multichain synthetic polypeptide poly(*l*Tyr, *l*Glu)-poly*l*Pro--poly*l*Lys, abbreviated (T,G)-Poly--L, or were used as donors of spleen, thymus, and/or bone marrow cells in transfer experiments. The virus-treated mice exhibited reduced anti-(T,G)-Pro--L titers compared to the responses of control animals. The immunosuppressive effects of the leukemogenic agent were strain-specific, since a radiation leukemia virus preparation from C57BL/6 mice had no effect in SJL/J animals. The suppressed responses of the virus-treated mice were not affected by injection of either poly adenylic:poly uridylic acid or thioglycolate, although they were abolished by complexing the immunogen to methylated bovine serum albumin. Results of the transfer experiments indicated that the immunosuppressive effects were expressed at the immunocompetent cell level, and that the virus affected both the thymus- and marrow-derived populations of immunocytes. The immunosuppressive effects on marrow cells independent of any contribution by helper cells of thymic origin were verified with a thymus-independent synthetic polypeptide composed of *d* amino acids.

1499 RECONSTITUTION OF T CELL-DEPLETED SPLEEN CELL POPULATIONS BY FACTORS DERIVED FROM T CELLS. I. CONDITIONS FOR THE PRODUCTION OF ACTIVE T CELL SUPERNATANTS.

(E.) Gorczynski, R. M. (Dept. Med. Biophys., U. Toronto, Canada), R. G. Miller and R. A. Phillips. *J Immunol* 10(4):968-983, 1973.

A factor has been characterized which replaces a T-cell function in the initiation of an *in vitro* antibody response to sheep erythrocytes (SRBC) by anti-Θ-treated mouse spleen cells. The factor, which was apparently nonspecific, was released into the medium of cultured T-cells. Release required stimulation with specific antigen, but the activity produced by heterologous antigens could reconstitute an *in vitro* response to SRBC. Preparation active in the assay could also initiate immune response in B-cells obtained from thymectomized, irradiated, bone marrow-reconstituted mice and in B-cells separated from normal bone marrow. It was concluded that the source of this factor was the T-cell since 1) factor-producing ability was abolished by treatment with anti-Θ antiserum plus complement, 2) cells producing the factor had the same sedimentation profile as functional T-cells; and 3) the sedimentation profile of factor-producing cells in a population of alloantigen-stimulated cells was identical to that of cells involved in cell-mediated immunity in the same population.

1500 LYMPHOCYTE TRANSFORMATION INDUCED BY AUTOLOGOUS CELLS. II. STIMULATION BY MITOGEN-INDUCED LYMPHOBLASTS.

(E.) Weksler, M. E. (Cornell U. Med. Coll., New York, N.Y.). *J Exp Med* 137(3): 799-806, 1973.

Human lymphocytes transformed *in vitro* by phytohemagglutinin (PHA) or concanavalin A (con A), x-irradiated, and washed free of excess mitogen were able to induce transformation of autologous lymphocytes as determined by their ability to incorporate ^{3}H-thymidine into DNA. Lymphocyte cultures containing only irradiated lymphocytes not exposed to mitogen were not stimulated. Since peak ^{3}H-thymidine incorporation into lymphocytes exposed to mitogen-transformed lymphocytes (i.e., lymphoblasts) occurred days later than that into PHA-stimulated lymphocytes, and since dissociation of mitogen from stimulated lymphoblasts increased their ability to transform autologous lymphocytes, it was concluded that the transforming ability of the treated lymphoblasts was not due to residual mitogen contamination. The ability of mitogen-stimulated lymphocytes to stimulate autologous lymphocyte transformation preceeded their morphological transformation.

1501 CROSS-IMMUNITY AMONG SYNGENEIC TUMORS OF MICE.

(E.) Usubuchi, I. (Hirosaki U. Sch. Med., Japan), Y. Sobajima, H. Kudo, T. Hongo and M. Sugawara. *Tohoku J Exp Med* 108(1):79-84, 1972.

Cross-immunity among mammary carcinomas of C3H/He mice and that between mammary carcinoma and methylcholanthrene (MCA)-induced sarcoma of C3H/He mice were observed. Immunization was usually performed by the inoculation of a fragment of the tumor into the subcutaneous tissue of the mouse. When the engrafted tumors reached a size of approximately 10 mm, surgical excision or ligation-and-release was done. Some of these inoculated tumors showed spontaneous reduction. The challenging tumor was inoculated into the subcutaneous tissue of the dorsum of the test mice two to three wk after the immunizing procedure. The degree of immunity in cases of spontaneous regression of the immunizing tumor was more marked than that in cases of ligation-and-release or of surgical excision of the immunizing tumor. Although antigenicity peculiar to the animal in which the tumor originated, in addition to common antigens, had been shown in allogeneic tumors, such antigenicity could not be demonstrated in syngeneic tumors. Autologous transplantation of mammary carcinomas of C3H/He mice was inhibited effectively by immunization with syngeneic MCA-induced sarcoma. The degree of inhibition was more marked in cases of spontaneous regression of the immunizing tumor than in cases of ligation-and-release or of surgical excision of the immunizing tumor.

1502 DETECTION OF NEOANTIGENS IN THE SERUM OF PATIENTS WITH ACTIVE NEOPLASTIC DISEASES BY THE ABSORPTION-IMMUNODIFFUSION METHOD.

(E.) DeCarvalho, S. (Belmont Med. Clin., Bellflower, Calif.). *Oncology* 27(3):193-234, 1973.

Horse precipitating antibodies produced against antigens isolated from a large number of human tumors have revealed identical tumor associated antigens in the serum of many patients with active malignant disease but not in the serum of patients afflicted with a variety of disorders including infectious, inflammatory and autoimmune disease, or in the serum of healthy individuals, in persons bearing benign tumors or in the serum of cancer patients in clinical remission. In the first 791 coded serum samples studied by an absorption-immunodiffusion method, there was 97-34% consistency and accuracy, and a conditional probability for screening correctly of 86%. Intricate Ouchterlony patterns indicated complex antigenic composition and a relationship between different neoplasms. Although a common antigen could not be excluded, the presence of group specific and single tumor antigens was observed. Quantitation of antigens in the serum with view to a measure of tumor specific tolerance was attempted but no information was obtained to correlate the amount of tumor tissue with the amount of circulating tumor antigen. The value of this test in presymptomatic detection was not established at this time. Conditions that may lead to false negatives include: 1) lack of serologic specificity of the reagent antiserum; 2) sub-minimal amounts of cancer tissue in the patient; 3) walling off of the tumor or limited tumor decay, catabolism, or turnover; and 4) removal of tumor antigen by macrophages or immunocytes, non-specific absorption on normal tissues, neutralization by auto-antibody or other means of detoxification.

1503 NATURE OF LYMPHOCYTE-TUMOR INTERACTION. A GENERAL METHOD FOR CELLULAR IMMUNOABSORPTION. *(E.)* Stulting, R. D. (Duke U. Med. Ctr., Durham, N.C.) and G. Berke. *J Exp Med* 137(4): 932-942, 1973.

The binding of sensitized lymphocytes to tumor cells that leads to tumor cell lysis *in vitro* has been investigated using poly-L-lysine-fixed tumor cell monolayers and lymphocytes obtained from the anatomical site of tumor allograft rejection. The results show that magnesium is an important prerequisite for this interaction and that the extent of lymphocyte-tumor cell binding depends upon temperature as well as pH. Binding can occur in the absence of serum, although serum factors are necessary for the completion of the cytolytic process. The poly-L-lysine technique is applicable to the formation of confluent monolayers with virtually any normal or neoplastic cell type, including those that are otherwise nonadherent to surfaces. Cells immobilized by this technique can be used for the specific immunoabsorption and subsequent recovery of effector lymphocytes sensitized against transplantation or tumor cell antigens.

1504 ANDENOVIRUS TYPE 12 SURFACE ANTIGEN DETECTED BY IMMUNOFLUORESCENCE IN INFECTED KB CELLS. *(E.)* Vasconcelos-Costa, J. (Lab. Cell Biol., Gulbenkian Inst. Sci., Oeiras, Portugal), A. Geraldes and Z. G. Carvalho. *Virology* 52(2): 337-343, 1973.

KB cells infected with adenovirus type 12 (Ad12) synthesize a new specific surface antigen, apparently the same as that of cells from hamster tumors induced by Ad12. The antigen is detectable by 12 hr after infection, reaches a peak at the 18th hour, and then decays rapidly, disappearing by the 24th hour. The kinetics of synthesis of this antigen are not superimposable to that of T antigen. The proportion of surface antigen-positive cells is dependent on the multiplicity of infection. The antigen in infected KB cells can no longer be detected after absorption of serum with cells from a hamster tumor induced by Ad12, previously tested for surface antigen. This strongly suggests that the surface antigens induced in the two types of cell-virus interaction are identical. There is no cross reactivity with sera against polyoma or SV40-induced surface antigens, and absorption of anti-Ad12 surface antigen antiserum with SV40-transformed hamster cells does not eliminate the activity against infected KB cells and Ad12-tumor cells.

1505 DETECTION OF A HUMAN SPLENO-THYMIC ANTIGEN AND ITS PRESENCE IN LEUKEMIA AND IN THE FETAL THYMUS. *(Fr.)* Martin, J.-P (Dept. Ctr. Blood Transf. Hum. Genet., Bois-Guillaume, France), R. Charlionet and C. Ropartz. *C R Acad Sci [D] (Paris)* 276(5):865-867, 1973.

1506 YEAST AND ANTITUMOR VACCINATION, NEW ACHIEVEMENTS. *(Fr.)* Vermeil, C. (Lab. Parasit. Exot. Path., Nantes, France), O. Morin, M. Van Roy and M. Pidoux. *C R Acad Sci [D] (Paris)* 276(1):129-132, 1973.

1507 SERUM CYTOTOXIN LEVELS OCCURRING IN RATS DURING THE DEVELOPMENT OF ASCITIC TUMORS. *(Rus.)* Kondrat'yeva, A.F. (N.N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Vopr Onkol* 18(12): 50-54, 1972.

1508 A STUDY OF GROUP-SPECIFIC AND TYPE-SPECIFIC ANTIGENS DURING THE DEVELOPMENT OF CERTAIN VIRAL LEUKEMIAS IN MICE. *(Rus.)* Gurtsevich, V. E. (Inst. Exp. Clin. Oncol. Moscow, USSR). *Vopr Onkol* 19(1):66-70, 1973.

1509 A STUDY OF THE CAPACITY OF FETAL ANTIGENS TO INDUCE TRANSPLANTATION RESISTANCE AGAINST HUMAN ADENOVIRUS-INDUCED TUMORS. *(Rus.)* Vardosanidze, E. Sh. (Inst. Oncol. Tbilisi, Georgian SSR, USSR), V. V. Meunargiya, V. Sh. Serebryakov and G. S. Kopilashvili. *Soobshch Akad Nauk Gruz SSR* 68(3):717-719, 1972.

1510 COMPARATIVE EVALUATION OF PARTICULATE AND BACTERIAL OPSONIZATION BY PLASMA OF NORMAL AND NEOPLASTIC INDIVIDUALS. *(E.)* Pisano, J. C. (Tulane U. Sch. Med., New Orleans, La.), J. P. Jackson and N. R. Di Luzio. *Proc Soc Exp Biol Med* 142(4):1355-1358, 1973.

1511 GONADOTROPIC ACTIVITIES OF THYROTROPIC TUMORS; DEMONSTRATION BY IMMUNOHISTOCHEMICAL STAINING. *(E.)* Furth, J. (Columbia U. Coll. Phys., Surg., New York, N.Y.), P. Moy, D. S. Schalch and G. Ueda. *Proc Soc Exp Biol Med* 142(4):1180-1184, 1973.

1512 CANCER CAUSED BY AN INHERITED SELECTIVE DEFECT IN IMMUNOLOGICAL SURVEILLANCE. *(E.)* Gleichmann, E. (Med. Sch., Hannover, West Germany) and H. Gleichmann. *Lancet* (7808):883, 1973.

1513 ROSETTE FORMATION AND T CELLS. *(E.)* Gergely, P. (I Dept. Med., U. Debrecen, Hungary), G. Szegedi, B. Fekete, G. Szabo and G. Petranyi. *Lancet* (7808):883, 1973.

1514 LYMPHOCYTE TRANSFORMATION IN KAPOSI'S SAR-

COMA. (*E.*) Taylor, J. F. (Dept. Orthopaedic Surg., U. Liverpool, England). *Lancet* (7808):883-884, 1973.

1515 PARADOXICAL EFFECT OF ANTI-THYMOCYTE SERUM ON THE THYMUS. (*E.*) Cantor, H. (Stanford U. Sch. Med., California) and R. Asofsky. *Nature* 243(5401):39-41, 1973.

1516 REPRESSION OF PROPAGATION OF EHRLICH ASCITES TUMORS BY MEANS OF ATTENUATING TUMOR CELLS. II. (*E.*) Maekawa, K. (Fac. Agriculture, Kyushu U., Japan) and A. Momii. *Proc Jap Acad* 48(9):690-694, 1972.

1517 LEUKAEMIA AND IMMUNODEFICIENCY. (*E.*) Parker, J. E. (Lions Gate Hosp., North Vancouver, Canada) and R. A. Rockerbie. *Lancet* (7810):1002, 1973.

1518 LYMPHOCYTE TRANSFORMATION IN MALIGNANT DISEASE. (*E.*) Lauder, I. (Royal Victoria Infirmary, Newcastle upon Tyne, England) and G. Bone. *Lancet* (7809):937, 1973.

1519 SERUM ALPHA FETOPROTEIN CONCENTRATION AND TUMOR GROWTH DISSOCIATION IN A PATIENT WITH OVARIAN TERATOCARCINOMA. (*E.*) Esterhay, R. J., Jr. (Baltimore Cancer Res. Ctr., Md.), H. M. Shapiro, J. C. Sutherland, K. R. McIntire and P. H. Wiernik. *Cancer* 31(4):835-839, 1973.

1520 USE OF PLASMA-FREE MEDIUM FOR THE *IN VITRO* CULTURE OF HUMAN LYMPHOCYTES. (*E.*) Bagai, R. C. (Ottawa Gen. Hosp., Ontario, Canada), T. H. M. Stewart and J. E. Harris. *Cancer* 31(4):882-888, 1973.

1521 DISTRIBUTION OF ANTIGEN FRAGMENTS OF IgG AMONG THE NUCLEOTIDES OF RNA. (*E.*) Nachkov, D. (Inst. Infectious, Parasitic Dis., Sofia, Bulgaria), L. Wassilewa, K. Tsankova and L. Christophorov. *Experientia* 15(2):209, 1973.

1522 INTERCELLULAR LACUNAE: SEQUESTERED MICROENVIRONMENTS IN STIMULATED LEUKOCYTE CULTURES. (*E.*) Robbins, J. H. (Natl. Cancer Inst., Bethesda, Md.), P. M. Elias and L. Ornstein. *Experientia* 15(2):212-213, 1973.

1523 PASSIVE ADMINISTRATION OF ANTISERUM AND COMPLEMENT IN PRODUCING ANTI-EL4 CYTOTOXIC ACTIVITY IN THE SERUM OF C57BL/6 MICE. (*E.*) Drake, W. P. (Natl. Cancer Inst., Bethesda, Md.), P. C. Ungaro and M. R. Mardiney, Jr. *J Natl Cancer Inst* 50 (4):909-914, 1973.

1524 L-ASPARAGINASE-INDUCED ALTERATIONS OF BURKITT'S LYMPHOMA CELLS. (*E.*) Perez-Bercoff, R. (Inst. Virol., U. Rome, Italy), G. Carrara, L. Cioe, G. Rita, A. Caputo and L. Castelli. *J Natl Cancer Inst* 50(4):833-840, 1973.

1525 THE INCIDENCE AND SIGNIFICANCE OF AUSTRALIA ANTIGEN IN CANCER PATIENTS. (*E.*) Al-Sarraf, M. (Oncol. Div., Wayne State U., Detroit, Mich.) K. Kithier, S. Sardesai, V. K. Vaitkevicius and M. D. Poulik. *Oncology* 27(2):128-136, 1973.

1526 DETERGENT EFFECTS ON THE DEVELOPMENT OF ANTIBODY-PRODUCING CELLS *IN VITRO*: A MODEL FOR THE STUDY OF CHEMICAL EFFECTS ON CELL DIFFERENTIATION IN A DEVELOPING SYSTEM. (*E.*) Garry, V. F. (U. Kansas Med. Ctr., Kansas City) and E. M. Uyeki. *Oncology* 27(2):160-167, 1973.

1527 BIOSYNTHESIS, INTRACELLULAR TRANSPORT, AND SECRETION OF IMMUNOGLOBULINS. EFFECT OF 2-DEOXY-D-GLUCOSE IN TUMOR PLASMA CELLS PRODUCING AND SECRETING IMMUNOGLOBULIN G1. (*E.*) Melchers, F. (Basel Inst. Immunol., Switzerland). *Biochemistry* 12(8):1471-1476, 1973.

1528 A FACTOR PREVENTING THE DEVELOPMENT OF LUNG METASTASES IN RATS WITH SARCOMAS. (*E.*) Proctor, J. W. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England), C. M. Rudenstam and P. Alexander. *Nature* 242(5392):29-31, 1973.

1529 HAPTEN-REACTIVE HELPER LYMPHOCYTES. (*E.*) Rubin, B. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden) and H. Wigzell. *Nature* 242(5398):467-468, 1973.

1530 DEPRESSION OF IMMUNE RESPONSE TO MOLONEY LEUKAEMIA VIRUS BY MALARIAL INFECTION. (*E.*) Bomford, R. (Imperial Cancer Res. Fund, London, England) and N. Wedderburn. *Nature* 242(5398):471-473, 1973.

1531 EVIDENCE FOR THE SUPPRESSION OF THE DEVELOPMENT OF CYTOTOXIC LYMPHOID CELLS IN TUMOR IMMUNIZED MICE. (*E.*) Martin, W. J. (Natl. Cancer Inst. Bethesda, Md.), E. Esber and J. R. Wunderlich. *Fed Proc* 32(2):173-179, 1973.

1532 IMMUNITY IN THE ACUTE LEUKAEMIAS. (*E.*) Oliver, R. A. M. (Mayday Hosp., Thornton Heath, Surrey, England). *Lancet* (7806):771, 1973.

1533 ROSETTE-FORMING CELLS IN MICE IMMUNIZED WITH SHEEP AND CHICKEN RED CELLS. (*E.*) Laskov, R. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel) and E. Simon. *Isr J Med Sci* 9(2):175-184, 1973.

1534 INTRACELLULAR IgA IMMUNOGLOBULIN CRYSTALS IN CHRONIC LYMPHOCYTIC LEUKAEMIA. (*E.*) Cawley,

J. C. (Dept. Med., U. Cambridge, England), C. R. Barker, R. D. Britchford and J. L. Smith. *Clin Exp Immunol* 13(3):407-416, 1973.

1535 POLYSOMES OF CULTURED NORMAL AND LEUKAEMIC LYMPHOCYTES: RESPONSE TO PHYTOHAEMAGGLUTININ. *(E.)* Rabinowitz, Y. (VA Hosp., Hines, Ill.), P. Justice, P. Wong and D. Y. Y. Hsia. *Br J Haematol* 24:355-365, 1973.

1536 THE IMPAIRED RESPONSIVENESS OF CHRONIC LYMPHATIC LEUKEMIA LYMPHOCYTES TO ALLOGENEIC LYMPHOCYTES. *(E.)* Smith, M. J. (Gundersen Clin., Ltd., La Crosse, Wisc.), E. Browne and A. Slungaard. *Blood* 41(4):505-509, 1973.

1537 RESEARCH ON THE CARCINOEMBRYONIC ANTIGEN IN GASTRO-INTESTINAL CANCERS. *(Fr.)* Burtin, P. (Inst. Sci. Res. Cancer, Villejuif, France) and S. von Kleist. *Ann Anat Pathol (Paris)* 17(3):419-424, 1972.

1538 RELATION BETWEEN CELLULAR IMMUNITY TO NEPHROBLASTOMA AND THE PHASE OF THE DISEASE. *(E.)* Jereb, B. (Karolinska Inst., Stockholm, Sweden), V. Diehl, L. Ahström and I. Juhlin. *Acta Paediatr Scand* 61:709-714, 1972.

1539 STUDIES IN MULTIPLE MYELOMA. I. CHARACTERISTICS BY IMMUNOGLOBULIN CLASS. *(E.)* Murphy, W. M. (Cleveland Clin., Ohio) and S. D. Deodhar. *Cleve Clin Q* 40(1):1-7, 1973.

1540 STUDIES IN MULTIPLE MYELOMA. II. LIGHT CHAINS. *(E.)* Murphy, W. M. (Cleveland Clin., Ohio) and S. D. Deodhar. *Cleve Clin Q* 40(1):9-14, 1973.

1541 DETECTION OF TUMOUR-ASSOCIATED TRANSPLANTATION ANTIGEN OF BO-IV CARCINOMA ON SOMATIC CELLS OF TUMOUR-FREE SYNGENEIC MICE. II. SEARCH FOR A COMMON ANTIGEN IN SOME ORGANS AND SPONTANEOUS MAMMARY CARCINOMAS. *(E.)* Rejthar, A. (Fac. Med., J. Ev. Purkyne U., Brno, Czechoslovakia) and J. Wotke. *Folia Biol (Praha)* 19(1):52-57, 1972.

1542 CYTOTOXIC LYMPHOCYTES. II. ANTAGONISM AND SYNERGISM BETWEEN SERUM FACTORS AND LYMPHOCYTES OF PATIENTS WITH SARCOMAS AS TESTED AGAINST CULTURED TUMOR CELLS. *(E.)* Sinkovics, J. G. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), N. Ahmed, M. J. Hrgovcic, J. R. Cabiness and J. R. Wilbur. *Tex Rep Biol Med* 30(4):347-360, 1972.

1543 ANTIBODIES TO CELL ORGANOIDS IN THE SERA OF NASOPHARYNGEAL CARCINOMA PATIENTS AND OF SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS. *(E.)* Utsumi, K. R. (Aichi Cancer Ctr. Res. Inst., Nagoya, Japan), T. O. Yoshida, M. Sekikawa and G. Klein. *Scand J Immunol* 2(2):159-171, 1973.

1544 FACILITATION OF MACROPHAGE-MEDIATED DESTRUCTION OF ALLOGENEIC FIBROSARCOMA CELLS BY TUMOR-ENHANCING IgG$_2$ *IN VITRO*. *(E.)* Cruse, J. M. (U. Mississippi Grad. Sch., University), H. D. Whitten, G. K. Lewis and E. S. Watson. *Transplant Proc* 5(1):961-967, 1973.

1545 APPARENT HL-A5 DEFICIENCY IN MALIGNANT MELANOMA. *(E.)* Clark, D. A. (New England Med. Ctr. Hosp., Boston, Mass.), T. Necheles, L. Nathanson and E. Silverman. *Transplantation* 15(3):326-328, 1973.

1546 THETA ANTIGEN ON A MURINE LYMPHOSARCOMA. *(E.)* Merino, F. (Sch. Med., State U. New York, Buffalo), C. J. Abeyounis and F. Milgrom. *Transplant Proc* 5(1):975-976, 1973.

1547 IMMUNOSUPPRESSION AND CANCER. *(E.)* Penn, I. (U. Colorado Sch. Med., Denver) and T. E. Starzl. *Transplant Proc* 5(1):943-947, 1973.

1548 EFFECT OF IMMUNIZATION WITH NORMAL SYNGENEIC TISSUES ON TUMOR INDUCTION IN MICE. *(E.)* Fel', V. Ya. (Inst. Cytology, Acad. Sci. USSR, Leningrad) and A. M. Malygin. *Bull Exp Biol Med (USSR)* 74(10):1306-1308, 1972.

1549 TUBULAR STRUCTURES IN HUMAN LYMPH NODE GERMINAL CENTRES SHARING ANTIGENIC DETERMINANTS WITH A β-GLOBULIN IN AN IgD MYELOMA SERUM. *(E.)* Ahlqvist, J. (Aurora Hosp., Helsinki, Finland) and J. A. Räsänen. *Ann Clin Res* 4(2):110-112, 1972.

1550 SURFACE MOLECULAR COMPONENTS OF EHRLICH ASCITES TUMOUR CELLS. *(E.)* Mehrishi, J. N. (U. Cambridge Addenbrooke's Hosp., England). *Blut* 24:388-397, 1972.

1551 TUMOR CELL DESTRUCTION BY MACROPHAGES. *(E.)* Holtermann, O. A. (Roswell Park Mem. Inst., Buffalo, N.Y.), G. P. Casale and E. Klein. *J Med* 3(4-5):305-309, 1972.

1552 CARCINOEMBRYONIC ANTIGEN AND UROTHELIAL CARCINOMA. *(E.)* Hall, R. R. (Royal Marsden Hosp., London, England), D. J. R. Laurence, A. M. Neville and D. M. Wallace. *Br J Urol* 45(1):88-92, 1973.

1553 SYNGENEIC IMMUNE SERUM SPECIFICALLY INHIBITS THE MOTILITY OF TUMOUR CELLS. *(E.)* Currie, G. A. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England) and G. C. Sime. *Nature [New Biol]* 241(113):284-285, 1973.

1554 PRODUCTION OF A NERVOUS-SYSTEM-SPECIFIC PROTEIN (14-3-2) BY HUMAN NEUROBLASTOMA CELLS IN CULTURE. *(E.)* Herschman, H. R. (Sch. Med., U. California, Los Angeles) and M. P. Lerner. *[New Biol]* 241(112):242-244, 1973.

1555 THE EFFECT OF TRANSPLANT AGAINST HOST REACTION ON THE GROWTH OF SYNGENIC SARCOMA IN MICE. *(Rus.)* Scherbakov, V. Ya. (Smolensk Med. Inst., USSR) and V. F. Semenkov. *Biull Eksp Biol Med* 74(2):70-73, 1973.

1556 IMMUNOGENICITY AND ENHANCEMENT OF NORMAL AND TUMOR TISSUE IN THE MOUSE. *(E.)* McKenzie, I. F. C. (Massachusetts Gen. Hosp., Boston) and G. D. Snell. *Transplant Proc* 5(1):953-956, 1973.

1557 IMMUNOLOGICAL STUDY OF TUMORS INDUCED BY HIGHLY ONCOGENIC FORMS OF ROUS VIRUS IN ADULT MICE. *(E.)* Kryukova, I. N. (Acad. Med. Sci., USSR, Moscow), I. B. Obukh and O. V. Babkova. *Bull Exp Biol Med (USSR)* 74(9):1078-1081, 1973.

1558 ACQUIRED C1 INHIBITOR DEFICIENCY IN LYMPHOSARCOMA. *(E.)* Caldwell, J. R. (Harvard Med. Sch., Boston, Mass.), S. Ruddy, P. H. Schur and K. F. Austen. *Clin Immunol Immunopathol* 1(1):39-52, 1972.

1559 MALIGNANT LYMPHOMA WITH A MONOCLONAL $F(ab)_\mu$ FRAGMENT. *(E.)* DeCoteau, W. E. (State U. New York, Sch. Med., Buffalo), N. J. Calvanico and T. B. Tomasi, Jr. *Clin Immunol Immunopathol* 1(2): 190-202, 1973.

1560 LYMPHOCYTE SENSITIVITY AND CELLULAR IMMUNITY IN NEOPLASIA. *(E.)* Faguet, G. B. (Ohio State U., Coll. Med., Columbus), S. P. Balcerzak and A. F. Lobuglio. *Biomedicine* 19:43-47, 1973.

1561 NON-REACTIVITY OF INCOMPATIBLE LYMPHOID CELLS FROM F_1 MODIFIED RADIATION CHIMERAS TO HOST TUMOUR CELLS *IN VITRO*. *(E.)* Grant, C. K. (Chester Beatty Res. Inst., Sutton, Surrey, England) and P. L. T. Ilbery. *Biomedicine* 19:56-60, 1973.

1562 MACROPHAGE DEPENDENT PROTECTION OF TUMOUR CELLS. *(E.)* Hersey, P. (Radcliffe Infirmary, Oxford, England) and I. C. M. MacLennan. *Immunology* 24(2):385-393, 1973.

1563 PROLONGED SURVIVAL OF MURINE FIBROSARCOMA ALLOTRANSPLANTS BY ANTI-ENHANCING Fab ALLOANTIBODY. *(E.)* Cruse, J. M. (Dept. Biol., Grad. Sch., Mississippi, University), G. K. Lewis, H. D. Whitten, E. S. Watson, R. B. Hester and J. F. Fields. *Immunol Commun* 1(4):367-373, 1972.

1564 EXPRESSION OF H-2 ANTIGENS DURING GROWTH OF CULTURED TUMOR CELLS. *(E.)* Götze, D. (Scripps Clin., Res. Fdn., La Jolla, Calif.), M. A. Pellegrino, S. Ferrone and R. A. Reisfeld. *Immunol Commun* 1(6):533-544, 1972.

1565 PRODUCTION OF ANTIBODIES TO CARCINOEMBRYONIC ANTIGEN IN TOLERANT RABBITS. *(E.)* Saravis, C. A. (Boston City Hosp., Mass.), H. Z. Kupchik and N. Zamcheck. *Immunol Commun* 1(4):395-406, 1972.

1566 MEMBRANE ANTIGENS SPECIFIC FOR HUMAN LYMPHOID CELLS IN THE DIVIDING PHASE. *(E.)* Thomas, D. B. (Middlesex Hosp. Med. Sch., London, England) and B. Phillips. *J Exp Med* 138(1):1973.

1567 EXPERIMENTAL METASTASIZING IN SYNGENEIC AND AUTOCHTHONOUS TUMOUR-HOST SYSTEMS. *(E.)* Arnold, W. (Central Inst. Cancer Res., Berlin, East Germany) and A. Graffi. *Acta Biol Med Germ* 29(6): 7-13, 1972.

1568 AUGMENTATION OF IMMUNE ACTIVITY BY ELIMINATION OF ANTIBODY AND ITS IMPLICATIONS IN CANCER. *(E.)* Rose, S. (M.D. Anderson Hosp., Tumor Inst., Houston, Tex.). *J Surg Oncol* 5(2):137-166, 1973.

1569 DEVELOPMENT OF A SIMPLIFIED ISOTOPIC ANTIGLOBULIN ASSAY FOR THE STUDY OF TUMOR-SPECIFIC ANTIBODY. *(E.)* Burdick, J. F. (Natl. Cancer Inst., Bethesda, Md.), S. A. Wells, Jr., A. M. Cohen and A. S. Ketcham. *J Surg Res* 14(3):200-206, 1973.

1570 THERAPEUTIC AND IMMUNOLOGIC EFFECTS OF IODOACETATE IN MATURE AKR MICE. *(E.)* Rheins, M. S. (Dept. Microbiol., Ohio St. U., Columbus) and A. D. Barker. *Experientia* 29(4): 476-477, 1973.

1571 STRUCTURAL CONFORMATION OF TUMOUR ANTIGEN. *(E.)* Mitchell, H. (Charing Cross Hosp., London, England). *Lancet* (7811):1061, 1973.

1572 β_2-MICROGLOBULIN PRODUCTION AND SECRETION BY LYMPHOCYTES IN CULTURE. *(E.)* Poulik, M. D. (William Beaumont Hosp., Royal Oak, Mich.) and A. D. Bloom. *J Immunol* 110(5):1430-1433, 1973.

1573 SUPPRESSION OF THE ANTIBODY RESPONSE TO SRBC WITH $F(ab')_2$ AND IgG *IN VITRO*. *(E.)* Wason, W. M. (Argonne Cancer Res. Hosp., Chicago, Ill.) and F. W. Fitch. *J Immunol* 110(5):1427-1429, 1973.

1574 SPECIFICITY OF METAL ION INTERACTION WITH CONCANAVALIN A. *(E.)* Shoham, M. (Dept. Biophys., Weizmann Inst. Sci., Rehovot, Israel), A. J. Kalb and I. Pecht. *Biochemistry* 12(10):1914-1917, 1973.

1575 B.C.G. IN MALIGNANT MELANOMA. *(E.)* Baker, M. A. (Toronto Western Hosp., Canada) and R. N. Taub. *Lancet* (7812):1117-1118, 1973.

1576 "FORBIDDEN CLONES" AND AUTOIMMUNITY. *(E.)* Cox, K. O. (Perth Med. Ctr., Australia) and D. Keast. *Lancet* (7812):1123-1124, 1973.

See also:

* (Rev): 1201, 1204, 1205, 1210
* (Chem): 1257, 1259, 1281
* (Viral): 1321, 1340, 1341, 1345, 1362, 1386, 1422

1577 THE FAILURE OF NORMAL HAEMOPOIESIS IN RATS DURING THE DEVELOPMENT OF ACUTE LEUKAEMIA. *(E.)* Hoelzer, D. (Clin. Res. Ctr., U. Ulm, Germany) and E. B. Harriss. *Acta Haemat* 49(1):36-47, 1973.

Rats were inoculated i.v. with 10^7 leukocytes from donors with myelomonocytic leukemia (originally induced by ethylnitrosourea). Beginning 12 hr postinjection and continuing for six days, blood and bone marrow samples were taken and hemopoiesis was studied. In the erythropoietic series, basophilic normoblasts were unaffected in leukemic rats for two days postinjection; they then decreased to one tenth of initial numbers. Polychromatic normoblasts began to decrease later. In the myelopoietic series, cells in dividing stages showed no changes until 4.5 days postinjection. At that time a slight decrease in these cells was seen. Marrow granulocytes decreased in number, especially between two and four days postinjection. Megakaryocytes and platelets did not change drastically in leukemic mice. Bone marrow lymphocytes increased seven- to eight-fold beginning 3.5 days postinjection.

1578 DEVELOPMENT AND REGRESSION OF MURINE LEUKEMIA INDUCED BY FRIEND VIRUS COMPLEX. *(E.)* Karnjanaprakorn, C. (Mahidol U., Bangkok, Thailand) and S. Thomson. *Proc Soc Exp Biol Med* 143(1):300-303, 1973.

The roles which inoculating dose size and mouse strain play in the development and regression of Friend virus complex (FV)-induced leukemia have been studied. Large inocula (5000 FFU) produced a rapid development of leukemia which progressed to death of inbred A/J mice. Inoculation with 5 FFU also caused progressive disease but at a slower rate than that resulting from the higher FV dose. Although inoculation of 0.5 FFU produced leukemia, tumor cell growth increased very slowly and tumors were no longer present after 2.5 months. Thus, the possibility for regression of FV-induced tumors was dose-dependent. (A/J x C57BL/6J) F_1 mice and their reciprocal hybrids were about 40 times more resistant to FV than A/J mice. Hybrid mice rapidly developed leukemia after inoculation of 500 FFU of FV but the leukemia regressed.

1579 LYMPHANGIOMYOMA: CLINICOPATHOLOGIC STUDY AND ULTRASTRUCTURAL CONFIRMATION OF ITS HISTOGENESIS. *(E.)* Wolff, M. (Coll. Physicians Surg., Columbia U., New York, N.Y.). *Cancer* 31(4):988-1007, 1973.

An analysis of two new cases of lymphangiomyoma, as well as of 28 previously reported ones, is presented. One of the new cases was studied ultrastructurally, with findings that support the designation "lymphangiomyoma", that is, smooth muscle and lymphatic channels. All 30 cases reported to date have occurred in females. The condition consists of one or more nodules of tumor-like proliferation of smooth muscle fascicles, divided from each other by lymphatic channels, and involves major lymphatic trunks in the retroperitoneum or mediastinum, lymph nodes in these regions, and in about two-thirds of cases, lymphatics of the lung. Symptons were frequently related to chylothorax, pneumothorax and/or pulmonary parenchymal involvement by the disease. The pulmonary lesion in lymphangiomyoma is morphologically indistinguishable from that found in some patients with tuberous sclerosis. Several other clinical and anatomical features are shared by lymphangiomyoma and tuberous sclerosis; the coexistence of the two entities in a single patient, which was recently reported, gives additional support to the concept that they are related disorders. Lymphangiography has been used as a diagnostic tool in six of the reported cases. Although diverse modes of therapy have been applied, those patients with the longest survival had received radiotherapy following various surgical procedures.

1580 *IN VITRO* AND *IN VIVO* LABELLING OF ANIMAL TUMORS WITH TRITIATED THYMIDINE. *(E.)* Denekamp, J. (Mount Vernon Hosp., Northwood, Middlesex, England) and R. F. Kallman. *Cell Tissue Kinet* 6(3):217-227, 1973.

An attempt was made to correlate *in vivo* ^{3}H-thymidine labeling kinetics of tumors in tumor-bearing mice with *in vitro* labeling kinetics of sections of the same types of tumors. Labeling indices were determined from autoradiographs of tissue sections. The labeling indices of the ten different sarcomas, lymphomas and carcinomas studies varied over a wide range both *in vivo* and *in vitro*. The *in vitro* and *in vivo* specimens also showed wide variations in labeling index from one area to another within the same specimens. No consistent evidence for variation for labeling index across a tumor was found although a few tumor sections tended to have higher peripheral values than control labeling indices. Of 27 tumor samples tested, only three *in vivo* and *in vitro* pairs gave identical labeling indices. The results suggested that there was a tendency in small tumors for *in vitro* slices to give lower labeling indices than the *in vivo* slices, with exactly the opposite for large tumors. There was no apparent relationship between histologic type and the accuracy with which the *in vitro* labeling simulated that measured *in vivo*.

1581 MECHANISMS OF ENHANCED INTRAHEPATIC METASTASIS IN SURFACTANT-TREATED HAMSTERS; AN ELECTRON MICROSCOPY STUDY. *(E.)* Cotmore, S. F. (Inst. Cancer Res., London, England) and R. L. Carter. *Int J Cancer* 11(3):725-738, 1973.

The effects of administration of the surfactant, triton-WR on the growth of metastatic tumors was investigated. A metastasizing lymphoma (ML) in female hamsters approximately 8 wk old and weighing 100 g, grafted into the subcutaneous tissues, gave rise to moderate numbers of nodular intrahepatic deposits; but when the animals were treated with triton-WR 1339 (4 i.p. injections, 1,000 mg/kg body weight) the liver was massively and diffusely involved with secondary tumor. The results of serial ultra-structural studies indicate some explanations for this finding. The major factor appears to be that triton-WR inflicts mechanical damage on cells of the sinusoidal lining, primarily as a result of lysosomal lesions which distend and distort the cells; this substantially facilitates the egress of tumor cells from the circulation into the extra-hepatic tissue. Two possible subsidiary mechanisms are also considered: (1) Extensive lysosomal damage seen in Küpffer cells and in free, intravascular macrophages may perhaps result in impaired phagocytic activity within the liver; (2) Drug-induced lysosomal damage in hepatic parenchymal cells may favor the establishment of tumor cells lying in the extravascular space. Triton-WR is reported to exert an antifibrinolytic effect but this is apparently irrelevant to the enhanced intrahepatic growth of ML; there is no evidence that fibrin plays any part in the metastatic spread of this tumor.

1582 A REAPPRAISAL OF HISTOPATHOLOGY IN LUNG CANCER AND CORRELATION OF CELL TYPES WITH ANTECEDENT CIGARETTE SMOKING. *(E.)* Yesner, R. (VA Hosp., West Haven, Conn.), N.A. Gelfman and A. R. Feinstein. *Am Rev Resp Dis* 107(5):790-797, 1973.

The histologic types of 449 lung cancers were reevaluated according to WHO criteria approximately ten yr after the original specimens had been classified (1953 to 1959). Since the WHO nomenclature was much more detailed than that originally used, the rate of agreement between the new and old classifications was 64%. Most of the changes were due to reclassification of many tumors originally designated as "epidermoid" or "well differentiated epidermoid" into other categories. Among 42 noncigarette smokers, including nine pipe or cigar smokers, the predominant tumor cell type was adenocarcinoma in both the old and new classifications. Of all histologic types, only small cell undifferentiated cancer showed a positive correlation with the extent of cigarette smoking obtained from the history. Prevalence ranged from 10% in nonsmokers to 29% in heavy smokers (>2 packs/day). The rate of poorly differentiated epidermoid carcinoma was lower in the high smoking groups, while large cell carcinoma and adenocarcinoma rates showed no consistent relationship to the amount of smoking.

1583 METABOLIC CONTROLS IN PRECANCEROUS LIVER - IV. LOSS OF FEEDBACK CONTROL OF CHOLESTEROL SYNTHESIS AND IMPAIRED CHOLESTEROL UPTAKE IN ETHIONINE-FED RATS. *(E.)* Horton, B. J. (U. Adelaide, South Australia) and J. R. Sabine. *Eur J Cancer* 9(1):11-17, 1973.

Several aspects of the control of cholesterol synthesis were examined *in vivo* (measured as plasma levels of 7-dehydrocholesterol following a single dose of trans-1,4-*bis*(2-chlorobenzylaminomethyl) cyclohexane dihydrochloride, an inhibitor of cholesterogenesis) in the livers of rats fed the hepatocarcinogen, ethionine. Male, hooded-Wistar rats, aged 2-3 months at the beginning of each experiment, were fed a commercial laboratory ration *ad libitum*, supplemented with DL-ethionine. Within 2 wk of beginning ethionine treatment hepatic cholesterol synthesis was increased and feedback control over this synthesis was lost. (Inhibition of cholesterol synthesis by cholesterol feeding is referred to as "feedback" inhibition.) The degree of loss of control of cholesterol synthesis did not correlate with the variable toxicity of the ethionine to the test rats. These studies indicate that feeding the hepatocarcinogen, ethionine, causes extensive loss of control of hepatic cholesterol synthesis. There appears to be two possible explanations for these effects: 1) alteration of the external cell membrane, resulting in impaired permeability of dietary cholesterol and therefore no feedback control; and 2) alteration in the microsomal membane which prevents it from recognizing and taking up dietary cholesterol.

1584 FLORID SUBAREOLAR DUCT PAPILLOMATOSIS (NIPPLE ADENOMA) AND MAMMARY CARCINOMA: REPORT OF THREE CASES. *(E.)* Bhagavan, B. S. (Sinai Hosp. Baltimore, Md.), A. Patchefsky and L. G. Koss. *Hum Pathol* 4(2):289-295, 1973.

Three rare cases of subareolar florid duct papillomatosis (nipple adenoma) associated with breast cancer are presented. In two of the women, aged 43 and 67, the areas of papillomatosis were directly adjacent to and continuous with areas of intraductal carcinoma. The third case, a 63-year-old female, showed noncontinuous areas of papillomatosis and carcinoma. These observations suggest that subareolar florid duct papillomatosis is a premalignant state.

1585 THE STRUCTURE AND DEVELOPMENT OF A GENETIC TUMOUR OF THE PEA. *(E.)* Burgess, J. (John Innes Inst., Norwich, England) and E. N. Fleming. *Protoplasma* 76(3-4):315-325, 1973.

1586 CEREBELLAR HEMANGIOBLASTOMA: HISTOGENESIS OF STROMA CELLS. *(E.)* Kawamura, J. (U. Maryland Sch. Med., Baltimore), J. H. Garcia and Y. Kamijyo. *Cancer* 31(6):1528-1540, 1973.

1587 SQUAMOUS CELL CARCINOMA OCCURRING IN ORAL LICHEN PLANUS. *(E.)* Stillman, M. A. (New York U. Sch. Med., New York), R. S. Bart and A. W. Kopf. *Cutis* 11(4):487-490, 1973.

See also:

* (Viral): 1343, 1393

1588 CANCER MORTALITY AMONG URANIUM MILL WORKERS. *(E.)* Archer, V. E. (Dept. Hlth., Educ. Welfare, Salt Lake City, Utah), J. K. Wagoner and F. E. Lundin, Jr. *J Occup Med* 15(1):11-14, 1973.

Mortality from all causes was determined from 1950 through 1967 for a group of 662 white male uranium mill workers living in the Colorado Plateau states. Of the 104 deaths occurring among the mill workers during this time period, the only significant increase over that of the general white male population of this area was death due to malignant diseases of the lymphatic and hematopoietic tissue other than leukemia. All men who died of this type of malignant disease either worked directly with the raw ore or worked within the processing plant during 1943-1953 when industrial hygiene practices at mills were minimal. None of the men worked in the furnace area where exposure to dust or fumes was greatest. Previous results from animal experiments implicated excessive irradiation of lymph nodes by thorium-230 as a possible etiologic factor.

1589 EPIDEMIOLOGY OF CANCER OF THE PANCREAS. *(E.)* Wynder, E. L. (Amer. Hlth. Fdn., New York, N.Y.), K. Mabuchi, N. Maruchi and J. G. Fortner. *J Natl Cancer Inst* 50(3):645-667, 1973.

Epidemiological features of pancreatic cancer were observed in a survey of 100 males and 42 females with adenocarcinoma of the pancreas, four males and seven females with islet cell cancer, and seven males and three females with cancer of the ampulla of Vater. The sex ratio (male/female) for pancreatic cancer among patients of all ages was 2.4:1; the sex ratio for patients less than 50 yr old was 3.8:1. No significant differences were found between cancer and control groups in socioeconomic status. Cigarette smoking and cigar smoking were both associated with high pancreatic cancer risks. Among women, correlations with pancreatic cancer were found for early-onset diabetes and, possibly, for cholecystectomy. It was hypothesized that carcinogens in the bile act on the pancreas via reflux into the pancreatic duct.

1590 LEUKEMIA AND OCCUPATIONS. *(E.)* Viadena, E. (Park Mem. Inst., Buffalo, N.Y.) and I. D. J. Bross. *Preventive Med* 1(4):513-521, 1972.

The Tri-State Leukemia Survey, including leukemia cases recorded in 1959-1962 from Minnesota, Baltimore and New York State excluding New York City, was used to study leukemia risk among various occupational groups. A total of 1345 leukemia cases were matched with 1237 randomly selected controls. No relationship was found between occupation and leukemia for women. Among men, construction workers showed a higher leukemia incidence than other groups, painters accounting for most of the excess. The relative risk of leukemia among painters was about three times the risk of persons in other occupations. Nonsignificant values as far as increased probability of getting leukemia, were found among printers.

1591 TESTICULAR CANCER IN CALIFORNIA FROM 1942 TO 1969: THE CALIFORNIA TUMOR REGISTRY EXPERIENCE. *(E.)* Krain, L. S. (UCLA Med. Ctr., Los Angeles, Calif.). *Oncology* 27(1):45-51, 1973.

A total of 1,278 testicular cancer cases from the California Tumor Registry was surveyed. Testicular cancer incidence was found to have increased 67% between 1942-1954 and 1955-1969. The incidence rate for testicular cancer in California was 2.6 cases/100,000 population. There was no significant difference in the distribution of testicular tumors as a whole, or of seminomas alone, in urban versus rural California counties. The median age at diagnosis for testicular cancer was 38 in 1942-1954 versus 34 in 1955-1969. Testicular cancer showed peak incidences in age groups 25-34 yr old and 75-84 yr old. Testicular cancer was rare in nonwhite patients.

1592 SURVEY OF CANCER OF THE OESOPHAGUS IN RELATION TO OTHER MALIGNANT NEOPLASMS. *(E.)* Zeynek, E. R. von (Glen Grey Mission Hosp., Lady Frere, Ciskei, South Africa). *S Afr Med J* 47(8):325-331, 1973.

The results of a survey of esophageal carcinoma and other malignant neoplasms carried on since 1958 in the Glen Grey district, one of the oldest Bantu reserves in South Africa, are reported. From 1958 to 1970 the occurrence of esophageal carcinoma as well as of other malignancies, appeared fairly static, with an annual incidence of 16.45 esophageal cancer cases per 100,000 people, representing 46% of all malignancies, and an annual incidence of 22.23 per 100,000 cases of all other malignant neoplasms. In 1971 a sharp rise, 40/100,000, in the incidence of esophageal cancer was noted with a further rise in 1972. It is suggested that this rise represents a genuine increase in the incidence of cancer of the esophagus. No concomitant rise of other neoplasms was recorded. An explanation for this increase cannot be offered. Esophageal carcinoma was virtually unknown in this area 30 or 40 yr ago.

1593 CELL KINETICS IN HUMAN ACUTE LYMPHOBLASTIC LEUKEMIA: COMPUTER SIMULATION WITH DISCRETE MODELING TECHNIQUES. *(E.)* Mauer, A. M. (Children's Hosp. Res. Fdn., Cincinnati, O.), C. F. Evert, Jr., B. C. Lampkin and N. B. McWilliams. *Blood* 41(1):141-154, 1973.

A model for cell kinetics of human acute lymphoblastic leukemia has been constructed with discrete modeling techniques for computer use with the GPSS/360 computer language. The background information used in the construction of the model, the development of the model itself, and the results of some explorations of leukemic cell kinetic behavior with the computer simulation process are described. The model has produced results corresponding to observed biological data. Mechanisms for the control of the growth of the leukemic cell population have been explored. Two potentially important regulatory mechanisms for control of cell population growth were indicated from the results of studies with the computer model: 1) the role of cell death; and 2) intracellular events after mitosis. When chemotherapeutic drug effects were simulated, an unsuspected effect of vincristine was suggested. This effect of vincristine on transformation of the resting cell to an active proliferative phase has been supported by studies of vincristine effect on blast transformation of phytohemagglutinin-stimulated lymphocytes. A single cell was found to take 3½ yr to grow to a population of 10^{12} cells, a clinically recognizable number. Although this observation was not confirmed by biological studies, the time period corresponds to the peak incidence of acute lymphoblastic leukemia in childhood, suggesting that a mutational event in a single cell could account for the leukemic process in childhood acute leukemia.

1594 ENVIRONMENTAL FACTORS AND CANCER OF THE COLON AND BREAST. *(E.)* Drasar, B. S. (St. Mary's Hosp. Med. Sch., London, England) and D. Irving. *Br J Cancer* 27(2):167-172, 1973.

Nutritional surveys using Food and Agriculture Organization data were compared with published cancer registry data to determine whether a correlation exists between living standards and diet and cancers of the breast, colon and stomach in 37 countries. Cancer of the colon was highly correlated with breast cancer; however, neither cancer was significantly associated with stomach cancer. Cancer of the stomach was not correlated with protein, fat, coloric or fiber content of the diet. Both colon and breast cancer, however, showed a high correlation with high fat and animal protein intake as well as other socioeconomic indicators of affluence.

1595 RACE AND PLACE IN THE ETIOLOGY OF NASOPHARYNGEAL CANCER: A STUDY BASED ON CALIFORNIA DEATH CERTIFICATES. *(E.)* Buell, P. (California Dept. Publ. Hlth., Berkeley). *Int J Cancer* 11(2):268-272, 1973.

1596 A CASE CONTROL STUDY OF CANCER OF THE PANCREAS. *(E.)* Wynder, E. L. (Am. Hlth. Fdn., New York, N.Y.), K. Mabuchi, N. Maruchi and J. G. Fortner. *Cancer* 31(3):641-648, 1973.

1597 CARCINOMA OF THE CECUM - INCIDENCE AND PROGNOSIS: A SURVEY IN THE STATE OF WEST VIRGINIA. *(E.)* Mendoza, C. B., Jr. (West Virginia U. Sch. Med., Morgantown), G. W. Easley, J. D. Carney and A. L. Watne. *Dis Colon Rectum* 16(2):94-97, 1973.

1598 SMOKING AND CANCER. *(E.)* Burch, P. R. J. (Gen. Infirmary, Leeds, England). *Lancet* (7809):939-940, 1973.

1599 ULTRASONIC VISUALIZATION IN DETERMINATION OF TUMOR GROWTH RATE. *(E.)* Heimburger, R. F. (Indiana U. Med. Sch., Indianapolis), R. C. Eggleton and F. J. Fry. *JAMA* 224(4):497-501, 1973.

1600 GROWTH OF ABNORMAL CELLS. *(E.)* Downham, D. Y. (Dept. Computational, Statistical Sci., Liverpool U., England) and R. K. B. Morgan. *Nature* 242(5399):528-530, 1973.

1601 CANCER STATISTICS 1973. *(E.)* Silverberg, E. (Am. Cancer Soc., New York, N.Y.) and A. I. Holleb. *CA* 23(1):2-27, 1973.

1602 CANCER FREQUENCY VARIATIONS AMONG AND WITHIN FAMILIES. *(E.)* Lynch, H. T. (Creighton U. Sch. Med., Omaha, Neb.), A. J. Krush and A. R. Kaplan. *Acta Genet Med Gemellol* 21(1-2):53-65, 1972.

1603 THE PROLIFERATION KINETICS OF L 1210 ASCITES TUMOUR. *(E.)* Dombernowsky, P. (Finsen Inst., Dept. Int. Med., Copenhagen, Denmark). *Acta Pathol Microbiol Scand* [*A*] 80(5):603-611, 1972.

1604 GROWTH PATTERN OF MENINGEOMAS PENETRATING THE SKULL BASE. *(E.)* Saksela, E. (Dept. Path., U. Helsinki, Finland), T. Holmström and B. Grahne. *Acta Otolaryngol (Stockh)* 74(5):363-370, 1972.

1605 MALIGNANT LYMPHOMA INCLUDING HODGKIN'S DISEASE OCCURRING IN THE VICINITY OF A LARGE MEDICAL CENTER (OLMSTED COUNTY, MIN., 1945 THROUGH 1969). *(E.)* Nobrega, F. T. (Mayo Clin. Fdn., Rochester, Minn.), R. A. Kyle and E. G. Harrison, Jr. *Cancer* 31(2):295-302, 1973.

1606 THE INCIDENCE OF CARCINOMA OF THE PROSTATE IN A BIOPSY MATERIAL. *(Ger.)* Lüchtrath, H. (Path. Inst., Koblenz, West Germany). *Urologe* 11(5):278-283, 1972.

See also:

- * (Rev): 1217
- * (Chem): 1258
- * (Viral): 1366

1607 BIOCHEMICAL CORRELATION OF ONCOGENESIS WITH ONTOGENESIS. *(E.)* Wu, C. (U. Michigan Med. Sch., Ann Arbor). *Int J Cancer* 11(2):438-447, 1973.

Literature is reviewed and new experimental evidence is presented to support the hypothesis that carcinogenesis involves a reversion of cellular control mechanisms from the adult to the fetal type. Results of studies on induction of 10 different enzymes (arginase, glutamine synthetase, benzpyrene hydroxylase, tryptophan pyrrolase, δ-aminolevulinic acid synthetase, adenylate kinase, glutaminase, DNA polymerase, pyruvate kinase and ornithine aminotransferase) in rat liver and kidney tumors have shown a close resemblance between the tumors and perinatal liver and kidney on the one hand, and a sharp contrast between the tumors and adult liver and kidney on the other. Similar results have been obtained from studies on isozyme distribution patterns. It thus seems possible that, during carcinogenesis, some regulatory mechanisms of the adult tissues undergo alterations such that their responsiveness to metabolic modulations resembles those which exist in perinatal life.

1608 BIOCHEMICAL ABNORMALITY OF DNA SYNTHESIS OF ERYTHROBLASTS IN ACUTE AND CHRONIC MYELOID LEUKEMIA. *(E.)* Ernst, P. (U. Hosp. Copenhagen, Denmark), M. Baccarani and S.-A. Killmann. *Blood* 41(4):497-504, 1973.

The *in vitro* incorporation of labeled DNA precursors (^{3}H-thymidine, ^{3}H-TDR) into basophilic erythroblasts was studied in four patients with acute and two patients with chronic myeloid leukemia (AML and CML) and was found to be low. Based on mitotic indices and the use of vincristine as a stathmokinetic tool, it was evident that brisk proliferation took place in this cell compartment. Microspectrophotometric determination of single-cell DNA content showed that a considerable fraction of the erythroid cells were in DNA synthesis without incorporating an exogeneously supplied DNA precursor. Following inhibition of the *de novo* synthesis of nucleoside triphosphates, the cells showed capacity for precursor incorporation. It is suggested that a biochemical abnormality, which leads to a high intranuclear nucleoside triphosphate pool, may inhibit nucleoside kinases and therefore, the utilization of labeled nucleosides, as well as diluting the labeled precursors. The defect should probably be considered to be a biochemical expression of "leukemicness". The data reported here imply that cytokinetic studies of erythroid cells in the myeloid leukemias using nucleoside tracers should, as far as possible, be checked with mitotic index determination. In cases of discrepancies between the data, stathmokinetic techniques and single cell DNA content determinations are necessary to obtain interpretable results.

1609 THE SPECIFICITY OF THE CHROMOSOMAL ABNORMALITIES IN HUMAN COLONIC POLYPS. A CYTOGENETIC STUDY OF MULTIPLE POLYPS IN A CASE OF GARDNER'S SYNDROME. *(E.)* Mark, J. (Dept. Med., U. Lund, Sweden), F. Mittelman, H. Dencker, C. Norryd and K.-G. Tranberg. *Acta Pathol Microbiol Scand (A)* 81(1):85-90, 1973.

The chromosomes in seven different polyps from a patient with Gardner's syndrome were studied. The patient was a 36-yr old female on whom a prophylactic colectomy was performed. The entire colon was obtained from the operating room within a few minutes after operation. Five small polyps, less than 4 mm in diameter, and five large ones, 1.4 - 1.8 cm in diameter, were subjected to chromosome analyses. Chromosome preparations of good quality were obtained from seven of the polyps. Histologically, all polyps showed areas with atypia; all of the tumors were non-invasive. Four polyps had a normal diploid stemline and showed a predominantly hyperdiploid chromosome variation. Three polyps had heteroploid stemlines: two were pseudo-diploid and one was hyperdiploid. The chromosomal difference between polyps of different size was merely quantitative. The same non-random karyotypic pattern was found in all polyps irrespective of size and histological grading.

1610 QUANTITATIVE ESTIMATION OF A PRIMATE-SPECIFIC ESTERASE IN NORMAL AND MALIGNANT TISSUE. *(E.)* Vladutiu, G. D. (Sch. Med., State U. New York, Buffalo) and N. R. Rose. *Proc Soc Exp Biol Med* 143(1):109-113, 1973.

The source of the primate-specific cathodal esterase, found in the urine and characterized as an aliesterase, is examined and preliminary evidence presented that quantitative differences exist in the esterase content of certain malignant tissues. Normal and malignant tissues from human males and females, ranging in age from 23 to 88 yr, were obtained at autopsy and tissue extracts prepared. The amount of cathodal esterase in the various tissues were determined by quantitative immunoelectrophoresis. Kidney had the highest relative concentration of 2.65 units/mg. This suggests that the esterase, as it exists in the urine in relatively large amounts, could be derived from the kidney. Kidney had twice the concentration of esterase found in the adrenals; liver closely followed adrenal with heart and brain occupying a middle position. Spleen, lung and pancreas had the lowest (0.36 units/mg). A comparison of the mean of four lung tumors with that of normal lung showed no significant difference; however, the mean esterase concentration of three liver tumors was more than twice that of normal liver. The significance of the variation in esterase concentration in different tissues has not been assessed. Esterase content is seen to be relatively low in normal lung; accordingly, there is no increase in esterase concentration in malignant tissues of the lung. On the other hand, liver has high levels of cathodal esterase and in hepatomas the esterase is greatly increased. It is postulated that in the change from normal to malignant growth, a cancerous cell may retain the unique isoenzyme pattern of the tissue of origin even though the quantity of the isoenzyme may vary.

1611 SUBUNITS OF HUMAN CHORIONIC GONADOTROPIN: UNBALANCED SYNTHESIS AND SECRETION BY CLONAL CELL STRAINS DERIVED FROM A BRONCHOGENIC CARCINOMA.

(E.) Tashjian, A. H., Jr. (Harvard Sch. Dental Med., Boston, Mass.), B. D. Weintraub, N. J. Barowsky, A. S. Rabson and S. W. Rosen. *Proc Nat Acad Sci USA* 70(5):1419-1422, 1973.

Three clonal strains of cells derived from a bronchogenic carcinoma previously shown to secrete biologically active human chorionic gonadotropin (hCG) both *in vivo* and *in vitro* were studied to examine whether isolated or unbalanced subunit synthesis observed *in vivo* might reflect metabolism of intact hCG molecules and to study the mechanisms underlying control and expression of ectopic hormone production. The intact hormone, hCG, and each of its subunits, hCG-α and hCG-β, was measured in culture medium and broken-cell preparations. hCG, and its alpha and beta subunits, were determined by three independent radioimmunoassay methods; a second antibody was used to separate bound from free-labeled hormone or subunit. The results show different rates of synthesis and secretion of hCG and its alpha and beta subunits by the three clones. For each clone, the amount of one or the other subunit always exceeded that of the complete hormone molecule. The results of these experiments show clearly the variability of control of the expression of differentiated function in malignant cells. Despite the differences in subunit production, each of these three clones contributed to the inappropriate production of complete hCG. It is suggested that the availability of clonal strains of such cells should permit the design of experiments to examine the mechanisms underlying the reappearance in adult neoplasms of the expression of previously stringently repressed genes.

1612 RETURN OF ALKALINE PHOSPHATASE IN CHRONIC MYELOCYTIC LEUKEMIA CELLS IN DIFFUSION CHAMBER CULTURES.

(E.) Chikkappa, G. (Brookhaven Natl. Lab., Upton, N.Y.), W. R. Boecker, G. Borner, A. L. Carsten, K. Conkling, L. Cook, E. P. Cronkite and S. Dunwoody. *Proc Soc Exp Biol Med* 143(1):212-217, 1973.

The growth pattern, cell survival kinetics, leukocyte alkaline phosphatase (LAP) activity changes, and karyotypes of cultured bone marrow cells from two human chronic myelocytic leukemia (CML) patients were studied. Cell numbers reached a peak three to five days after initial culturing and then declined exponentially with a possible plateau around day six. CML cells persisted and showed an initial proliferation of myeloblasts and promyelocytes followed by a rise in myelocytes and, later, in nondividing neutrophils. Histochemical assay of LAP activity showed an increase in the number of positive leukocytes which roughly coincided with the peak in the number of segmented neutrophils. Philadelphia chromosome was present in most cultures karyotyped up to 21 days after initial culturing.

1613 CATECHOLAMINES IN NEUROBLASTOMA CELLS FROM HUMAN BONE MARROW, TISSUE CULTURE, AND MURINE C-1300 TUMOR.

(E.) Helson, L. (Sloan-Kettering Inst. Cancer. Res. New York, N.Y.) and J. L. Biedler. *Cancer* 31(5):1087-1091, 1973.

The clinical use of the formaldehyde-induced fluorescence technique for visualizing catecholamine-containing cells in bone marrow smears of patients with metastatic neuroblastoma, and in neuroblastoma cells in culture derived from a metastatic site was examined. Duplicate smears were subjected to a conventional staining technique and to the formaldehyde-induced fluorescence technique. Cells fluorescing a bright apple green hue were observed in 6 of 9 bone marrow smears from patients with neuroblastoma. Cells with similar hues of equal fluorescent intensities were not detected in the marrow smears of six control patients. The fluorescing tumor cells occur singly, or in clumps. The intensity of fluorescence was equal to the maximum fluorescence obtained with human neuroblastoma cells grown in tissue culture and slightly less than that of murine C-1300 neuroblastoma prepared in the same manner. The percentage of fluorescent cells found in the bone marrow did not correlate with the degree of abnormal urinary excretion of catecholamines, vanilmandelic acid, or the percentage of abnormal cells detected by tetrachrome staining. This discrepancy could not explained on the basis of different cytologic characteristics. It is suggested, however, that the variations in storage and secretion of biogenic amines observed in human neuroblastoma tissue cultures during different growth phases may account for this discrepancy and that the neuroblastoma cells have little catecholamine-fluorescence during their rapid proliferative growth phase, and maximum fluorescence just after completion of this phase. It is concluded that the histofluorescence method could be used in patients with neuroblastoma, and to corroborate the diagnosis in those cases where the conventional stain technique or tissue culture methods fail to differentiate it from other round cell tumors.

1614 INFIDELITY OF DNA SYNTHESIS BY REVERSE TRANSCRIPTASE.

(E.) Springgate, C. F. (Inst. Cancer Res., Phila., Pa.), N. Battula and L. A. Loeb. *Biochem Biophys Res Comm* 52(2):401-406, 1973.

The fidelity of purified DNA polymerase (reverse transcriptase) from avian myeloblastosis virus (AMV) in precisely copying polynucleotide templates was determined. In experiments measuring the extent of polymerization of radiolabeled nucleotides, one molecule of the incorrect base-paired nucleotide (dCTP) was incorporated for every 6000 nucleotides polymerized from the poly(dA-dT) poly(dA-dT) template. The error rate for copying the ribo strand of poly(rA)·poly(dT) was about one in 600. The same reaction mixtures incubated with homogeneous *E. coli* DNA polymerase I made fewer than one mistake for every 100,000 nucleotides polymerized. Incorporation of incorrect nucleotide paralleled that of the correct nucleotide suggesting that the incorrect base was not added just terminally but was inserted as polymerization progressed. The level of infidelity observed was similar to that reported using nucleic-acid-free extracts from human acute leukemic lymphocytes suggesting that these cells may contain a polymerase similar to that found in AMV.

1615 COMPARISON OF NUCLEOLAR PROTEINS OF NORMAL RAT LIVER AND NOVIKOFF HEPATOMA ASCITES CELLS BY TWO-DIMENSIONAL POLYACRYLAMIDE GEL ELECTROPHORESIS. *(E.)* Orrick, L. R. (Baylor Coll. Med., Houston, Texas), M. O. J. Olson and H. Busch. *Proc Nat Acad Sci USA* 70(5):1316-1320, 1973.

A two-dimensional polyacrylamide gel electrophoresis technique for the separation of acid-extracted proteins of isolated nucleoli of normal rat liver and Novikoff hepatoma ascites cells was examined. Electrophoresis in the first dimension was performed with a gel composition of 10% acrylamide, 0.35% bisacrylamide, 0.1% ammonium persulfate, 1% N,N,N',N'-tetramethylenediamine (TEMED), and 4.5 M urea. A 20 to 50 µl sample was loaded on the gel and subjected to electrophoresis for 5 hr at 120 V and about 2.5 mA per gel tube. Half of the gel samples of the first dimension were then adapted for second dimension electrophoresis by dialysis in a series of solutions containing various concentrations of sodium dodecyl sulfate, phosphate buffer (pH 7.1), and 6M urea. Slab-gel electrophoresis in the second-dimension was performed with a gel composition of 12% acrylamide, 0.31% bisacrylamide, 0.05% TEMED, 0.10% sodium dodecyl sulfate, 0.075% persulfate, 6 M urea, and 0.1 M phosphate buffer (pH 7.1). Ninety-seven stained protein spots were found both in the patterns for Novikoff hepatoma samples and in samples of normal liver. The reproducibility of the relative intensities and positions of spots for samples of nucleolar proteins from individual preparations of Novikoff hepatoma and normal liver was considered excellent both for duplicates in a single run and among runs of separate series. Comparison of the patterns for normal liver and Novikoff hepatoma revealed nine spots with markedly different intensities in the two patterns. Two spots were found that were unique to the normal liver pattern, and one spot to the hepatoma pattern. The results indicate that protein fingerprinting methods may be of special value in distinguishing significant changes in protein complements of nuclear and nucleolar extracts of tissues in different functional states.

1616 THE REGULATION OF CELL GROWTH. I. IDENTIFICATION AND PARTIAL CHARACTERIZATION OF A DNA SYNTHESIS STIMULATING FACTOR FROM THE SERUM OF PARTIALLY HEPATECTOMIZED RATS. *(E.)* Morley, C. G. D. (Argonne Cancer Res. Hosp., Chicago, Ill.) and H. S. Kingdon. *Biochim Biophys Acta* 308(2):260-275, 1973.

The presence of at least one factor in the serum of partially hepatectomized rats which is capable of stimulating DNA synthesis in normal hepatocytes is demonstrated. Sprague-Dawley rats, six months old and weighing between 300 and 400 g, were used for partial hepatectomy and as a source of the serum factor. Three months old CF1 female mice, weighing 23-30 g, were used for the assay of the factor. The factors were assayed by *in vivo* injection of samples, followed by *in vitro* exposure to radioactive thymidine. The serum factor appears in the blood stream approximately 12 hr following partial hepatectomy and, when injected into mice, requires a few more hours to produce increased DNA synthesis. This delay might reflect a species difference because a rate factor was being assayed in mice. It was clear, however, that DNA synthesis was appreciable 12-24 hr after injection of the serum factor into mice, after a time interval during which DNA synthesis would not be appreciable following partial hepatectomy in mice. The factor is a heat stable protein with a molecular weight of approximately 26,000 and is liver specific. 400-fold purification of the protein was achieved using ion-exchange cellulose and Sephadex G-100 chromatography. There is no evidence to date that the rat serum factor needs a low-molecular-weight cofactor for activity, although the *in vivo* assays have not tested this possibility rigorously. It is suggested that other well-documented growth-promoting factors, such as the epidermal growth factor and the nerve growth factor, may have some relationship to the liver regenerating factor investigated in this study and that all are members of a family of growth promoting proteins which will continue to enlarge with further investigation.

1617 A TRANSFERABLE "RESISTANCE FACTOR" FROM *IN VITRO* CULTURED MDMS-RESISTANT YOSHIDA SARCOMA CELLS. *(E.)* Szende, B. (Paterson Labs., Manchester, England), M. Fox and B. W. Fox. *Br J Cancer* 27(3):245-252, 1973.

A transferable factor has been discovered in the medium of growing methylene dimethanesulphonate (MDMS)-resistant Yoshida sarcoma cells which is able to confer MDMS resistance to MDMS-sensitive Yoshida sarcoma cells. Resistance could be transferred by co-culturing resistant and sensitive cells for

24 hr or by growing sensitive cells in non-dialyzed medium from growing resistant cell cultures. Such newly resistant cells were derived from sensitive cells since they retained ability to form colonies in soft agar, a characteristic of sensitive but not of resistant lines. The resistance factor, which was of low molecular weight since it could pass through a dialysis bag, did not directly inactivate MDMS or its metabolites. It had no influence on sensitivity of cells if added after MDMS treatment.

1618 THE SELECTIVE CONCENTRATION OF SULPHADIAZINE AND RELATED COMPOUNDS IN MALIGNANT TISSUE. *(E.)* Abel, G. (Chester Beatty. Res. Inst., London, England), T. A. Connors, W. C. J. Ross, Nguyen-Hoang-Nam, H. Hoellinger and L. Pichat. *Eur J Cancer* 9(1):49-54, 1973.

The mechanism by which sulphadiazine is concentrated in tumor cells was studied in sarcoma 180, ADJ/PC6 plasma cell tumor and NK lymphoma bearing Balb/c female mice and in Walker carcinoma bearing female Wistar rats. Animals were injected i.p. with ^{14}C-labeled sulphadiazine or related compounds and at various times thereafter their concentrations in bone marrow or tumor homogenates were determined by chemical and liquid scintillation techniques. All tumors concentrated sulphadiazine to a greater extent than liver. The ability to concentrate sulphadiazine was dependent upon its chemical structure as related compounds such as sulphadiazine mustard and sulphathiazole were not selectively incorporated by tumor tissue. Three compounds closely related to sulphadiazine could also be selectively concentrated in tumor tissue. Sulphadiazine did not selectively concentrate in bone marrow. The fact that very low sulphonamide doses, which were soluble at low pH, were still selectively concentrated by tumors indicated that the drug does not concentrate because of precipitation in tumors with a low intracellular pH.

1619 A MATHEMATICAL EVALUATION OF TUMOUR GROWTH CURVES IN RAPID, INTERMEDIATE AND SLOW GROWING RAT HEPATOMATA. *(E.)* Looney, W. B. (Virginia Sch. Med. Charlottesville), A. A. Mayo, P. M. Allen, J. Y. Morrow and H. P. Morris. *Br J Cancer* 27(4):341-344, 1973.

Tumor growth rates of nine rat hepatoma lines which vary by a factor of ten have been mathematically evaluated. The degree of fit to several growth models of data obtained by vernier caliper measurement and weighing after sacrifice and excision of tumors was evaluated by regression analysis. Of all methods evaluated, one-half the product of the length x height x width (½ lhw) of the tumor was the function which gave the most meaningful biological results with respect to describing the relationship between tumor size and time after inoculation.

1620 CYCLIC AMP PHOSPHODIESTERASE IN CLONED ASTROCYTOMA CELLS: NOREPINEPHRINE INDUCES A SPECIFIC ENZYME FORM. *(E.)* Uzunov, P. (St. Elizabeth's Hosp., Washington, D.C.), H. M. Shein and B. Weiss. *Science* 180(4083):304-306, 1973.

The effect of norepinephrine on the two forms of cyclic AMP (adenosine 3,5'-monophosphate) phosphodiesterase in cloned C-2A rat astrocytoma cells was examined as part of a series of studies to determine the role of the sympathetic nervous system and other endogenous factors in controlling the intracellular concentration of cyclic nucleotides. The C-2A rat astrocytoma cell line was derived by cloning rat brain glial tumors induced by the i.v. injection of N-nitrosomethylurea. The soluble supernatant fraction of homogenates of these cloned cells was subjected to polyacrylamide gel electrophoresis. Two peaks of cyclic AMP phosphodiesterase activity were found, corresponding to peaks I and IV of a similarly prepared homogenate of rat brain. Incubating cells with norepinephrine (0.3 mM) caused about a 3-fold increase in the activity of peak IV but no change in peak I. This increase was completely inhibited by prior incubation with propranolol (0.1 mM), a β-adrenergic blocking agent, or with cyclohexamine (40 μM), a protein synthesis inhibitor. These results suggest that norepinephrine increased phosphodiesterase activity by inducing the synthesis of new protein. It is also suggested that the induction of a specific phosphodiesterase form by norepinephrine indicates a feedback control mechanism whereby an organism can prevent the effect of excessive sympathetic activity.

1621 THYMOMA AND HODGKIN'S DISEASE OF THE THYMUS. *(E.)* Nickels, J. (Ctr. Lab. Path., U. Helsinki, Finland), K. Franssila and L. Hjelt. *Acta Pathol Microbiol Scand (A)* 81(1):1-5, 1973.

The relationship between the granulomatous thymoma, the ordinary epithelial thymoma and Hodgkin's disease was examined. The material was collected from the files of the Finnish Cancer Registry (1953-1966) and the Department of Pathology, University of Helsinki (1957-1970). All specimens from cases primarily diagnosed as thymomas, either epithelial or granulomatous, were histologically re-examined and reclassified without knowledge of any clinical data. Only cases regarded at re-examination as epithelial (14) or granulomatous (8) thymomata were included in the study. The following variables were compared: age and sex, symptoms, operation status, metastases, prognosis, and histology. It was found that the age distribution, histological picture and clinical course of granulomatous thymoma were quite different from those of epithelial thymoma but similar to those of the nodular sclerosing type of Hodgkin's disease. It is concluded that granulomatous thymoma and epithelial thymoma are 2 distinct entities and should be distinguished; that granulomatous thymoma is apparently identical to the nodular sclerosing type of Hodgkin's disease; and that the term granulomatous thymoma should be changed to Hodgkin's disease of the thymus.

1622 LOSS OF HEPATOMA RIBOSOMAL RNA DURING WARFARIN THERAPY.

(E.) Van Buskirk, J. J. (U. Colorado Med. Ctr., Denver) and W. M. Kirsch. *Biochem Biophys Res Comm* 52(2):562-568, 1973.

The effect of warfarin treatment on the amount of cytoplasmic ribosomal RNA in BW7756 hepatoma implants and normal liver of C57L/J male mice was studied. Spectrophotometric measurements of purified RNA from hepatoma homogenates from mice receiving four to five daily warfarin injections showed a 50% decrease. Warfarin injections, however, had little or no effect on normal liver microsomal RNA from controls. Pulse labeling experiments using ^{14}C-orotate confirmed these results. Sedimentation analysis of ^{14}C-orotate labeled microsomal and nuclear rRNA from warfarin-treated hepatoma cells suggested that warfarin affected either the assembly of viable ribosomes or their survival in the cytoplasm.

1623 ARE SOME WOMEN'S BREAST CANCERS ANDROGEN DEPENDENT?

(E.) Flax, H. (Westminster Hosp. Med. Sch., London, England), H. Salih, K. A. Newton and J. R. Hobbs. *Lancet* 1(7814):1204-1207, 1973.

Biopsies of primary and metastatic breast tumors from 130 patients were tested *in vitro* for hormone dependence. Tumor cells from 14 women, 13 of which were post-menopausal, exhibited testosterone dependence (10^{-5} and 10^{-6} M). Six of these 14 patients had been treated at one time with either androgens or some form of anti-estrogenic therapy, and thus their tumors could have been preconditioned. Antiandrogen therapy was successful in six of eight patients, with adrenalectomy possibly being the most effective measure. Two patients whose *in vitro* tumor cells were not affected by estrogens failed to respond to estrogen treatment. In addition to the 14 tumors which showed a pure testosterone dependence, eight others were dependent on testosterone plus prolactin.

1624 MINUTE RING-SHAPED PARTICLES IN CULTURED CELLS OF MALIGNANT ORIGIN.

(E.) Shankar Narakan, K. (Pasadena Fdn. Med. Res., Calif.) and D. E. Rounds. *Nature* [*New Biol*] 243(126):146-150, 1973.

Ring-shaped particles have been detected in partially purified fractions of human adenocarcinoma (CMP), HeLa and KB cells, three lines of malignant origin. Electron microscopy of 105,000 g pellets revealed ring-shaped particles 10-12 nm in diameter and composed of up to seven subunits. Particle suspensions showed a peak absorption at A_{260}, suggesting the presence of nucleic acids. The particles incorporated ^{3}H-thymidine and 5-^{3}H-uridine, and sucrose density centrifugation of labeled culture media from each of the three cell lines showed peak radioactivity in the density range of 1.16-1.19 g/cm^3. The particles which were susceptible to DNase, RNase and pronase digestion and were detected only in culture media previously in contact with growing cells, were found, after cell fractionation, in predominant association with the polyribosomes. The extremely small size and the inability to demonstrate cell to cell transmission precluded a classification of these particles as viral.

1625 TRIPEPTIDE IN HUMAN SERUM WHICH PROLONGS SURVIVAL OF NORMAL LIVER CELLS AND STIMULATES GROWTH IN NEOPLASTIC LIVER.

(E.) Pickart, L. (Dept. Pediatrics, U. California, San Francisco) and M. M. Thaler. *Nature* [*New Biol*] 243(124):85-87, 1973.

A tripeptide growth factor which is biologically active in nanomolar concentrations has been purified from fresh human serum, plasma and albumin by ion exchange and thin-layer chromatography. The purified active factor, which had a molecular weight of approximately 300, was composed primarily of glycine, lysine and histidine. Addition of serum factor to "basal" (i.e., medium with 1% serum) Morris rat hepatoma cultures initially stimulated ^{3}H-labeled thymidine, uridine and leucine into acid insoluble material by 4-, 12-, and 4-fold, resp. Cell growth was accelerated after 48 hr incubation with the factor. Serum factor had no effect on acid-soluble intracellular pool radioactivity. In standard media, the factor was most effective in stimulating RNA synthesis at concentrations below 50 ng/ml. In contrast to results under "basal" conditions, serum factor had no appreciable stimulatory activity when conditions were optimal for cell growth (i.e., medium with 10% serum). In normal rat liver cultures, serum factor stimulated macromolecular synthesis, but not growth, and markedly prolonged cell culture life.

1626 IS MALIGNANT MELANOMA AN ENDOCRINE-DEPENDENT TUMOR? THE POSSIBLE ADVERSE EFFECT OF ESTROGEN.

(E.) Sadoff, L. (Kaiser Fdn. Hosp., Los Angeles, Calif.), J. Winkley and S. Tyson. *Oncology* 27(3):244-257, 1973.

Evidence is presented from case studies and statistical analyses of hospital records indicating the estrogen-dependence of malignant melanoma. Five cases are reported from the Kaiser Foundation Hospital, in which malignant melanoma occurred after the administration of an estrogen; an additional four cases reported from the literature describe the same relationship. The statistical analysis is based on 403 cases which were treated and followed at the Kaiser Foundation Hospital between 1951 and 1969. Of these patients, 211 were female and 192 were male. When the age of onset was analyzed from decade to decade, there was a striking disparity noted between the sexes. In the third decade (peak child-bearing period) and fifth decade (menopause), there was a marked female preponderance in incidence of melanoma. If malignant melanoma is an estrogen-dependent tumor, there are several implications: 1) therapeutic trials with antiestrogenic compounds appear warranted in melanoma; 2) patients with known malignant melanoma should be cautioned against the use of oral contraceptives, exogenous estrogens and pregnancy; 3) patients with breast or prostate cancer receiving estrogen therapy should be observed for primary melanoma. Suspicious lesions in these patients should be biopsied to differentiate between melanoma and metastases from the primary disease.

1627 THE EFFECT OF CHANGES IN ANTRAL pH ON THE BASAL RELEASE OF GASTRIN. *(E.)* Becker, H. D. (U. Texas Med. Branch, Galveston), D. D. Reeder and J. C. Thompson. *Proc Soc Exp Biol Med* 143(1):238-240, 1973.

The effect of antral pH change on basal release of gastrin was studied in adult mongrel dogs which had previously undergone laparotomy to expose the antral venous outflow. Perfusion of the antrum with physiologic saline at pH 7 to 8 resulted in more than a two-fold increase in the amount of gastrin secretion over basal levels at pH 3.5 as determined by radioimmunoassay. Gradual lowering of the antral pH to 1 decreased gastrin secretion to below basal pH 3.5 levels.

1628 STIMULATION OF EUKARYOTIC DNA-DEPENDENT RNA POLYMERASE BY PROTEIN FACTORS. *(E.)* Lee, S.-C. (Dept. Biochem., U. California, Davis) and M. E. Dahmus. *Proc Natl Acad Sci* 70(5):1383-1387, 1973.

Two protein factors which stimulate ascites cell DNA-dependent RNA polymerase activity *in vitro* have been purified from Novikoff ascites tumor cells. The factors, which were originally recovered in the DEAE-cellulose column flow-through fraction of the polymerase purification, were partially purified by $(NH_4)_2SO_4$ precipitation followed by CM-cellulose chromatography. Two peaks (HSF and HLF), which could stimulate ^{3}H-UTP incorporation into RNA in the *in vitro* assay, eluted from the CM-cellulose at 0.1 M and 0.3 M NH_4Cl, resp. HSF, the minor peak, was stable to heat treatment at 100 C for 10 min. HLF, which was heat labile, was further separated into two peaks (HLF_1 and HLF_2) eluting at 0.21 M and 0.42 M NH_4Cl, resp., from phosphocellulose columns. HLF_1 contained considerable nuclease activity and was not studied. Both HSF and HLF_2 were protease sensitive. Polyacrylamide gel electrophoresis of nondenatured material revealed several peaks for HSF but only one peak for HLF_2. The molecular weight of HLF_2 was estimated at 85,000. Both HSF and HLF_2 stimulated only ascites cell RNA polymerase and only when native ascites cell DNA was used as template. Addition of both HSF and HLF_2 to the same assay mixture produced more than an additive effect suggesting that these two factors act independently, probably at different steps in the polymerase reaction.

1629 MITOTIC AUTOREGULATION, GROWTH CONTROL AND NEOPLASIA. *(E.)* Wheldon, T. E. (Reg. Dept. Clin. Physics Bio-Engineering, Glasgow, Scotland), J. Kirk and W. M. Gray. *J Theor Biol* 38(3):627-639, 1973.

A model is proposed for the mitotic autoregulation of the hemopoietic stem cell population. Conditions are derived for the model population to exhibit spontaneous self-limitation; these consist of the satisfaction of algebraic inequality relationships between mathematical parameters referring to the global properties of the model cell population. Violation of these conditions precludes the possibility of spontaneous self-limitation of the population, which therefore grows indefinitely, but is not synonymous with abolition of all responsiveness to homeostatic regulation. Non-limiting growth modes exist for which "cybernetic" responsiveness to perturbations occur. It is suggested that certain features of the model accord well with some observed characteristics of neoplasia, such as the non-localized "field phenomena" frequently seen in tumor growth and the continued responsiveness of some tumors to hormones and other physiological signals. Radically new methods of cancer therapy should be made available by the elucidation of control mechanisms at the physiological level. It is concluded that even more complex mathematical analysis will be necessary, however, because the postulated cellular interactions are more complicated than may be readily unravelled by purely verbal reasoning.

1630 PLATELET--TUMOR-CELL INTERACTIONS IN MICE. THE ROLE OF PLATELETS IN THE SPREAD OF MALIGNANT DISEASE. *(E.)* Gasic, G. J. (U. Pennsylvania Sch. Med., Hematology Res., Philadelphia), T. B. Gasic, N. Galanti, T. Johnson and S. Murphy. *Int J Cancer* 11(3):704-718, 1973.

A series of studies was performed to investigate: 1) how tumor cells and platelets interact *in vitro* and *in vivo*; 2) whether agents which alter platelet function and behavior *in vivo* may also influence metastasis; and 3) the mechanism by which thrombocytopenia reduces tumor spread. Many tumors aggregated platelets *in vitro* and/or produced thrombocytopenia *in vivo*. In some instances, tumor-cell-induced thrombocytopenia *in vivo* was accompanied by accumulation of platelets in the lung. Thrombocytopenia was most active against metastases produced by tumors with the capacity to aggregate platelets *in vitro* and/or *in vivo* but it was also effective against metastases produced by tumors lacking such a capacity. Further studies, aimed at increasing platelet aggregation *in vivo*, as when fibroblasts were added to the tumor inoculum, or decreasing the platelet response to aggregating agents, as when aspirin was administered to mice, strongly support the role of platelet aggregation and the platelet release reaction in metastasis. As expected, fibroblasts enhanced while aspirin decreased tumor spread, the latter being equally effective against both artificially induced and spontaneously-occurring metastases. Formation of platelet aggregates not only enhances but also seems to change the distribution of metastases. Tumors with platelet aggregating capacities usually give lung metastases while those devoid of such a capacity may show metastases of wide-spread distribution. Research with ^{125}IUDR-labelled B16 melanoma cells indicates that thrombocytopenia does not affect the initial vascular arrest of tumor cells but seems to influence their subsequent retention by the lung. It is proposed that agents, such as aspirin, which inhibit platelet formation might favorably influence human malignancies, particulary during the early phases when the primary tumor is resectable and metastases have not been established.

1631 HISTOCHEMICAL STUDY OF NUCLEASE ACTIVITY IN CELLS OF TRANSPLANTABLE TUMORS AND THEIR METASTASES. *(E.)*

Vinarchuk, M. P. (Inst. Problems in Oncology, Acad. Sci. Ukrainian SSR, Kiev) and A. I. Bykorez. *Bull Exp Biol Med (USSR)* 74(11): 1423-1425, 1973.

The distribution and activity of acid and neutral DNases and of acid and alkaline RNases were studied by histochemical methods in tissue sections taken from growing primary and metastatic nodules of Walker's tumor and carcinoma PA (transplantable renal carcinoma) in rats. All four types of nuclease activity were found in the cytoplasm and nucleus of both tumor cell types. Although nuclease activity in the primary Walker and carcinoma PA tumors was low, the activity in both types of metastatic tumor tissues was even lower, despite similar intracellular distribution patterns. This finding suggests that the metastatic tumors are less differentiated than their primaries.

1632 HEPATIC TUMORS OF THE MASTOMYS. SPONTANEOUS AND EXPERIMENTAL PRODUCTION: A PRELIMINARY REPORT. *(E.)*

Kanahara, H. (Niigata U. Sch. Med., Japan), J. Soga and K. Tazawa. *Acta Med Biol (Niigata)* 20(2):91-96, 1972.

Three hundred and seventy-mastomys (wild African rodents) maintained untreated in laboratory cages for five yr were autopsied for microscopic and macroscopic evidence of spontaneous hepatic tumors. Fifty-one animals developed spontaneous hepatic tumors at an average age of 26 months. In addition, 11 of 28 animals treated with oral N-N'-2-7-fluorenylenebisacetamine (2,7-FAA) developed hepatic tumors at an average age of 22 months. The lesions consisted grossly of cysts, parenchymatous nodules or mixtures of these. Microscopically, the tumors could be classified as cystic, angiomatous, hepatocellular or miscellaneous (e.g., cholangioma, fibrosarcoma). None of the livers containing spontaneous or 2,7-FAA induced tumors showed cirrhotic changes and few showed bile duct proliferation as had been reported in an earlier study.

1633 ENHANCED MALIGNANT BEHAVIOUR OF CELLS TREATED WITH CRUDE RAT LIVER HISTONE. *(E.)*

Latner, A. L. (Royal Victoria Infirm., Newcastle upon Tyne, England), E. Longstaff and G. A. Turner. *Br J Cancer* 27(3):218-229, 1973.

Neonatal hamster kidney cells (BHK21/C13), challenged in monolayer culture for three days with crude rat liver histone, exhibited increased malignant characteristics when injected s.c. into syrian hamsters, 3 to 6 months old. In contrast to the controls, the challenged cells produced tumors which invaded either the epidermis or the body wall of their hosts and frequently caused extensive visceral metastases. *In vitro* studies of the cell cultures, during and after histone treatment, suggested that cellular "transformation" rather than selection was effected by the crude histone preparation. Cells from the primary tumors of both control and test groups appeared morphologically identical but after sub-culture *in vitro* they retained their respective growth characteristics on reinoculation.

1634 GONODOTROPIN PRODUCING HEPATOMA. *(E.)*

Floyd, W. S. (Wayne State U. Sch. Med., Detroit, Mich.) and S. L. Cohn. *Obstet Gynecol* 41(5): 665-668, 1973.

A case is presented in which a gonadotropin-producing hepatocellular carcinoma was detected in a 23-year-old white female during the immediate postpartum period. The patient's chief complaint when she first presented several hr before delivery was acute upper abdominal colicky pain radiating to the right subscapular region. The pain persisted postpartum along with slight fever. Histologic diagnosis of liver biopsy specimens taken at laparotomy was hepatocellular carcinoma. The patient was started on i.v. 5-fluorouracil before confirmation of the diagnosis. A chorionic gonadotropin titer measured by "Pregnosticon" was positive at 7500 IU. Alpha-fetoglobulin was negative and all hematologic studies were normal. Gonadotropin levels remained elevated (7500-75,000 IU) six wk later. Microscopically, the tumor consisted of irregular nests of cells with large, pale nuclei, irregular outline, prominent nucleoli, mitoses and focal areas of multinucleated giant cell formatin. Broad areas of necrosis and hemorrhage were evident. Although elevated gonadotropin titers had been reported in five cases of hepatocellular carcinoma in children, this was the first case reported in an adult.

1635 EVIDENCE OF MULTIPLE FORMS OF DNA POLYMERASE IN HODGKIN'S DISEASE. *(E.)*

Piperno, J. R. (U. Pennsylvania, Sch. Med., Philadelphia) and R. G. Kallen. *Cancer Res* 33(4):838-842, 1973.

Tissues with Hodgkin's disease involvement were examined to determine whether differences exist at the molecular level which might be detected and correlated with the four histologically differentiated forms of the disease. It was found that the DNA-dependent DNA polymerase activities from nodular sclerosis and mixed-cellularity forms of Hodgkin's disease can be distinguished from one another and from control human polymerases by their elution behavior upon diethylaminoethyl cellulose chromatography and Sephadex gel filtration, by the magnesium ion concentration for optimal activity, by the stimulatory or inhibitory effects upon enzymatic activity of monovalent cations, and by sensitivity of enzymatic activity to heparin inhibition. These studies at the molecular level support the mounting evidence of heterogeneity in Hodgkin's disease.

1636 THE NET EXTRUSION OF CALCIUM AND ITS TEMPORAL RELATION TO THE ACCUMULATION OF POTASSIUM IN SLICES OF RAT LIVER AND OF MORRIS HEPATOMATA 5123tc AND 3924A. *(E.)* Rossum, G. D. V. van (Temple U. Sch. Med., Philadelphia, Pa.), K. P. Smith and H. P. Morris. *Cancer Res* 33(5):1086-1091, 1973.

The ion-transporting activity of slices of liver and of Morris hepatomata 5123tc and 3924A was determined by studying the net movements of sodium, potassium, chloride, and calcium at 38 C after preincubation at 1 C. Liver slices showed an extrusion of 5 mmoles calcium per kg dry weight that was completed after 6 min, while potassium accumulation showed a lag period of about 15 min. Similar results were obtained from livers of rats of different strains and from livers of goose, opossum, and baboon. Slices of 5123tc extruded 16 mmoles calcium per kg dry weight during about 10 min, the initial rate being about 5 times that seen in liver; potassium accumulation was first observed after a delay of 6 min. Slices of 3924A extruded similar amounts of calcium at an initial rate similar to that of slices of 5123tc, but the final level of calcium remained higher; this is attributed to retention of calcium in micronecrotic areas of the slices of 3924A. This tumor also showed a lag period before potassium accumulation, although of only 3-min duration. The results show that the ability of respiring liver cells to extrude much of the excess calcium that enters the tissue during a period of metabolic inactivity is widespread among strains of rats and other vertebrate species and that it is an attribute of the parenchymal cells which persists in a quantitatively modified form in cells which have developed neoplastic properties.

1637 INFLUENCE OF HORMONES ON THE GROWTH OF HEPATOMAS AND INDUCTION OF ADENYLATE AND PYRUVATE KINASES. *(E.)* Criss, W. E. (U. Florida Coll. Med., Gainesville) and H. P. Morris. *Cancer Res* 33(5):1023-1027, 1973.

Tumor growth and the induction of adenylate kinase (EC 2.7.4.3) and pyruvate kinase (EC 2.7.1.40) in transplantable rat hepatomas were studied in organ-ablated and hormonally treated male, CFN strain rats that were fed different diets. Tumor growth was slowed when the rats were (a) fasted, (b) diabetic and fed chow, or (c) diabetic and fasted. Tumor growth increased in diabetic rats fed high-glucose diets or treated with insulin. Adenylate kinase, which was induced by fasting and/or hydrocortisone and which was repressed by glucose and/or insulin in normal rat liver, was found to be hyperresponsive to hydrocortisone and hyporesponsive to glucose and/or insulin in hepatomas. Pyruvate kinase, which was induced by glucose and/or insulin in normal rat liver, was not responsive to glucose and/or insulin in the liver tumors but was hyperresponsive to hydrocortisone in the hepatomas. If steroid binders are directly involved in the genetic expression of their target cells, modification within the chain of events leading from the binding of the hormone to the intracellular protein to the newly synthesized and active enzymes could be involved in altering the metabolic state of the cell such that the result might lead to a state of unchecked growth or cancer. The results from these studies illustrate a "commonness" in the modified responses of two enzymes in hepatomas.

1638 PARTIAL CHARACTERIZATION OF THE DNA-DEPENDENT DNA POLYMERASES OF RAT LIVER AND HEPATOMA. *(E.)* Hunter, G. R. (Jefferson Med. Coll., Philadelphia, Pa.), G. F. Kalf and H. P. Morris. *Cancer Res* 33(5):987-992, 1973.

Four DNA-dependent DNA polymerases, which include a soluble nuclear polymerase, the mitochondrial polymerase, and two polymerases associated with the smooth membrane fraction, have been partially purified from normal rat liver and a fast-growing Morris hepatoma, 7777, and compared. The membrane-associated enzymes have been designated as the 0.10 and 0.25 smooth membrane polymerases on the basis of their elution from a diethylaminoethyl cellulose column with the respective concentration of KCl. The polymerases were shown to be separate enzymes by their ability to use synthetic polymers as templates and their response to several inhibitors. The mitochondrial DNA polymerase was readily distinguished from the smooth membrane polymerases by its ability to utilize poly(dA)d$(pT)_{10}$ efficiently as template and by its complete inhibition by concentrations of ethidium bromide which do not inhibit the other enzymes. The mitochondrial enzyme is the only one of the four polymerases which is insensitive to inhibition by *p*-hydroxymercuribenzoate. The various polymerases were also distinguished by the effects of monovalent salts and the alkaloid antibiotic, camptothecin, on their activities. NaCl (0.05 to 0.10 M) stimulates the mitochondrial and 0.10 smooth membrane polymerases but is inhibitory to the nuclear and 0.25 smooth membrane enzymes. Camptothecin inhibits the nuclear and 0.25 smooth membrane polymerases, stimulates the 0.10 smooth membrane enzyme, and has no effect on the mitochondrial polymerase. On the basis of these data, neither of the smooth membrane polymerases appears to represent the mitochondrial DNA polymerase in the process of synthesis on cytoribosomes for transit into the organelle while attached to smooth membrane vesicles. It is not known whether any of these DNA polymerases carries out replicative or repair synthesis.

1639 CHROMATOGRAPHIC STUDIES OF TYROSYL AND PHENYLALANYL TRANSFER RNA'S OF LIVER AND TUMOR CELLS. *(E.)* Hayashi, M. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), A. C. Griffin, R.

Duff and F. Rapp. *Cancer Res* 33(4):902-905, 1973.

Elution profiles of tyrosyl and phenylalanyl transfer RNA's from several normal liver and cancerous tissues were studied by means of the newly developed reverse-phase chromatography system, RP-5. Tyrosyl transfer RNA from normal rat, mouse, and hamster livers emerged as a single peak. All three species exhibited identical profiles. Tyrosyl transfer RNA from *Escherichia coli* required a considerably higher NaCl concentration for elution from the RP-5 column. Tyrosyl transfer RNA from the Novikoff ascites hepatoma emerged as two separate peaks--one identical with the normal liver and a second late-eluting peak. Tyrosyl transfer RNA from SV40-induced tumors in hamsters and also from Ehrlich ascites tumor cells exhibited a broad-based peak which eluted later than the normal and which probably consists of two or more tyrosyl transfer RNA's. The phenylalanyl transfer RNA elution profile from normal mouse liver consists of two smaller peak areas, followed by a major third peak. The transfer RNA from Ehrlich tumor cells followed a similar pattern; however, the middle peak was larger than normal. In contrast, Ehrlich tumor cells with an acquired resistance to nitrogen mustard exhibited a major middle peak, while the first and third peak areas were greatly diminished. The findings with the use of the RP-5 system indicate that it is a rapid, sensitive, and highly reproducible procedure for studying the patterns of isoaccepting transfer RNA species. There is indication that tumors differ from normal counterparts in several of the transfer RNA elution profiles.

1640 FACTORS IN HUMAN SERUM AFFECTING THE PROLIFERATION OF NORMAL AND LEUKEMIC CELLS.

(E.) Burke, P. J. (Johns Hopkins U. Sch. Med., Baltimore, Md.), C. H. Diggs and A. H. Owens, Jr. *Cancer Res* 33(4):800-806, 1973.

Serum factor(s) regulate the rate of proliferation of normal and leukemic bone marrow cells. Nine patients with solid tumors and normal marrow function were treated with cyclophosphamide (60.0 mg/kg) on each of 2 successive days. Following aplasia, the granulocytic proliferative fraction of the patients' marrow increased twofold (tritiated thymidine autoradiography). With maturation of the marrow elements, the tritiated thymidine indices fell below pretreatment values. Serial serum samples obtained from these patients affected the incorporation of tritiated thymidine into DNA in primary cultures of normal bone marrows. In relation to pretreatment sera, samples obtained during the proliferative phase caused a twofold increase in tritiated thymidine incorporation by target cells, and sera obtained during the marrow maturation phase caused a twofold decrease. Identical results were obtained when these sera were incubated with marrows from patients with acute lymphoblastic and acute myelocytic leukemia (>95% tumor) and when they were assayed with the long-term cultured cell lines. Thus, factor(s) are present in these sera that can affect tritiated thymidine uptake in both normal and leukemic hematopoietic cells. The precise identity and source(s) of these factors are unknown. The observations indicate that the serum factor(s) that affect tritiated thymidine uptake in the assay systems used are nonspecific with respect to the type of cell affected or the species of origin of the cell. Multiple factors may be present or there may be one factor which is universal in its activity. The effect of these factor(s) on leukemic cells suggests that humoral factors may be involved in mediating the proliferative kinetics of hematopoietic neoplasia.

1641 COMPLEX MITOCHONDRIAL DNA IN HUMAN TUMORS.

(E.) Smith, C. A. (Norman W. Church Lab. Chem. Biol., California Inst. Technology, Pasadena) and J. Vinograd. *Cancer Res* 33(5):1065-1070, 1973.

The frequency with which complex mitochondrial DNA forms occur in DNA extracted from a number of human solid tumors was examined. The catenated forms in all but one sample were within the ranges of frequency reported previously in animal tissue. One sample contained 14% catenated dimers and 10% catenated higher oligomers. Circular dimers were present in 12 of 15 samples, ranging in frequency from 0.2 to 9%. No circular dimers were seen in a sample of human placenta. The results extend the previous correlation between human neoplasia and the presence of the circular dimer form of mitochondrial DNA in many neoplastic cells. Detailed interpretation of the variation of frequencies of the form, as well as the meaning of the fact that 3 tumor samples were found to be free of circular dimers, is made difficult by several factors such as the amount of normal tissue in the sample, tumor physiology, degree of malignancy, and type of neoplasia.

1642 CHARACTERIZATION OF GLYCOGEN SYNTHETASES AND PHOSPHORYLASES IN TRANSPLANTABLE RAT HEPATOMAS.

(E.) Sato, K. (Temple U. Sch. Med., Philadelphia, Pa.), H. P. Morris and S. Weinhouse. *Cancer Res* 33(4):724-733, 1973.

A study of the chemical and kinetic properties of a series of transplantable, chemically induced rat hepatomas has revealed profound alterations in isozyme patterns of glycogen synthetase and phosphorylase. Normal male rats of a pathogen-free (CFN) strain were used when they were approximately 6 months old and weighed 200 to 300 g. All experiments were conducted at uniform times to minimize the effects of diurnal variations on glycogen and enzyme levels. Highly differentiated, slow-growing hepatomas retain the liver-type isozymes, but with decreased differentiation and increased growth rate these are largely or entirely replaced by nonhepatic isozymes. The phosphorylase of poorly differentiated hepatomas was a third form, shown by isoelectric focusing and by immunochemical criteria to be different from either the liver or muscle isozymes. This form was also present in 21-day fetal liver, together with the adult liver isozyme. All of the hepatomas contained the phosphatases and kinases that interconvert the *a* and *b* forms, and preliminary data indicate that the synthetase phosphatase of the poorly differentiated hepatomas also is different from the liver phosphatase. Total synthetase activity was high in liver and muscle, lower in the well- to highly differentiated hepatomas, and very low in poorly differentiated tumors, except for the Novikoff ascites hepatoma, the only one of the poorly differentiated tumors that stored appreciable glycogen. In all tissues, *b* form predominated. Total phosphorylase activity was highest in muscle, predominantly in the *b* form; lower in liver; and still lower in tumors. There was no correlation between the activity of tumor phosphorylase and the degree of differentiation or the storage of glycogen, and most of the liver and hepatoma phosphorylase was in the *a* form.

1643 MICROFLUOROMETRIC COMPARISONS OF CHROMATIN THERMAL STABILITY *IN SITU* BETWEEN NORMAL AND NEOPLASTIC CELLS.

(E.) Alvarez, M. R. (Dept. Biol., U. South Florida, Tampa). *Cancer Res* 33(4): 786-790, 1973.

Microfluorometric comparisons of chromatin thermal stability *in situ* were made among nuclei isolated from various types of normal and neoplastic cells of the mouse in an attempt to distinguish tumor cells objectively on the basis of their acridine orange staining properties after thermal denaturation of chromatin. Normal tissue cell nuclei were isolated from liver, kidney, and spleen. Neoplastic cell nuclei were derived from transplanted murine lymphoma, sarcoma, mammary adenocarcinoma, and Ehrlich ascites tumor. The isolated nuclei were seeded on glass slides in equal densities, fixed, and heated to various temperatures in 0.15 M NaCl-0.015 M sodium citrate containing formaldehyde in order to induce denaturation of the chromatin. The nuclei were subsequently stained with acridine orange. Chromatin thermal stability was compared by measuring the flourescence emission ratios of the chromatin-ligand complex at 530 and 590 nm for each temperature. The flourescence emission profiles obtained were characteristic of each tissue type. However, the curves from all normal tissue cells showed a *gradual* shift in the emission ratio with temperature while those of the neoplastic cells, in general, showed a *rapid* shift in the fluorescence emission ratio and achieved a consistently higher maximum ratio. The data indicate that the chromatin of these neoplastic cells is more thermolabile than that of normal tissue cells and suggest the possible application of the method to cytodiagnosis.

1644 DIFFERENTIAL RELEASE OF SIALIC ACID FROM NORMAL AND MALIGNANT CELLS BY *VIBRIO CHOLERAE* NEURAMINIDASE OR INFLUENZA VIRUS NEURAMINIDASE.

(E.) Ray, P. K. (Dept. Surg., U. Minnesota, Minneapolis) and R. L. Simmons. *Cancer Res* 33(5): 936-939, 1973.

A comparison was made between the amounts of sialic acid released from viable cell suspensions of normal and malignant cells by *Vibrio cholerae* neuraminidase (VCN) and by influenza virus neuraminidase. More sialic acid can be removed by enzyme digestion from various tumors than from normal lymphoid cells. More sialic acid can be removed from the surface of both normal and tumor cells by VCN than by influenza virus neuraminidase. VCN lyses the 2-3, 2-6, and 2-8 glycosidic linkages between sialic acid and the mucopolysaccharides of the cell surface, and influenza virus neuraminidase lyses only the 2-3 and 2-8 linkages. Therefore, it appears that most cell-surface sialic acid residues are bound by the 2-6 linkage on both normal and malignant cells. The relative increase in cell-surface sialic acid residues may be important to tumor immunotherapy, since the removal of cell-surface sialic acid residues with VCN increases the immunogenicity of tumor cells.

1645 CHANGES IN THE SURFACE COATS OF NEOPLASTIC HUMAN BREAST EPITHELIUM.

(E.) Dermer, G. B. (Hosp. Good Samaritan Med Ctr., Los Angeles, Ca.). *Cancer Res* 33(5):999-1002, 1973.

Phosphotungstic acid at low pH was used as a section stain to make visible the surface coats of benign (5) and malignant (10) epithelial cells from human breast biopsies in the electron microscope. For this purpose, tissue is fixed in glutaraldehyde and embedded in the water-soluble plastic glycol methacrylate. Duct epithelial cells from all the biopsies containing benign tissue showed consistently more phosphotungstic acid-positive material at their surfaces than did the malignant cells. These results from an *in vivo* system are consistent with the observations of others, who have shown that there are lower amounts of sugars at the surfaces of transformed cells in culture. Thus, alterations of surface polymers, such as glycoproteins, may be important in the development and maintenance of the altered growth properties of malignant breast epithelial cells *in vivo*.

1646 RAPID UPTAKE OF DIETARY CHOLESTEROL BY HYPERPLASTIC LIVER NODULES AND PRIMARY HEPATOMAS.

(E.) Horton, B. J. (McArdle Lab. Cancer Res., U. Wisconsin, Madison), G. E. Mott, H. C. Pitot and S. Goldfarb. *Cancer Res* 33(3):460-464, 1973.

The rate of cholesterol-^{3}H uptake in hyperplastic liver nodules and into primary hepatomas induced by 2-acetylaminofluorene in male, Sprague-Dawley CFE rats, was measured. It was found that the hyperplastic liver nodules and hepatomas take up cholesterol-^{3}H, given p.o., at a rate only slightly less than that in normal liver, and 10- to 30-fold greater than that reported for transplantable hepatomas. In the case of primary hepatomas, the rate of uptake appeared to be related to the degree of differentiation and hence a function of tumor progression. In view of the findings that primary hepatomas do not show a decrease in cholesterol uptake similar to that in transplantable hepatomas, 3 possibilities are considered: 1) the defect in cholesterol uptake in transplantable hepatomas may not be a consequence of the malignant change at all; 2) since primary, well-differentiated hepatomas do accumulate dietary cholesterol, inhibition of cholesterol synthesis in response to cholesterol feeding may occur in a fashion similar to normal liver; and 3) if well-differentiated primary hepatomas do in fact lack inhibition of cholesterol synthesis, it is likely to be the result of an intracellular defect rather than a defect of cell membrane transport.

1647 STUDIES ON TRYPTOPHAN METABOLISM IN PATIENTS WITH BLADDER CANCER. *(E.)* Gailani, S. (Roswell Park Mem. Inst., Buffalo, N.Y.), G. Murphy, G. Kenny, A. Nussbaum and P. Silvernail. *Cancer Res* 33(5):1071-1077, 1973.

An attempt was made to correlate hepatic tryptophan pyrrolase and kynureninase activity in 36 patients with bladder cancer with the urinary excretion of some of the tryptophan metabolites after 2 g L-tryptophan p.o. Assay of hepatic tryptophan pyrrolase activity was done on 21 patients, and assay of kynureninase was done on all patients. Of these patients, increased excretion of kynurenine was found in 12, 3-hydroxykynurenine was found in 25, kynurenic acid was found in 9, xanthurenic acid was found in 4, and acetylkynurenine was found in 10. There was no correlation between tryptophan pyrrolase activity and the level of the urinary excretion of tryptophan metabolites, while patients with low kynureninase activity tended to excrete increased quantities of these metabolites. Depressed kynureninase activity and increased excretion of tryptophan metabolites were more marked in the more advanced Stage D_1 and D_2 bladder cancer. Measurements of excretion of the tryptophan metabolites 6 to 12 months after eradication of the disease in 19 patients showed a statistically significant decrease in the excretion of kynurenine, while increased excretion of 3-hydroxykynurenine persisted in 9 of the patients. The excretion of the other metabolites was within normal range in all the patients postoperatively. Similar measurements on two other patients with known evidence of metastases showed an increase in the excretion of the various metabolites in the postoperative compared to the preoperative measurements. Kynureninase may play a significant, although not the sole, role in the control of the level of various metabolites of the tryptophan-niacin pathway. Low activity of kynureninase also was observed in patients with other neoplasms.

1648 *IN VIVO* EFFECTS OF PROTEASES ON CELL SURFACE ARCHITECTURE AND CELL PROLIFERATION IN THE LIVER. *(E.)* Yamamoto, K. (Zoological Inst., U. Tokyo, Japan), S. Omata, T. Ohnishi and H. Terayama. *Cancer Res* 33(3):567-572, 1973.

Effects of purified proteases such as trypsin, papain, pronase E, and collagenase injected i.p. in male Wistar rats, weighing 115 to 130 g, and male ICR mice, weighing 30 g, on the cell surface architecture, as well as on the DNA synthesis and mitosis in the liver of the rats and mice, were investigated biochemically and histologically. Among the proteases examined, only papain induced the stimulation of DNA synthesis and mitosis, as well as the alteration in the pericellular constitution. Histochemical staining showed that the pericellular acidic material in the liver, probably the cell coat acid mucopolysaccharide, disappears temporarily prior to the stimulated DNA synthesis and mitosis in papain-treated animals. Glucosamine-^{3}H incorporations into the plasma membranes, as well as other subcellular fractions of the liver, were markedly reduced soon after papain injection, reaching a minimum at 12 hr but recovering to the normal level at 36 hr. On the other hand, the rate of removal of previously incorporated glucosamine-^{3}H was increased soon after papain injection in the cytomembranes (microsomes plus plasma membrane fragments) but not in the cytosol. The *in vivo* effects of proteases on the DNA synthesis, as well as the cellular proliferation in the liver, may not be correlated with the acute toxicities of these proteases because all the rats that received 3 mg of papain or trypsin survived beyond 2 days, while 1/3 to 2/3 of rats that received similar doses of Pronase E or collagenase died within several hr after injection. It is suggested rather that the specific effect of papain *in vivo* might be due to the greater permeability of the enzyme through the liver capsule.

1649 INHIBITION OF RIBONUCLEOTIDE REDUCTASE FROM EHRLICH TUMOR CELLS BY RNA. *(E.)* Cory, J. G. (Dept. Med. Microbiol., Chem., U. South Florida, Tampa). *Cancer Res* 33(5):993-998, 1973.

Ribonucleotide reductase activity from Ehrlich ascites cells taken from mice seven days after transplantation was inhibited by RNA. Inhibition of cytidine 5'-diphosphate reduction was observed with Ehrlich tumor cell transfer RNA, ribosomal RNA, yeast transfer RNA, and synthetic polycytidylate. Polyguanylate and polyuridylate were only slightly effective as inhibitors, while polyadenylate did not inhibit at all. The RNA's and polycytidylate appeared to be competitive inhibitors with respect to cytidine 5'-diphosphate as substrate. Adenosine 5'-diphosphate reduction was inhibited by transfer RNA but was only slightly inhibited by polyadenylate.

1650 DISTRIBUTION OF RIBOSOMAL CISTRONS IN THE NUCLEI OF EHRLICH ASCITES TUMOR AND HEPATIC CELLS FROM MICE. *(E.)* Higashi, K. (Osaka U. Med. Sch., Japan), T. Kuragano, N. Hanasaki, T. Matsuhisa and Y. Sakamoto. *Cancer Res* 33(4):734-738, 1973.

The distributions of ribosomal cistrons in subfractions of nuclei from Ehrlich ascites tumor and mouse liver cells were compared. The mean number of nucleoli per nucleus in mouse liver (diploid cells) is 3.7 while, in hyperdiploid and hypotetraploid strains of Ehrlich ascites tumors, the means are 1.8 and 3.6, resp. However, in spite of the relatively few nucleoli per nucleus in tumor cells, the amount of DNA in the nucleoli is increased significantly (three- to fourfold). The ratio of annealing of radioactive ribosomal RNA with nucleolar DNA was similar in tumor cells and mouse liver cells, so the total amount of ribosomal genes in individual nucleoli of tumor cells must be greater than in mouse liver cells, and this increase may be enough to compensate for the reduction of nucleolar number in the nuclei of tumor cells. It is probable that fusion of nucleolar organizers (rather than amplification of ribosomal genes) occurs in the nucleoli of tumor cells, because no significant difference was found in the numbers of ribosomal cistrons in whole nuclear DNA of tumor and normal liver cells. It is uncertain whether this fusion is indispensable for the change in gene expression for rRNA or whether it is required only for the stimulation of biosynthesis of rRNA. Several ribosomal genes in extranucleolar regions are not expressed *in vivo* but were detected by hybridization *in vitro*.

1651 URINARY AND PLASMA ANDROGENS IN BENIGN BREAST DISEASE. POSSIBLE RELATION TO BREAST CANCER. *(E.)* Brennan, M. J. (Michigan Cancer Fdn., Detroit), R. D. Bulbrook, N. Deshpande, D. Y. Wang and J. L. Hayward. *Lancet* (7812):1076-1080, 1973.

Concentrations of plasma total 11-deoxy-17-ketosteroids and dehydroepiandrosterone sulphate were measured in normal healthy women and in women with benign breast disease. The mean plasma concentrations of these steroids were significantly subnormal in woman aged 20-40 years with benign breast disease. Older women with benign breast disease had mean steroid levels similar to those in normal women. The same trend was found for urinary 11-deoxy-17-ketosteroids. These findings may be relevant to the increased risk of subsequent breast cancer in women with benign breast disease. Such a concept might resolve the arguments about the risk of breast cancer in women with benign breast disease, since little or no increase in the incidence of breast cancer might be expected if the majority of women studied were postmenopausal. The only significant abnormalities were found in relatively young women and in this group only may changes in breast structure be a true disease process.

1652 RENAL CARCINOMA IN A LIVING KIDNEY GRAFT DONOR. *(E.)* Fox, M. (Royal Hosp., Sheffield, England). *Transplantation* 15(5):523-525, 1973.

A renal carcinoma was discovered in the kidney of a 62-year-old male renal transplant donor at the time of transplantation. The tumor appeared grossly as a ½ x 1 cm raised, firm, white nodule which, on frozen section, was diagnosed as invasive adenocarcinoma. The kidney was removed for therapeutic purposes. This case history emphasizes the importance of adequate examination of donor kidneys, with biopsy of any suspicious areas prior to transplantation.

1653 LYMPHOCYTE LYSOSOMES AND LYSOSOMAL ENZYMES IN CHRONIC LYMPHOCYTIC LEUKEMIA. *(E.)* Douglas, S. D. (Mt. Sinai Sch. Med., New York, N. Y.), G. Cohnen, E. Konig and G. Brittinger. *Blood* 41(4):511-518, 1973.

The activities of the lysosomal enzymes, acid phosphatase and β-glucuronidase, and the non-lysosomal enzyme, malate dehydrogenase, were studied histochemically and biochemically in thin sections and lysates, resp., of lymphocytes from four healthy and five chronic lymphatic leukemia (CLL) patients. Compared with normal lymphocytes, CLL lymphocytes showed decreased activity of both lysosomal enzymes, but not of the non-lysosomal enzyme. Determination of the numbers of membrane-limited acid-phosphatase positive organelles by electron microscopy revealed an average of only 0.17 organelles in CLL cells compared with 1.07 per normal cell. These observations suggest that the diminished lysosomal enzyme activity of CLL cells is due to a reduced number of lysosomes.

1654 STIMULATION OF DNA SYNTHESIS BY ADENOSINE DIPHOSPHORIBOSYLATION OF HELA NUCLEAR PROTEINS DURING THE CELL CYCLE. *(E.)* Roberts, J. H. (Sch. Med., Georgetown U., Washington, D.C.), P. Stark and M. Smulson. *Biochem Biophys Res Commun* 52(1):43-50, 1973.

1655 BLACK-PIGMENTED ADENOMA OF THE ADRENAL GLAND. REPORT OF THREE CASES INCLUDING ELECTRON MICROSCOPIC STUDY. *(E.)* Garret, R. (Roosevelt Hosp., New York, N.Y.) and R. P. Ames. *Arch Pathol* 95(5):349-353, 1973.

1656 HUMAN TUMOR MICROSPORIDIOSIS. FIRST REPORTED CASE. *(E.)* Marcus, P. B. (U. Stellenbosch Med. Sch., Tiervlei, Cape, South Africa), J. J. van der Walt and P. J. Burger. *Arch Pathol* 95(5):341-343, 1973.

1657 INVASION OF THE FACIAL BONES BY BASAL MENINGIOMAS. *(E.)* Kendall, B. (Natl. Hosp. Nervous Dis., London, England). *Br J Radiol* 46(544):237-244, 1973.

1658 DEMONSTRATION OF PROGENITOR CRYPTOCIDES IN THE BLOOD OF PATIENTS WITH COLLAGEN AND NEOPLASTIC DISEASES. *(E.)* Livingston, V. W.-C. (U. San Diego, Dept. Biol., Calif.) and A. M. Livingston. *Trans NY Acad Sci* 34(5):433-453, 1972.

1659 BASAL CELL CARCINOMA WITH PULMONARY METASTASIS: A CASE ASSOCIATED WITH RETICULUM CELL SARCOMA AND THYROID CARCINOMA. *(E.)* Liu, P. I. (Atomic Bomb Casualty Commission, Hiroshima, Nagasaki, Japan), D. H. McGregor, R. W. Cihak, Y. It and T. Ishimaru. *Hiroshima J Med Sci* 20(4):281-289, 1971.

1660 EFFECT OF DIETARY CARBOHYDRATE ON FATTY ACIDS IN THE R3230AC MAMMARY ADENOCARCINOMA. *(E.)* Zalenski, D. (U. Rochester Sch. Med., Dent., N.Y.) and R. Hilf. *Proc Soc Exp Biol Med* 142(4):1137-1140, 1973.

1661 OXYGEN CONSUMPTION OF HUMAN LEUKOCYTES FROM BLOOD, LYMPH NODES, AND PATIENTS

WITH LEUKEMIA. *(E.)* Evans, A. E. (Children's Hosp. Philadelphia, Pa.) and J. Blore. *Proc Soc Exp Biol Med* 142(4):1118-1122, 1973.

1662 EXTRASKELETAL MESENCHYMAL CHONDROSARCOMA. *(E.)* Guccion, J. G. (VA Hosp., Washington, D.C.), R. L. Font, F. M. Enzinger and L. E. Zimmerman. *Arch Pathol* 95(5):336-340, 1973.

1663 THE MISSING Y CHROMOSOME AND HUMAN LEUKAEMIA. *(E.)* Pierre, R. V. (Mayo Clin., Mayo Fdn., Rochester, Minn.) and H. C. Hoagland. *Lancet* (7810):1008-1009, 1973.

1664 ISOLATION OF PLASMA MEMBRANES FROM EHRLICH ASCITES TUMOR CELLS. INFLUENCE OF AMINO ACIDS ON (Na$^+$ + K$^+$)-ATPase AND K$^+$-STIMULATED PHOSPHATASE. *(E.)* Forte, J. G. (Dept. Phys.-Anat., U. California, Berkeley), T. M. Forte and E. Heinz. *Biochim Biophys Acta* 298(4):827-841, 1973.

1665 ADENOSINE 3',5'-MONOPHOSPHATE IN GLIAL TUMOR CELLS. ALTERATIONS BY 5-BROMODEOXYURIDINE. *(E.)* Schwartz, J. P. (Rutgers Med. Sch., Piscataway, N.J.), N. R. Morris and B. McL. Breckenridge. *J Biol Chem* 248(8):2699-2704, 1973.

1666 STIMULATION OF STEROID SECRETION BY ANTIMICROTUBULAR AGENTS. *(E.)* Temple, R. (Natl. Inst. Hlth., Bethesda, Md.) and J. Wolff. *J Biol Chem* 248(8):2691-2698, 1973.

1667 THE UTILIZATION OF SYNTHETIC RIBONUCLEIC ACID TEMPLATES BY A NEW DEOXYRIBONUCLEIC ACID POLYMERASE FROM CULTURED MURINE CELLS. *(E.)* Fry, M. (Roche Inst. Molecular Biol., Nutley, N.J.) and A. Weissbach. *J Biol Chem* 248(8):2678-2683, 1973.

1668 CHANGES IN THE BINDING OF CONCANAVALIN A AND WHEAT GERM AGGLUTININ TO HUMAN LYMPHOCYTES DURING *IN VITRO* TRANSFORMATION. *(E.)* Krug, U. (Johns Hopkins U. Sch. Med., Baltimore, Md.), M. D. Hollenberg and P. Cuatrecasas. *Biochem Biophys Res Commun* 52(1):305-312, 1973.

1669 DIFFERENCES IN THE TRANSFER RNA METHYLTRANSFERASES FROM NORMAL RAT LIVER AND NOVIKOFF HEPATOMA. *(E.)* Sharma, O. K. (U. Colorado Med. Ctr., Denver). *Biochim Biophys Acta* 299(3): 415-427, 1973.

1670 IMMUNOASSAYABLE INSULIN IN CARCINOMA OF THE CERVIX ASSOCIATED WITH HYPOGLYCEMIA. *(E.)* Kiang, D. T. (U. Minnesota Sch. Med., Minneapolis), G. E. Bauer and B. J. Kennedy. *Cancer* 31(4): 801-805, 1973.

1671 METASTATIC MAMMARY CARCINOMA IN THE EYELID WITH HISTIOCYTOID APPEARANCE. *(E.)* Hood, C. I. (Armed Forces Inst. Path., Washington, D.C.), R. L. Font and L. E. Zimmerman. *Cancer* 31(4):793-800, 1973.

1672 MALIGNANT MELANOMA AND THE BREAST. *(E.)* Pressman, P. I. (Mount Sinai Sch. Med., New York, N.Y.). *Cancer* 31(4):784-788, 1973.

1673 CERVICAL LYMPH NODE METASTASIS: UNKNOWN PRIMARY CANCER. *(E.)* Jesse, R. H. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.), C. A. Perez and G. H. Fletcher. *Cancer* 31(4):854-859, 1973.

1674 MEDULLARY CARCINOMA OF THE THYROID GLAND. A CLINICOPATHOLOGIC STUDY OF 40 CASES. *(E.)* Gordon, P. R. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), A. G. Huvos and E. W. Strong. *Cancer* 31(4):915-924, 1973.

1675 HODGKIN'S DISEASE IN THE FIRST DECADE. *(E.)* Chaves, E. (Cancer Hosp. Paraiba, Brazil). *Cancer* 31(4):925-930, 1973.

1676 STUDIES ON MURAMIDASE IN HEMATOLOGICAL DISORDERS. I. SERUM MURAMIDASE AND SERUM LACTIC DEHYDROGENASE IN LEUKEMIA. *(E.)* Levi, J. A. (Sydney Hosp., Australia), J. B. Speden, P. C. Vincent and F. W. Gunz. *Cancer* 31(4):939-947, 1973.

1677 CHRONIC GRANULOCYTIC LEUKEMIA ASSOCIATED WITH CENTRAL HYPOVENTILATION. *(E.)* Perlin, E. (Natl. Naval Med. Ctr., Bethesda, Md.), R. F. Granatir and R. B. Moquin. *Cancer* 31(4):956-958, 1973.

1678 TWO-DIMENSIONAL POLYACRYLAMIDE GEL ELECTROPHORESIS OF ACID EXTRACTABLE NUCLEAR PROTEINS OF NORMAL RAT LIVER AND NOVIKOFF HEPATOMA ASCITES CELLS. *(E.)* Yeoman, L. C. (Baylor Coll. Med., Houston, Tex.), C. W. Taylor and H. Busch. *Biochem Biophys Res Commun* 51(4):956-966, 1973.

1679 ADENOCARCINOMA DEVELOPING IN AN ILEOCYSTOPLASTY. *(E.)* Leedham, P. W. (London Hosp., England) and H. R. England. *Br J Surg* 60(2):158-160, 1973.

1680 THE PARTIAL PURIFICATION AND PROPERTIES OF URIDINE KINASES FROM EHRLICH ASCITES TUMOR CELLS. *(E.)* Krystal, G. (McGill U. Cancer Res. Unit, Montreal, Canada) and P. G. Scholefield. *Can J Biochem* 51(4):379-389, 1973.

1681 MITOTIC NESTS IN HUMAN VAGINAL SMEARS AND CULTURES OF AMPHIBIAN TISSUES. *(E.)* Burns, E. R. (U. Arkansas Med. Ctr., Little Rock), C. K.

Uyeda and L. E. Scheving. *Oncology* 27(1):92-96, 1973.

1682 CARCINO-FOETAL HUMAN LIVER FERRITINS. *(E.)* Alpert, E. (Harvard Med. Sch., Cambridge, Mass.), R. L. Coston and J. W. Drysdale. *Nature* 242(5394):194-195, 1973.

1683 NUCLEAR DNA IN SALIVARY GLAND TUMORS. II. ADENOCYSTIC CARCINOMAS. *(E.)* Kino, I. (Columbia Presbyterian Med. Ctr., New York, N.Y.), R. M. Richart and R. Lattes. *Arch Pathol* 95(5): 325-329, 1973.

1684 ^{67}GA-CITRATE AND ^{125}I-IODODEOXYURIDINE AS MARKERS FOR *IN VIVO* EVALUATION OF TUMOR CELL METASTASIS AND DEATH. *(E.)* Hofer, K. G. (Dept. Biol. Sci., Florida St. U., Tallahassee) and D. C. Swartzendruber. *J Nat Cancer Inst* 50(4); 1039-1045, 1973.

1685 ANTIOXIDANTS AND CANCER. I. SELENIUM IN THE BLOOD OF NORMALS AND CANCER PATIENTS. *(E.)* Shamberger, R. J. (Cleveland Clin. Fdn., Ohio), E. Rukovena, A. K. Longfield, S. A. Tytko, S. Deodhar and C. E. Willis. *J Natl Cancer Inst* 50(4):863-870, 1973.

1686 AN ESTABLISHED CULTURED CELL LINE (HBT-39) FROM HUMAN BREAST CARCINOMA. *(E.)* Plata, E. J. (Natl. Cancer Inst., Bethesda, Md.), T. Aoki, D. D. Robertson, E. W. Chu and B. I. Gerwin. *J Nat Cancer Inst* 50(4):849-862, 1973.

1687 STIMULATION AND RETARDATION OF LIMB REGENERATION BY ADENOSINE 3',5'-MONOPHOSPHATE AND RELATED COMPOUNDS. *(E.)* Foret, J. E. (Dept. Zool., U. New Hampshire, Durham). *Oncology* 27(2): 153-159, 1973.

1688 METASTASES OF HUMAN MELANOMAS TRANSPLANTED IN "NUDE" MICE. *(E.)* Giovanella, B. C. (St. Joseph Hosp., Houston Tex.), S. O. Yim, A. C. Morgan, J. S. Stehlin and L. J. Williams, Jr. *J Nat Cancer Inst* 50(4):1051-1053, 1973.

1689 RAPID GENERATION OF WHOLE-BODY RADIOAUTOGRAMS WITH MICROCHANNEL PLATE ELECTRON IMAGE AMPLIFIERS. *(E.)* Liss, R. H. (Arthur D. Little, Inc., Cambridge, Mass.), D. B. Lindsay and C. E. Catchpole. *Nature* 242(5399):523-525, 1973.

1690 COLORECTAL CARCINOMA AT THE LAHEY CLINIC, 1962 TO 1966. *(E.)* Corman, M. L. (Lahey Clin. Fdn., Boston, Mass.), N. W. Swinton, D. D. O'Keefe and M. C. Veidenheimer. *Am J Surg* 125(4):424-428, 1973.

1691 ASSOCIATION OF SOFT TISSUE SARCOMA, LEUKEMIA, AND BRAIN TUMORS IN FAMILIES AFFECTED WITH BREAST CANCER. *(E.)* Lynch, H. T. (Creighton U. Sch. Med., Omaha, Neb.), A. J. Krush, W. L. Harlan and E. A. Sharp. *Am Surg* 39(4):199-206, 1973.

1692 A MURINE RENAL CELL CARCINOMA. *(E.)* Murphy, G. P. (Roswell Park Mem. Inst., Buffalo, N.Y.) and W. J. Hrushesky. *J Natl Cancer Inst* 50(4):1013-1025, 1973.

1693 ELECTRON MICROSCOPY OF AN ACTH-SECRETING ISLET CELL CARCINOMA. *(E.)* Rawlinson, D. G. (Stanford U. Sch. Med., Calif.). *Cancer* 31(4):1015-1019, 1973.

1694 TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE ACTIVITY IN A CELL LINE (MOLT-4) DERIVED FROM THE PERIPHERAL BLOOD OF A PATIENT WITH ACUTE LYMPHOBLASTIC LEUKEMIA. *(E.)* Srivastava, B. I. S. (Roswell Park Mem. Inst., Buffalo, N.Y.) and J. Minowada. *Biochem Biophys Res Commun* 51(3):529-535, 1973.

1695 DEFECTIVE THYROGLOBULIN SYNTHESIS IN AN EXPERIMENTAL RAT THYROID TUMOR. LACK OF MEMBRANE-BOUND SIALYLTRANSFERASE ACTIVITY. *(E.)* Monaco, F. (Natl. Inst. Hlth., Bethesda, Md.) and J. Robbins. *J Biol Chem* 218(7):2328-2336, 1973.

1696 DISTRIBUTIONS OF CELL VOLUME AND DNA CONTENT OF RHABDOMYOSARCOMA CELLS GROWING *IN VITRO* AND *IN VIVO* AFTER IRRADIATION. *(E.)* Kal, H. B. (Radiobiol. Inst. TNO, Rijswijk, Netherlands). *Eur J Cancer* 9(1):77-79, 1973.

1697 SOME INTERNATIONAL DIFFERENCES IN HISTOLOGY AND SURVIVAL IN BREAST CANCER. *(E.)* Morrison, A. S. (Harvard Sch. Public Hlth., Boston, Mass.), M. M. Black, C. R. Lowe, B. MacMahon and S. Yuasa. *Int J Cancer* 11(2):261-267, 1973.

1698 MULTIPLE TUMORS IN THE PERICARDIAL CAVITY OF AN AUSTRALIAN ROCK OYSTER, *CRASSOSTREA COMMERCIALIS* (IREDALE AND ROUGHLEY). *(E.)* Dinamani, P. (Min. Agri. Fisheries, Wellington, New Zealand) and P. H. Wolf. *Int J Cancer* 11(2):293-299, 1973.

1699 HISTOLOGIC CHARACTERISTICS OF BREAST CANCER IN BOSTON AND TOKYO. *(E.)* MacMahon, B. (Harvard Sch. Public Hlth., Boston, Mass.), A. S. Morrison, L. V. Ackerman, R. Lattes, H. B. Taylor and S. Yuasa. *Int J Cancer* 11(2):338-344, 1973.

1700 LACK OF TpψP IN LOOP IV OF A MAMMALIAN INITIATOR TRANSFER RNA. *(E.)* Piper, P. W. (Med. Res. Council, Cambridge, England) and B. F. C. Clark. *FEBS Letters* 30(3):265-267, 1973.

1701 STIMULATION OF DIVISION OF DENSITY INHIBITED FIBROBLASTS BY GLUCOCORTICOIDS. *(E.)* Thrash, C. R. (California Coll. Med., U. California, Irvine) and D. D. Cunningham. *Nature* 242(5397):399-401, 1973.

1702 SERUM ALKALINE PHOSPHATASE ACTIVITY IN NORMAL AND TRANSPLANTABLE OSTEOSARCOMA-BEARING MICE. *(E.)* Shimokawa, K. (Gifu U. Sch. Med., Japan). *Acta Pathol Jap* 23(1):111-119, 1973.

1703 INFLUENCE OF CELL-FREE ASCITES FLUID AND ADENOSINE 3'5'-CYCLIC MONOPHOSPHATE UPON THE CELL KINETICS OF EHRLICH'S ASCITES CARCINOMA. *(E.)* Cooper, P. R. (Sch. Pharm., Sunderland Polytechnic, England) and H. Smith. *Nature* 241(5390):457-458, 1973.

1704 PLASMA CATECHOLAMINE AND DOPAMINE β-HYDROXYLASE AMOUNTS IN PHAEOCHROMOCYTOMA. *(E.)* Geffen, L. B. (Physiology Dept., Monash U., Clayton, Australia), R. A. Rush, W. L. Louis and A. E. Doyle. *Clin Sci* 44(4):421-424, 1973.

1705 HODGKIN'S DISEASE IN CHILDREN. *(E.)* Schnitzer, B. (U. Michigan Med. Ctr., Ann Arbor), R. H. Nishiyama, K. P. Heidelberger and D. K. Weaver. *Cancer* 31(3):560-567, 1973.

1706 COLORIMETRIC RESPONSE TO EHRLICH'S REAGENT IN PLASMA FROM PATIENTS WITH AND WITHOUT CANCER. *(E.)* Nixon, D. W. (Massachusetts Gen. Hosp., Boston). *Cancer* 31(3):596-599, 1973.

1707 PARATHYROID CARCINOMA. A STUDY OF 70 CASES. *(E.)* Schantz, A. (Massachusetts Gen Hosp., Boston) and B. Castleman. *Cancer* 31(3):600-605, 1973.

1708 CANCER OF THE COLON IN SOUTH SASKATCHEWAN. A POPULATION STUDY. *(E.)* Weir, J. A. (Allan Blair Mem. Clin., Regina, Canada). *Cancer* 31(3):616-620, 1973.

1709 OSTEOCLASTOMA-LIKE GIANT CELL TUMOR OF THE THYROID. REPORT OF A CASE WITH PROLONGED SURVIVAL FOLLOWING PARTIAL EXCISION AND RADIOTHERAPY. *(E.)* Silverberg, S. G. (Med. Coll. Virginia, Richmond) and L. S. DeGiorgi. *Cancer* 31(3):621-625, 1973.

1710 SCLEROSING STROMAL TUMORS OF THE OVARY. *(E.)* Chalvardjian, A. (Harvard Med. Sch., Boston, Mass.) and R. E. Scully. *Cancer* 31(3):664-670, 1973

1711 FEMALE ADNEXAL TUMOR OF PROBABLE WOLFFIAN ORIGIN. A DISTINCTIVE PATHOLOGIC ENTITY. *(E.)* Kariminejad, M. H. (Massachusetts Gen Hosp., Boston) and R. E. Scully. *Cancer* 31(3):671-677, 1973.

1712 THE *IN VITRO* LOCALIZATION OF H_3 ESTRADIOL IN HUMAN PROSTATIC CARCINOMA. AN ELECTRON MICROSCOPIC AUTORADIOGRAPHIC STUDY. *(E.)* Sinha, A. A. (VA Hosp., Minneapolis, Minn.), C. E. Blackard, R. P. Doe and U. S. Seal. *Cancer* 31(3):682-688, 1973.

1713 ACID PHOSPHATASE ISOZYMES IN CANCER OF THE PROSTATE. *(E.)* Reif, A. E. (Tufts U. Sch. Med., Boston, Mass.), R. M. Schlesinger, C. A. Fish and C. M. Robinson. *Cancer* 31(3):689-699, 1973.

1714 SQUAMOUS CARCINOMA ARISING IN THE PITUITARY GLAND. *(E.)* Salyer, D. (Johns Hopkins Hosp., Baltimore, Md.) and D. Carter. *Cancer* 31(3):713-718, 1973.

1715 PRIMARY THYMIC SEMINOMA—A NEOPLASM ULTRASTRUCTURALLY SIMILAR TO TESTICULAR SEMINOMA AND DISTINCT FROM EPITHELIAL THYMONA. *(E.)* Levine, G. D. (Stanford Med. Ctr., Calif.). *Cancer* 31(3):729-741, 1973.

1716 CARCINOMA OF MECKEL'S DIVERTICULUM. *(E.)* Johnson, H. R. M. (St. Thomas' Hosp. Med. Sch., London, England). *Cancer* 31(3):742-745, 1973.

1717 THE CENTRAL NERVOUS SYSTEM IN CHILDHOOD LEUKEMIA: I. THE ARACHNOID. *(E.)* Price, R. A. (St. Jude Children's Res. Hosp., Memphis, Tenn.) and W. W. Johnson. *Cancer* 31(3):520-533, 1973.

1718 PROTEINACEOUS PRECIPITATE IN NODULAR (FOLLICULAR) LYMPHOMAS. *(E.)* Rosas-Uribe, A. (U. Chicago Sch. Med., Ill.), D. Variakojis and H. Rappaport. *Cancer* 31(3):534-542, 1973.

1719 LEUKEMIC PHASE OF RETICULUM CELL SARCOMA (HISTIOCYTIC LYMPHOMA). A CLINICOPATHOLOGIC AND ULTRASTRUCTURAL STUDY. *(E.)* Schnitzer, B. (U. Michigan Med. Ctr., Ann Arbor) and L. Kass. *Cancer* 31(3):547-559, 1973.

1720 LONG TERM CULTURES OF HODGKIN'S TISSUE. A MORPHOLOGIC AND RADIOAUTOGRAPHIC STUDY. *(E.)* Kadin, M. E. (U. California Med. Ctr., San Francisco) and A. K. Asbury. *Lab Invest* 28(2):181-184, 1973.

1721 CONTROL OF LIPID METABOLISM IN HEPATOMAS: CONVERSION OF GLUTAMATE CARBON TO FATTY ACID CARBON *VIA* CITRATE IN SEVERAL TRANSPLANTABLE HEPATOMAS. *(E.)* Sabine, J. R. (Dept. Phys.-Anat.,

U. California, Berkeley), L. Kopelovich, S. Abraham and H. P. Morris. *Biochim Biophys Acta* 296(3):493-498, 1973.

1722 HYBRIDIZATION STUDIES WITH NUCLEIC ACIDS FROM MURINE MYELOMAS. KINETICS OF THE REACTION AND CHARACTERIZATION OF THE HYBRID. *(E.)* Krueger, R. G. (Mayo Fdn., Rochester, Minn.) and K. Kosky. *Biochemistry* 12(7):1299-1306, 1973.

1723 ACTIVE OXIDATIVE DECARBOXYLATION OF MALATE BY MITOCHONDRIA ISOLATED FROM L-1210 ASCITES TUMOR CELLS. *(E.)* Hansford, R. G. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and A. L. Lehninger. *Biochem Biophys Res Commun* 51(2):480-486, 1973.

1724 *IN VITRO* COLONY FORMATION BY NORMAL AND LEUKEMIC HUMAN HEMATOPOIETIC CELLS: INTERACTION BETWEEN COLONY-FORMING AND COLONY-STIMULATING CELLS. *(E.)* Moore, M. A. S. (Royal Melbourne Hosp., Australia), N. Williams and D. Metcalf. *J Natl Cancer Inst* 50(3):591-602, 1973.

1725 *IN VITRO* COLONY FORMATION BY NORMAL AND LEUKEMIC HUMAN HEMATOPOIETIC CELLS: CHARACTERIZATION OF THE COLONY-FORMING CELLS. *(E.)* Moore, M. A. S. (Royal Melbourne Hosp., Australia), N. Williams and D. Metcalf. *J Nat Cancer Inst* 50(3): 603-623, 1973.

1726 SPONTANEOUS TUMORS AND PATHOLOGIC LESIONS IN SWR/J MICE. *(E.)* Rabstein, L. S. (Microbiol. Associates, Inc., Walkersville, Md.), R. L. Peters and G. J. Spahn. *J Nat Cancer Inst* 50(3):751-758, 1973.

1727 LIVER-CELL CARCINOMA PRESENTING AS BILE-DUCT TUMOUR. *(E.)* Waldron, R. L. (Mount Auburn Hosp., Cambridge, Mass.), G. Kenny and K. Sorger. *Br J Radiol* 46(543):195-197, 1973.

1728 CYST PUNCTURE AND ENDOMYELOGRAPHY IN CYSTIC TUMOURS OF THE SPINAL CORD. *(E.)* Kendall, B. (Natl. Hosp. Nervous Dis., London, England) and L. Symon. *Br J Radiol* 46(543):198-204, 1973.

1729 ACUTE LYMPHOBLASTIC LEUKAEMIA: OF T-CELL ORIGIN? *(E.)* Szekely, I. E. (Harrisburg Hosp., Pa.), D. R. Fisher and H. R. Schumacher. *Lancet* (7806):768-769, 1973.

1730 *IN VITRO* CHARACTERISTICS OF HUMAN GLIOBLASTOMAS MAINTAINED IN ORGANIC CULTURE SYSTEMS. LIGHT MICROSCOPY OBSERVATIONS. *(E.)* Rubinstein, L. J. (Stanford U. Sch. Med., Calif.), M. M. Herman and V. L. Foley. *Am J Pathol* 71(1):61-80, 1973.

1731 SUPPRESSION OF ANTERIOR PITUITARY IN RATS BEARING A TRANSPLANTABLE GROWTH HORMONE AND PROLACTIN-SECRETING TUMOR (MtT-W10). *(E.)* Nakayama, I. (Dept. Path. St. U. New York, Buffalo) and P. A. Nickerson. *Endocrinology* 92(2):516-524, 1973.

1732 HYPOXIC CELLS IN AN *IN VITRO* TUMOUR MODEL. *(E.)* Sutherland, R. M. (London Clin., Canada) and R. E. Durand. *Int J Radiat Biol* 23(3): 235-246, 1973.

1733 METHYLATION OF SATELLITE DEOXYRIBONUCLEIC ACID IN MOUSE NEOPLASTIC AND NON-NEOPLASTIC CELL CULTURES. *(E.)* Gantt, R. (Natl. Cancer Inst., Bethesda, Md.), F. Montes De Oca and V. J. Evans. *In Vitro* 8(4):288-294, 1973.

1734 THE CONTINUING CHALLENGE OF COLORECTAL CARCINOMA. *(E.)* Burdette, W. J. (U. Texas, Houston). *Hosp Pract* 8(5):146-153, 1973.

1735 SUPPRESSED FOLLICLE—STIMULATING HORMONE (FSH) IN WOMEN WITH GESTATIONAL TROPHOBLASTIC NEOPLASMS. *(E.)* Reiter, E. O. (Natl. Inst. Hlth., Bethesda, Md.), G. D. Braunstein, S. M. Hamwood and H. E. Kulin. *J Clin Endocrinol Metab* 36(4):697-701, 1973.

1736 LYSOZYME ACTIVITY AND NITROBLUE-TETRAZOLIUM REDUCTION IN LEUKAEMIC CELLS. *(E.)* Catovsky, D. (Royal Postgrad. Med. Sch., London, England) and D. A. G. Galton. *J Clin Pathol* 26(1): 60-69, 1973.

1737 VARIABLE SEX CHROMATIN PATTERN IN AN EARLY CARCINOMA OF THE BLADDER. *(E.)* Atkin, N. B. (Mount Vernon Hosp., Northwood, England) and I. Petkovic. *J Clin Pathol* 26(2):126-129, 1973.

1738 HEMOGLOBIN ABNORMALITIES IN NEOPLASTIC HEMATOLOGICAL DISORDERS. *(E.)* Walker, I. R. (Hamilton Gen. Hosp., Canada) and M. A. M. Ali. *Can Med Assoc J* 108(7):843-847, 1973.

1739 ON THE RESTING STAGES OF THE JB-1 ASCITES TUMOUR. *(E.)* Bichel, P. (Cancer Res. Inst., Radium Station, Aarhus, Denmark) and P. Dombernowsky. *Cell Tissue Kinet* 6(4):359-367, 1973.

1740 ADENOMATOUS POLYP WITH SUBMUCOSAL CYSTS. *(E.)* Fechner, R. E. (Baylor Coll. Med., Houston, Tex.). *Am J Clin Pathol* 59:498-502, 1973.

1741 ULTRASTRUCTURAL STUDY OF VIRILIZING ADRENOCORTICAL ADENOMA. *(E.)* Fisher, E.

R. (Shadyside Hosp., Pittsburgh, Pa.) and T. S. Danowski. *Am J Clin Pathol* 59(4):480-489, 1973.

1742 ACID MUCOPOLYSACCHARIDES IN TUMOURS OF THE MYELIN SHEATH CELLS, THE OLIGODENDROGLIOMA AND THE NEURILEMMOMA. *(E.)* Smith, B. (St. Bartholomew's Hosp., London, England) and M. Butler. *Acta Neuropathol (Berl)* 23(2):181-185, 1973.

1743 ULTRASTRUCTURE OF A COMPOSITE GLIOMA-SARCOMA OF THE BRAIN. *(E.)* Peña, C. E. (Georgetown U. Med. Ctr., Washington, D.C.) and R. Felter. *Acta Neuropathol (Berl)* 23(1):90-94, 1973.

1744 MALIGNANT GIANT-CELL TUMOUR OF THE COLON. *(E.)* Eshun-Wilson, K. (Odense U. Hosp., Denmark). *Acta Pathol Microbiol Scand* [*A*] 81(2): 137-144, 1973.

1745 RECURRENT FIBROSARCOMA OF THE SKIN PRECEDED BY INFLAMMATION. *(E.)* Welvaart, K. (Univ. Hosp., Leyden, Netherlands) and M. K. Polano. *Arch Chir Neerl* 25(1):35-42, 1973.

1746 TUMOURS OF THE SIGMOID COLON FOLLOWING URETEROSIGMOIDOSTOMY. *(E.)* Lien, W. M. (Queen Elizabeth Hosp., Birmingham, England). *Postgrad Med J* 49(569):209-211, 1973.

1747 CARCINOMA OF THE COLON ASSOCIATED WITH URETEROSIGMOIDOSTOMY: REPORT OF AN UNUSUAL CASE. *(E.)* Myers, J. W. (St. Joseph Mercy Hosp., Ann Arbor, Mich.). *Mich Med* 72(12): 285-287, 1973.

1748 HISTOCHEMICAL OBSERVATIONS ON NDPase AND TPPase IN CEREBRAL TUMOURS. *(E.)* Schiffer, D. (Clin. Nervous, Mental Dis., U. Turin, Italy). *Acta Neuropathol (Berl)* 23(2):112-117, 1973.

1749 ACTH AND CALCITONIN SECRETING MEDULLARY CARCINOMA OF THE THYROID. *(E.)* Croughs, R. J. M. (U. Hosp., Rotterdam, Netherlands), W. N. Eastham, W. H. L. Hackeng, W. Schopman, T. M. Feltkamp-Vroom, A. Dolman and G. Hennemann. *Clin Endocrinol* 1(2):157-171, 1972.

1750 SOME PROPERTIES OF A PYRIMIDINE PHOSPHORIBOSYLTRANSFERASE FROM MURINE LEUKEMIA CELLS. *(E.)* Kessel, D. (U. Rochester Sch. Med. Dent., N.Y.), J. Deacon, B. Coffey and A. Bakamjian. *Mol Pharmacol* 8(6):731-739, 1972.

1751 ARGENTAFFIN CELLS IN HUMAN THYROID AND PARATHYROID AND THEIR RELATIONSHIP TO C-CELLS AND MEDULLARY CARCINOMA. *(E.)* Ljungberg, O. (U. Lund, Gen. Hosp., Malmö, Sweden). *Acta Pathol Microbiol Scand* [*A*] 80(5):589-599, 1972.

1752 ABSENCE OF PROSTAGLANDIN SYNTHESIS IN THE MALIGNANT HUMAN TROPHOBLAST IN CULTURE. *(E.)* Pattillo, R. A. (Med. Coll. Wisconsin, Milwaukee), R. O. Hussa, N. A. Terragno, M. T. Story and R. F. Mattingly. *Am J Obstet Gynecol* 115(1): 91-94, 1973.

1753 CONSIDERATIONS ON THE CORRELATION BETWEEN THE BLOOD GROUPS AND THE INCIDENCE OF CEREBRAL TUMOURS. *(E.)* Arseni, C. (Gh. Marinescu Hosp., Bucharest, Romania), L. Danaila and A. I. Constantinescu. *Rev Roum Neurol* 9(3):135-138, 1972.

1754 CYTOGENETIC STUDIES ON CULTURED FIBROBLAST-LIKE CELLS DERIVED FROM BASAL CELL CARCINOMA TISSUE. *(E.)* Happle, R. (Inst. Human Genetics, U. Freiburg, West Germany) and H. Hoehn. *Clin Genet* 4(1): 17-24, 1973.

1755 INTRANUCLEAR MITOCHONDRIAE IN HODGKIN'S DISEASE. *(E.)* Oliva, H. (Dept. Path., U. Madrid, Spain), A. Valle, L. Diaz Flores and M. C. Rivas. *Virchows Arch* [*Zellpathol*] 12(2):189-194, 1973.

1756 TUMOR CELL INVASION OF HEPATOCYTES IN TRANSPLANTABLE PLASMA CELL NEOPLASM OF MICE. *(E.)* Sarkar, K. (Fac. Med., U. Ottawa, Canada). *Virchows Arch* [*Zellpathol*] 11(4):303-309, 1972.

1757 CYTOGENETIC STUDIES DURING REMISSION OF BLASTIC CRISIS IN A PATIENT WITH CHRONIC MYELOCYTIC LEUKAEMIA. *(E.)* Srodes, C. H. (U. Pittsburgh Sch. Med., Pa.), E. F. Hyde, S. F. Pan, P. A. Chervenick and D. R. Boggs. *Scand J Haematol* 10:130-135, 1973.

1758 STEROID SULFURYLATION IN NORMAL TISSUES AND MAMMARY TUMORS: A MICRO METHOD. *(E.)* Leung, B. S. (U. Oregon Med. Sch., Portland), W. Jack and D. C. Wood. *Physiol Chem Phys* 4(6):543-552, 1972.

1759 THE FINE STRUCTURE OF EPENDYMOBLASTOMA. *(E.)* Hirano, A. (Montefiore Hosp. Med. Ctr., New York, N.Y.), N. R. Ghatak and H. M. Zimmerman. *J Neuropathol Exp Neurol* 32(1):144-152, 1973.

1760 GRANULAR CELL TUMORS OF THE CENTRAL NERVOUS SYSTEM. *(E.)* Markesbery, W. R. (U. Rochester Sch. Med. Dent., N.Y.), P. E. Duffy and D. Cowen. *J Neuropathol Exp Neurol* 32(1):92-109, 1973. 92-109, 1973.

1761 SOLUBLE AND MEMBRANE-BOUND ADENYLATE CYCLASE ACTIVITY IN YOSHIDA ASCITES HEPATOMA. *(E.)* Tomasi, V. (Inst. Gen. Phys., U. Ferrara, Italy), A. Rethy and A. Trevisani. *Life Sci* [*II*] 12(4):145-150, 1973.

1762 KETONE BODY OXIDATION BY MOUSE HEPATOMA BW7756. *(E.)* Fenselau, A. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and K. Wallis. *Life Sci* [*II*] 12(4):185-191, 1973.

1763 TYROSINASE ACTIVITIES OF CELL-FREE EXTRACTS AND LIVING CELLS OF CULTURED MELANOMA CELLS. *(E.)* Oikawa, A. (Natl. Cancer Ctr. Res. Inst., Tokyo, Japan), M. Nakayasu and M. Nohara. *Dev Biol* 30(1):198-205, 1973.

1764 PSYCHOBIOLOGICAL CHARACTERISTICS IN YOUTH AS PREDICTORS OF FIVE DISEASE STATES: SUICIDE, MENTAL ILLNESS, HYPERTENSION, CORONARY HEART DISEASE AND TUMOR. *(E.)* Thomas, C. B. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and R. L. Greenstreet. *Johns Hopkins Med J* 132(1): 16-43, 1973.

1765 ELECTROKINETIC CHARACTERIZATION OF TWO ASCITES TUMOURS: TA3 AND M.Mel. 1. *(E.)* Roeder, C. (Inst. Path., U. Zürich, Switzerland), G. M. W. Cook, G. Haemmerli and P. Sträuli. *J Cell Sci* 12(1):235-251, 1973.

1766 HEATS OF ACTIVATION FOR THE EXOSMOTIC FLOW OF WATER ACROSS THE MEMBRANE OF LEUCOCYTES AND LEUKEMIC CELLS. *(E.)* Hempling, H. G. (Med. U. South Carolina, Charleston). *J Cell Physiol* 81(1): 1-10, 1973.

1767 NUCLEOTIDE POOLS OF NOVIKOFF RAT HEPATOMA CELLS GROWING IN SUSPENSION CULTURE. IV. NUCLEOSIDE TRANSPORT IN CELLS DEPLETED OF NUCLEOTIDES BY TREATMENT WITH KCN. *(E.)* Plagemann, P. G. W. (Med. Sch., U. Minnesota, Minneapolis) and J. Erbe. *J Cell Physiol* 81(1):101-112, 1973.

1768 HISTOLOGIC, ULTRASTRUCTURAL, AND HORMONAL CHARACTERIZATION OF A PITUITARY TUMOR SECRETING BOTH hGH AND PROLACTIN. *(E.)* Guyda, H. (Dept. Endocrinol., McGill U., Montreal, Canada), F. Robert, E. Colle and J. Hardy. *J Clin Endocrinol Metab* 36(3):531-547, 1973.

1769 ULTRASTRUCTURE OF GIANT CELL TUMORS OF BONE. *(E.)* Steiner, G. C. (Mount Sinai Sch. Med., City U. New York, N.Y.), L. Ghosh and H. D. Dorfman. *Hum Pathol* 3(4):569-586, 1973.

1770 THE ULTRASTRUCTURE OF THE LYMPHORETICULAR CELLS IN NON-LYMPHOID HUMAN NEOPLASMS. *(E.)* Underwood, J. C. E. (Weston Park Hosp., Sheffield, England) and I. Carr. *Virchows Arch* [*Zellpathol*] 12(1):39-50, 1972.

1771 TRANSPORT OF α-OXOGLUTARATE IN MITOCHONDRIA OF TUMOUR CELLS AND ITS FUNCTIONAL IMPLICATIONS. *(E.)* Papa, S. (Dept. Biochem. U. Bari, Italy), G. Paradies, T. Galeotti, O. Dionisi and M. L. Eboli. *Nature* [*New Biol*] 242(116):86-87, 1973.

1772 BIOLOGICAL RESPONSES OF TUMOUR CELLS AFTER DIFFERENT *IN VIVO* CHEMICAL STRESS. *(E.)* Chowdhury, J. R. (Chittaranjan Natl. Cancer Res. Ctr., Calcutta, India) and S. Bose. *Indian J Cancer* 9(3):203-209, 1972.

1773 PHOSPHATE DEPLETION AND GLUCOCORTICOID-INDUCED HYPERPHOSPHATEMIA IN LYMPHOBLASTIC LEUKEMIA. *(E.)* Clarkson, D. R. (Washington U. Sch. Med., St. Louis, Mo.), J. Blondin and P. E. Cryer. *Metabolism* 22(4):611-616, 1973.

1774 A NEW TECHNIQUE FOR DETERMINING THE NUMBER OF L1210 CLONOGENIC CELLS. *(Fr.)* Chevalie C. (Inst. Radiobiol. Clin., I.N.S.E.R.M., Villejuif France) and E. Frindel. *Bull Cancer (Paris)* 59(3): 339-344, 1972.

1775 ULTRASTRUCTURE OF A TUMOR OF THE RETINA. *(Fr.)* Choux, R. (Lab. Anat. Path. Neuropath., Marseille, France), M. F. Tripier, M. Bérard, J. Hassoun and M. Toga. *Bull Cancer (Paris)* 59(3): 301-314, 1972.

1776 SEPARATION OF VIABLE FROM NONVIABLE TUMOR CELLS BY FLOTATION ON A FICOLL-TRIOSIL MIXTURE. *(E.)* Vries, J. E. de (Netherlands Cancer Inst., Amsterdam), M. van Benthem and P. Rümke. *Transplantation* 15(4):409-410, 1973.

1777 CERTAIN FEATURES OF GLYCOGEN PHOSPHORYLASE B ISOLATED FROM HUMAN MYOMA OF THE UTERUS. *(Rus.)* Viktorova, L. N. (USSR Acad. Med. Sci., Moscow), E. V. Ramensky and G. V. Safina. *Biull Eksp Biol Med* 74(2):41-43, 1973.

1778 STUDIES OF MURAMIDASE IN HAEMATOLOGICAL DISORDERS: SERUM AND MARROW MURAMIDASE IN LEUKEMIA. *(E.)* Levi, J. A. (Sydney Hosp., Australia), J. B. Speden, P. C. Vincent and F. W. Gunz. *Pathology* 5(1):59-68, 1973.

1779 HAEMATOLOGICAL AND CLINICAL PARAMETERS IN ASSESSING ACTIVITY IN HODGKIN'S DISEASE AND OTHER MALIGNANT LYMPHOMAS. *(E.)* Simmons, A. V. (U. Coll. Hosp., London, England), A. S. D. Spiers and P. M. Fayers. *Q J Med* 42(165):111-124, 1973.

1780 CYTOMORPHOLOGY, IMMUNOCHEMISTRY AND THERAPEUTICAL FEATURES OF MULTIPLE MYELOMA. *(It.)* Maroncelli, P. (S. Camillo Hosp., Rome, Italy) and C. Casalino. *Minerva Med* 64(8):337-357, 1973.

1781 CONNATAL INTRACRANIAL TUMOURS. *(E.)* Jellinger, K. (Neurol. Inst., U. Vienna, Austria) and M. Sunder-Plassmann. *Neuropaediatrie* 4(1):46-63, 1973.

1782 INCORPORATION AND RELEASE OF ^{51}Cr AND ^{14}C-LABELED AMINO ACIDS IN EHRLICH ASCITES CARCINOMA CELLS. *(E.)* Maslow, D. E. (Roswell Park Mem. Inst., Buffalo, N.Y.). *J Nucl Med* 14(2):84-88, 1973.

1783 MORPHOLOGICAL STUDIES OF THE VASCULAR STRUCTURE OF IMPLANTED TUMORS IN RATS. *(Fr.)* Aumüller, G. (Fac. Med. Ruprecht-Karl-U., Heidelberg, West Germany), P. Vaupel and H. Günther. *Bull Cancer (Paris)* 59(2):225-232, 1972.

1784 MALIGNANT NEOPLASMS OF THE SKIN IN PONDICHERRY (A STUDY OF 102 CASES). *(E.)* Budhraja, S. N. (Jawaharlal Inst. Post-Grad. Med. Educ. Res., Pondicherry, India), V. C. Velayudhan Pillai, W. J. Periyanayagam, S. P. Kaushik and B. M. S. Bedi. *Indian J Cancer* 9(4):284-295, 1972.

1785 SEQUENTIAL ENZYME CHANGES IN SPONTANEOUS MAMMARY CARCINOGENESIS IN RECIPROCAL CROSSES OF ICRC AND C3H (JAX) STRAINS. *(E.)* Gupte, N. V. (Cancer Res. Inst., Bombay, India) and S. V. Bhide. *Indian J Cancer* 9(4):280-283, 1972.

1786 SEQUENTIAL STUDIES ON SPONTANEOUS MAMMARY AND HEPATOCARCINOGENESIS IN RECIPROCAL CROSSES OF ICRC AND C3H (JAX) STRAINS. *(E.)* Gupte, N. V. (Tata Mem. Hosp., Bombay, India), S. V. Bhide, G. V. Talvalkar and K. J. Ranadive. *Indian J Cancer* 9(4):273-279, 1972.

1787 CYTOKINETIC ANALYSIS OF THE JB-1 ASCITES TUMOUR AT DIFFERENT STAGES OF GROWTH. *(E.)* Dombernowsky, P. (Dept. Internal Med., Finsen Inst., Copenhagen, Denmark), P. Bichel and N. R. Hartmann. *Cell Tissue Kinet* 6(4):347-357, 1973.

1788 THROMBOPLASTIC ACTIVITY OF CHRONIC MYELOGENOUS LEUKAEMIA GRANULOCYTES. *(E.)* Lisiewicz, J. (Med. Acad., Cracow, Poland), B. Malkiewicz-Wasowicz and B. Biernacka. *Folia Haematol (Leipz)* 99(1):57-64, 1973.

1789 CHOLESTEROL CONTENT AND PHOSPHOLIPID COMPOSITION OF MORRIS HEPATOMA MICROSOMES. *(E.)* Filipek-Wender, H. (Med. Acad., Poznan, Poland), H. Karon and L. Torlinski. *Acta Biol Med Germ* 29(6): 823-829, 1972.

1790 INTERCISTERNAL STRUCTURES OF CLOSELY ARRANGED ENDOPLASMIC RETICULUM IN HUMAN MENINGIOMA. *(E.)* Tani, E. (Kyoto U. Med. Sch., Japan) and N. Higashi. *Acta Neuropathol (Berl)* 23(4): 291-299, 1973.

1791 FLUCTUATION OF LIPID CONTENT IN EHRLICH CARCINOMA CELLS DURING THE GROWTH PERIOD. *(E.)* Saito, T. (Dept. Clin. Cancer Chemotherap., Tohoku U., Sendai, Japan), A. Wakui, J.-I. Kikkawa, T. Kudo, K. Kikuchi and T. Ichikawa. *Tohoku J Exp Med* 108(1):63-77, 1972.

1792 AMYLOID-CONTAINING ODONTOGENIC TUMORS OF MAN. *(E.)* Meenaghan, M. A. (Sch. Dent., St. U. New York, Buffalo), B. N. Appel and G. W. Greene, Jr. *Oral Surg* 34(6):908-919, 1972.

1793 RIBONUCLEOTIDE REDUCTASE ACTIVITY OF VARIOUS STRUCTURAL ELEMENTS OF M 1 SARCOMA CELLS OF RATS. *(Rus.)* Silaeva, S. A. (1st Med. Sch., Moscow, USSR), S. S. Debov and N. B. Chesnokova. *Vopr Med Khim* 42(2):400-403, 1972.

1794 THE MICROTUBULES OF LEUKAEMIC RIEDER CELLS: AN ULTRASTRUCTURAL STUDY. *(E.)* Cawley, J. C. (Dept. Med., U. Cambridge, England). *Scand J Haematol* 9(5):417-423, 1972.

1795 FIBROSIS AND CARCINOMA OF THE LUNG IN A FAMILY WITH HAEMOGLOBIN MALMÖ. ANATOMIC FINDINGS. *(E.)* Berglund, S. (Malmö Gen. Hosp., Sweden) and F. Linell. *Scand J Haematol* 9(5):424-432, 1972.

1796 A STUDY OF THE CYCLIC AMP SYSTEM IN LEUKEMIA. *(E.)* Polgar, P. (Boston U. Sch. Med., Mass.), C. P. Emerson, Jr. and A. M. Rutenburg. *Proc Am Assoc Cancer Res* 13(March):119, 1972.

1797 PURINE METABOLISM IN HUMAN LYMPHOCYTES AND LEUKEMIC CELLS. *(E.)* Scholar, E. M. (Roger Williams Gen. Hosp., Providence, R. I.), R. E. Parks, Jr. and P. Calabresi. *Proc Am Assoc Cancer Res* 13(March):114, 1972.

1798 THE NATURE OF THE CYTOLOGICAL HLM (HYPOSULFITE-LEUKOBASE-METHYLENE BLUE) REACTION WHICH DISTINGUISHES NORMAL TISSUE FROM MALIGNANT NEOPLASTIC TISSUE CELLS IN HUMANS AND ANIMALS. *(Rus.)* Yevreinova, T. N. (Lomonosov U. Moscow, USSR) and M. Ye. Struve. *Dokl Akad Nauk SSSR* 209(3):725-727, 1973.

1799 CYTOLOGIC, HISTOLOGIC, AND CYTOPHOTOMETRIC FINDINGS IN AN ADENOCARCINOMA *IN SITU* OF THE UTERINE CERVIX. *(Ger.)* Sachs, H. (Women's Clin. U. Hamburg-Eppendorf, W. Germany) and K. Würthner. *Geburtshilfe Frauenheilkd* 32(10): 846-849, 1972.

1800 THE OCCURRENCE OF OVARIAN TUMORS AS AFFECTED BY HYSTERECTOMY AND UNILATERAL OVARECTOMY. *(Ger.)* Wagenbichler, P. (Women's Clin. U. Vienna, Austria), H. Frauendorfer and L. Havelec. *Geburtshilfe Frauenheilkd* 32(10):882-890, 1972.

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

AARONSON, S.A.
1379
ABACI, I.F.
1319*
ABEL, G.
1618
ABEYOUNIS, C.J.
1546*
ABRAHAM, S.
1721*
ACKERMAN, L.V.
1699*
ADLER, S.P.
1417*
AHLQVIST, J.
1549*
AHMED, N.
1542*
AHSTROEM, L.
1538*
AISENBERG, A.C.
1491
AL-SARRAF, M.
1525*
ALAVARES, A.P.
1298*
ALBERT, R.E.
1307
ALEXANDER, E.R.
1216
ALEXANDER, P.
1497, 1528*
ALI, M.A.M.
1738*
ALLALOUF, D.
1494
ALLEN, J.R.
1240
ALLEN, P.M.
1619
ALLISON, A.C.
1354
ALPERT, E.
1682*
ALPERT, L.I.
1319*
ALVAREZ, M.R.
1643
AMES, R.P.
1655*
ANDERSSON, B.
1339
ANDREWS, E.J.
1240
AOKI, T.
1686*
APPEL, B.N.
1792*
ARAI, M.
1271
ARCHER, V.E.
1588
ARMSTRONG, M.Y.K.
1321
ARNOLD, W.
1567*
ARSENI, C.
1753*
ASBURY, A.K.
1720*
ASOFSKY, R.
1515*
ATKIN, N.B.
1737*
AUMUELLER, G.
1783*
AUSTEN, K.F.
1558*
AUSTOKER, J.
1272
AXEL, R.
1392
BABKOVA, O.V.
1557*
BACCARANI, M.
1608
BACCICHETTI, F.
1316*
BADER, J.P.
1389
BAGAI, R.C.
1520*
BAHNER, C.T.
1255
BAKAMJIAN, A.
1750*
BAKER, M.A.
1575*
BALCERZAK, S.P.
1560*
BALDWIN, R.W.
1459, 1466
BANCEWICZ, J.
1287*
BANERJEE, S.N.
1305
BANG, F.B.
1351
BARBER, B.H.
1495
BARESOVA, M.
1458
BARKER, A.D.
1570*
BARKER, C.R.
1534*
BAROWSKY, N.J.
1611
BART, R.S.
1587*
BARTSCH, H.
1235
BASS, E.M.
1450
BATTULA, N.
1614
BATZING, B.L.
1377
BAUER, G.E.
1670*
BAUSHER, J.C.
1386
BECKER, H.D.
1627
BEDI, B.M.S.
1784*
BENACERROF, B.
1492
BENNETT, P.
1307
BENTVELZEN, P.
1211, 1355
BENTWICH, Z.
1498
BENVENISTE, R.E.
1334
BENYESH-MELNICK, M.
1432*
BER, A.
1494
BERARD, M.
1775*
BERGLUND, S.
1795*
BERKE, G.
1503
BEY, E.M.
1364, 1371
BHAGAVAN, B.S.
1584
BHAKTAVIZIAM, A.
1279
BHIDE, S.V.
1785*, 1786*
BICHEL, P.
1739*, 1787*
BICKERS, D.R.
1298*
BIEDLER, J.L.
1613
BIERNACKA, B.
1788*
BIGGS, P.M.
1487
BILLIAU, A.
1347
BINGHAM, E.
1277
BISHOP, J.M.
1411
BISWAL, N.
1432*
BISWAS, R.K.
1316*
BLACK, M.M.
1697*
BLACK, P.H.
1394, 1422*, 1436
BLACKARD, C.E.
1712*
BLAKE, E.R.
1448
BLANGY, D.
1358
BLAZKOVEC, A.A.
1475
BLOCH, K.J.
1491
BLONDIN, J.
1773*
BLOOM, A.D.
1572*
BLORE, J.
1661*

BOBOVICH, S.I. 1317*
BOECKER, W.R. 1612
BOGGS, D.R. 1757*
BOMFORD, R. 1530*
BON, W.F. 1256
BONE, G. 1518*
BONMASSAR, A. 1480
BONMASSAR, E. 1480
BORCHERT, P. 1262, 1274
BOREK, C. 1300
BOREL, D.M. 1223*
BORNER, G. 1612
BOSE, S. 1772*
BOSTOCK, D.E. 1228*
BOUCHER, B.J. 1294*
BOURNOUTIAN, C. 1366
BOUTWELL, R.K. 1214, 1275
BOWDEN, G.T. 1275
BOWEN, J.M. 1359
BRALOW, S.P. 1257
BRANTON, P.E. 1357
BRAUNSTEIN, G.D. 1735*
BRDAR, B. 1413*
BRECKENRIDGE, B.MCL. 1665*
BRENNAN, M.J. 1651
BRENT, T.P. 1313*
BRITCHFORD, R.D. 1534*
BRITTINGER, G. 1653
BRODSKY, I. 1428*
BROOKES, P. 1234
BROSS, I.D.J. 1590
BROWNE, E. 1536*
BUBENIK, J. 1458
BUDHRAJA, S.N. 1784*
BUELL, P. 1595*
BUETTNER, W. 1402
BULBROOK, R.D. 1651
BULL, M. 1225*
BURCH, P.R.J. 1598*
BURDETTE, W.J. 1734*
BURDICK, J.F. 1569*
BURGER, P.J. 1656*
BURGESS, J. 1585*
BURK, P.G. 1301
BURKE, P.J. 1640
BURKHOLDER, P.M. 1475
BURNET, F.M. 1201
BURNS, E.R. 1681*
BURNS, F.J. 1307
BURNY, A. 1392
BURTIN, P. 1537*
BUSCH, H. 1615, 1678*
BUTLER, M. 1742*
BYKOREZ, A.I. 1631
CABINESS, J.R. 1542*
CABRAL, J.R. 1238
CALABRESI, P. 1797*
CALDWELL, J.R. 1558*
CALVANICO, N.J. 1559*
CAMERON, E. 1213
CAMPBELL, J.S. 1286*
CANNAT, A. 1329
CANTERO, A. 1207
CANTOR, H. 1515*
CAPPS, M.J. 1239
CAPUTO, A. 1524*
CARNEY, J.D. 1597*
CARR, I. 1770*
CARRARA, G. 1524*
CARSTEN, A.L. 1612
CARTER, D. 1714*
CARTER, R.L. 1581
CARVALHO, Z.G. 1504
CARVER, J.P. 1495
CASALE, G.P. 1551*
CASALINO, C. 1780*
CASPARY, E.A. 1449
CASTELLI, L. 1524*
CASTLEMAN, B. 1707*
CASTO, B. 1363
CASTO, B.C. 1331
CASTRO, J.E. 1451
CATCHPOLE, C.E. 1689*
CATOVSKY, D. 1736*
CAWLEY, J.C. 1534*, 1794*
CHALVARDJIAN, A. 1710*
CHAN, J.C. 1373
CHANDRA, P. 1316*
CHANDRADASA, K.D. 1454
CHARLIONET, R. 1505*
CHARNEY, J. 1361
CHAUVEAU, J. 1233, 1245
CHAVES, E. 1675*
CHERRY, M. 1375
CHERVENICK, P.A. 1757*
CHESNOKOVA, N.B. 1793*
CHESTERMAN, F.C. 1354
CHEVALIER, C. 1774*
CHIKKAPPA, G. 1612
CHIRIGOS, M.A. 1421*
CHOUX, R. 1775*
CHOWANIEC, J. 1242

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

CHOWDHURY, J.R.
1772*
CHRISTOPHOROV, L.
1521*
CHU, E.W.
1686*
CIHAK, R.W.
1659*
CIOE, L.
1524*
CIUFECU, E.
1430*
CLAGETT, J.
1440, 1444
CLARK, B.F.C.
1700*
CLARK, D.A.
1545*
CLARKSON, D.R.
1773*
CLIFTON, K.H.
1303
COFFEY, B.
1750*
COHEN, A.M.
1569*
COHN, S.L.
1634
COHNEN, G.
1653
COLLE, E.
1768*
COLVIN, R.B.
1491
COMOGLIO, P.M.
1441
CONKLING, K.
1612
CONNORS, T.A.
1618
CONSTANTINESCU, A.I.
1753*
COOK, G.M.W.
1765*
COOK, L.
1612
COOPER, P.R.
1703*
CORMAN, M.L.
1690*
CORNELIUS, E.A.
1485
CORY, J.G.
1649
COSTON, R.L.
1682*
COTMORE, S.F.
1581
COWEN, D.
1760*
COX, D.
1272
COX, K.O.
1576*
CRAIG, A.M.
1276
CRAIG, A.W.
1239
CRISS, W.E.
1637
CRITTENDEN, L.B.
1335
CRONKITE, E.P.
1612
CROUGHS, R.J.M.
1749*
CRUSE, J.M.
1544*, 1563*
CRYER, P.E.
1773*
CSUKA, O.
1254
CUATRECASAS, P.
1668*
CUDKOWICZ, G.
1480
CULLITON, B.J.
1227*
CUNNINGHAM, D.D.
1701*
CUNNINGHAM, M.P.
1309*
CURRIE, G.A.
1553*
DAHMUS, M.E.
1628
DALL'ACQUA, F.
1316*
DALY, J.J.
1439
DANAILA, L.
1753*
DANNA, K.J.
1406
DANOWSKI, T.S.
1741*
DARAI, G.
1398
DAVIES, P.
1461
DAVIS, D.
1461
DAVISON, S.C.
1273
DAY, N.
1238
DE GIORGI, L.S.
1709*
DE LANDAZURI, M.O.
1489
DE VRIES, J.E.
1776*
DEACON, J.
1750*
DEBOV, S.S.
1793*
DEBRUYNE, J.
1345
DECARVALHO, S.
1502
DECLOITRE, F.
1233
DECOTEAU, W.E.
1559*
DEFENDI, V.
1328
DEHARVEN, E.
1337
DENCKER, H.
1609
DENEKAMP, J.
1580
DEODHAR, S.
1685*
DEODHAR, S.D.
1539*, 1540*
DERMER, G.B.
1645
DESCHNER, E.E.
1270
DESHPANDE, N.
1651
DEVITA, V.T.
1297*
DHAR, R.
1352
DI LUZIO, N.R.
1510*
DI STEFANO, H.S.
1488
DIAZ FLORES, L.
1755*
DICKINSON, J.P.
1449
DIEHL, V.
1538*
DIGGS, C.H.
1640
DIMITROV, N.V.
1428*
DINAMANI, P.
1698*
DION, A.S.
1361
DIONISI, O.
1771*
DIPAOLO, J.A.
1331
DMOCHOWSKI, L.
1359
DOBASHI, Y.
1291*
DOE, R.P.
1712*
DOLMAN, A.
1749*
DOMBERNOWSKY, P.
1603*, 1739*, 1787*
DOMBOS, L.
1367
DOMINA, F.
1426*
DONNER, J.
1458
DORFMAN, H.D.
1769*
DORFMAN, R.F.
1225*
DORMANDY, T.L.
1283*
DOUGHERTY, R.M.
1488
DOUGLAS, S.D.
1653

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

DOWNHAM, D.Y.
1600*
DOYLE, A.E.
1704*
DRAKE, W.P.
1457, 1523*
DRASAR, B.S.
1594
DRYSDALE, J.W.
1682*
DUFF, R.
1403, 1639
DUFFY, P.E.
1760*
DUH, F.G.
1369
DULBECCO, R.
1390
DUNCAN, M.E.
1234
DUNWOODY, S.
1612
DURAND, R.E.
1732*
DWORKIN, M.
1235
EASLEY, G.W.
1597*
EAST, J.L.
1359
EASTHAM, W.N.
1749*
EBBESEN, P.
1370
EBOLI, M.L.
1771*
EDELMAN, G.M.
1453
EGGLETON, R.C.
1599*
ELAM, D.L.
1473
ELIAS, P.M.
1522*
ELLIS, B.M.
1360
ELLIS, D.A.
1422*
ELOVSKAYA, L.T.
1304
EMERSON, C.P., JR.
1796*
ENDO, H.
1253
ENGLAND, H.R.
1679*
ENZINGER, F.M.
1662*
EPSTEIN, M.A.
1349
EPSTEIN, W.L.
1344
ERBE, J.
1767*
ERHARD, P.
1412*, 1427*
ERIKSON, E.
1388, 1400
ERIKSON, R.L.
1388, 1400
ERNST, P.
1608
ESBER, E.
1531*
ESHUN-WILSON, K.
1744*
ESTERHAY, R.J., JR.
1519*
ESTES, J.D.
1493
ESTOLA, T.
1474
EVANS, A.E.
1661*
EVANS, V.J.
1733*
EVERT, C.F., JR.
1593
EYSSEN, G.
1226*
FAGUET, G.B.
1560*
FAN, T.-Y.
1284*
FARAS, A.J.
1411
FAYERS, P.M.
1779*
FECHNER, R.E.
1740*
FEDULLO, A.J.
1464
FEINGOLD, N.
1329
FEINSTEIN, A.R.
1582
FEINTUCH, T.A.
1314*
FEKETE, B.
1513*
FEL', V.YA.
1548*
FELDMAN, J.D.
1440, 1444
FELTER, R.
1743*
FELTKAMP-VROOM, T.M.
1749*
FENSELAU, A.
1762*
FENYOE, E.M.
1341
FERRONE, S.
1564*
FIELD, E.J.
1449
FIELDS, B.N.
1387
FIELDS, J.F.
1563*
FIGARD, P.H.
1463
FIGUEROA, W.G.
1299*
FILIPEK-WENDER, H.
1789*
FISCHER, R.G.
1356
FISCHINGER, P.J.
1378
FISH, C.A.
1713*
FISHER, D.R.
1729*
FISHER, E.R.
1741*
FISHER, W.D.
1296*
FITCH, F.W.
1573*
FLAX, H.
1623
FLEMING, E.N.
1585*
FLETCHER, G.H.
1673*
FLOYD, W.S.
1634
FOGEL, M.
1407
FOK, K.-F.
1268
FOLEY, V.L.
1730*
FONG, C.K.Y.
1365
FONT, R.L.
1662*, 1671*
FORET, J.E.
1687*
FORNI, G.
1441
FORTE, J.G.
1664*
FORTE, T.M.
1664*
FORTNER, J.G.
1589, 1596*
FOX, B.W.
1617
FOX, M.
1617, 1652
FRANSSILA, K.
1621
FRAUENDORFER, H.
1800*
FREEMAN, C.B.
1461
FREI, J.V.
1280
FREIREICH, E.J.
1456
FRENKEL, N.
1397
FRIEND, C.
1337
FRINDEL, E.
1774*
FRY, F.J.
1599*
FRY, M.
1667*
FUKS, Z.
1225*

FUKUYAMA, K.
1344
FURTH, J.
1511*
FURUSAWA E.
1421*
GABELMAN, N.
1346
GABRIELIDES, C.G.
1255
GAILANI, S.
1647
GALANTI, N.
1630
GALEOTTI, T.
1771*
GALESLOOT, J.
1477
GALLO, R.C.
1383
GALTON, D.A.G.
1736*
GANGOLLI, S.D.
1237
GANTT, R.
1733*
GARAPIN, A.C.
1411
GARCIA, J.H.
1586*
GARDNER, M.B.
1493
GARRET, R.
1655*
GARRY, V.F.
1526*
GASIC, G.J.
1630
GASIC, T.B.
1630
GASTON, M.R.
1476
GAUGAS, J.M.
1354
GEFFEN, L.B.
1704*
GELB, L.D.
1396
GELFMAN, N.A.
1582
GELLHORN, A.
1220*
GENNARO, A.R.
1269
GERALDES, A.
1504
GERGELY, P.
1513*
GERWIN, B.I.
1686*
GHATAK, N.R.
1759*
GHOSH, L.
1769*
GIMBO, A.
1426*
GIOVANELLA, B.C.
1688*
GLAVES, D.
1459
GLEICHMANN, E.
1205, 1512*
GLEICHMANN, H.
1205, 1512*
GOEKCEN, M.
1463
GOETZE, D.
1564*
GOLDBERG, A.I.
1476
GOLDFARB, S.
1646
GOLDIN, A.
1476, 1480
GOLDSTEIN, A.L.
1452
GOMARD, E.
1490
GOOD, R.A.
1481
GOODMAN, N.C.
1380
GORCZYNSKI, R.M.
1499
GORDON, P.R.
1674*
GOTHOSKAR, S.V.
1246
GRAB, D.J.
1249
GRAFFI, A.
1567*
GRAHNE, B.
1604*
GRANATIR, R.F.
1677*
GRANT, C.K.
1561*
GRASSO, P.
1237
GRAY, W.M.
1629
GREEN, M.
1404
GREENBLATT, M.
1289*
GREENE, G.W., JR.
1792*
GREENSTREET, R.L.
1764*
GRIFFIN, A.C.
1639
GRIFFITH, K.
1283*
GROLLE, P.F.
1355
GROSS, P.A.
1365
GRUENSTEIN, M.
1257
GRUNDER, G.
1341
GUCCION, J.G.
1662*
GUENTHER, H.
1783*
GULATI, S.
1392
GUNZ, F.W.
1676*, 1778*
GUPTE, N.V.
1785*, 1786*
GURTSEVICH, V.E.
1508*
GUTTERMAN, J.U.
1456
GUYDA, H.
1768*
HAAPALA, D.K.
1378
HAASE, A.T.
1424*
HACKENG, W.H.L.
1749*
HADDOW, A.
1206
HAEMMERLI, G.
1765*
HAGEMAN, PH.C.
1442
HAGMAR, B.
1462
HAIJE, W.G.
1460
HAKURA, A.
1353
HALL, E.J.
1300
HALL, R.R.
1552*
HALLIDAY, W.J.
1210
HAMMERBERG, O.
1363
HAMWOOD, S.M.
1735*
HANAFUSA, H.
1338
HANAFUSA, T.
1338
HANASAKI, N.
1650
HANNA, M.G., JR.
1377, 1482
HANSFORD, R.G.
1723*
HAPPLE, R.
1754*
HARADA, T.
1247
HARAN-GHERA, N.
1498
HARBERGER, J.H.
1308
HARDY, J.
1768*
HAREWOOD, K.R.
1374
HARLAN, W.L.
1691*
HARLEY, E.H.
1327
HARRIS, H.
1341

HARRIS, J.E.
1520*
HARRIS, R.
1461
HARRISON, E.G., JR.
1605*
HARRISS, E.B.
1577
HARTER, D.H.
1392
HARTLEY, J.W.
1336
HARTMANN, N.R.
1787*
HASHIMOTO, Y.
1483
HASSOUN, J.
1775*
HATANAKA, M.
1384
HAVELEC, L.
1800*
HAYASHI, K.
1340
HAYASHI, M.
1332, 1639
HAYWARD, J.L.
1651
HEHLMANN, R.
1410
HEIDELBERGER, K.P.
1705*
HEIMBURGER, R.F.
1599*
HEINZ, E.
1664*
HELSON, L.
1613
HEMPLING, H.G.
1766*
HENDERSON, B.E.
1493
HENDERSON, I.W.D.
1286*
HENDERSON, R.E.L.
1244
HENLE, G.
1219
HENLE, W.
1219
HENNEMANN, G.
1749*
HENRY, B.
1400
HENSON, D.
1306
HERBERMAN, R.B.
1489
HERMAN, M.M.
1730*
HERNDON, B.J.
1267
HERSCHMAN, H.R.
1554*
HERSEY, P.
1562*
HERSH, E.M.
1456

HERZENBERG, L.A.
1438
HESTER, R.B.
1563*
HEWITT, H.B.
1448
HIASA, Y.
1271
HIBBS, J.B., JR.
1446
HICKS, R.M.
1242
HIGASHI, K.
1650
HIGASHI, N.
1790*
HILF, R.
1660*
HILGERS, J.
1477
HIRANO, A.
1759*
HIRSCH, M.S.
1354, 1422*, 1436
HIRSCHHORN, K.
1346
HJELT, L.
1621
HOAGLAND, H.C.
1663*
HOBBS, J.R.
1623
HODES, M.E.
1373
HOEGG, U.
1277
HOEHN, H.
1754*
HOELLINGER, H.
1618
HOELZER, D.
1577
HOFER, K.G.
1684*
HOLLEB, A.I.
1601*
HOLLENBERG, M.D.
1668*
HOLLMANN, K.H.
1215
HOLMES, S.
1290*
HOLMSTROEM, T.
1604*
HOLT, P.G.
1295*
HOLTERMANN, O.A.
1551*
HONGO, T.
1501
HOOD, C.I.
1671*
HOOSON, J.
1237
HORSLEY, R.J.
1305
HORTON, B.J.
1583, 1646

HOUCHENS, D.P.
1476
HOZOC, M.
1429*, 1430*
HRGOVCIC, M.J.
1542*
HRUSHESKY, W.J.
1692*
HSIA, D.Y.Y.
1535*
HSIA, J.C.
1447
HSIUNG, G.D.
1365
HUDSON, L.
1416*
HUEBNER, R.J.
1263, 1375, 1493
HUMPHREY, J.B.
1326
HUNT, R.
1451
HUNTER, G.R.
1638
HUSSA, R.O.
1752*
HUTCHISON, G.B.
1209
HUVOS, A.G.
1674*
HYDE, E.F.
1757*
HYMAN, L.R.
1475
HYMAN, R.
1438
ICHIKAWA, T.
1791*
ILBERY, P.L.T.
1561*
INGRAM, L.O.
1296*
INMAN, F.P.
1443
INUI, N.
1266
IRVING, D.
1594
ISENBERG, I.
1276
ISHIKAWA, T.
1469
ISHIMARU, T.
1659*
IT, Y.
1659*
ITO, N.
1271
IWASUTIAK, R.
1280
IYPE, P.T.
1459
JACK, W.
1758*
JACKSON, J.P.
1510*
JAIN, H.K.
1230*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

JAKOBSEN, A.
1259
JAKCUBKOVA, J.
1458
JASMIN, C.
1366
JELLINCK, P.H.
1252
JELLINGER, K.
1781*
JEREB, B.
1538*
JERKOFSKY, M.
1408
JESSE, R.H.
1673*
JOHNSON, H.R.M.
1716*
JOHNSON, R.E.
1308
JOHNSON, T.
1630
JOHNSON, W.W.
1717*
JOLLEY, G.M.
1251
JONES, S.E.
1225*
JORGENSEN, O.
1370
JOSE, D.G.
1481
JOSEY, W.E.
1393
JOWETT, D.
1240
JUHLIN, I.
1538*
JUSTICE, P.
1535*
KADIN, M.E.
1225*, 1720*
KAHN, C.R.
1306
KAL, H.B.
1696*
KALB, A.J.
1574*
KALF, G.F.
1638
KALLEN, R.G.
1635
KALLMAN, R.F.
1580
KAMAHORA, J.
1353
KAMAMOTO, Y.
1271
KAMIJYO, Y.
1586*
KANAHARA, H.
1632
KAPLAN, A.R.
1602*
KAPLAN, H.S.
1225*
KAPPAS, A.
1298*
KARA, J.
1431*
KARIMINEJAD, M.H.
1711*
KARNJANAPRAKORN, C.
1578
KARON, H.
1789*
KASS, L.
1719*
KATO, S.
1469
KATO, T.
1247
KATOH, A.
1425*
KATSUNUMA, H.
1291*
KAUSHIK, S.P.
1784*
KAWAI, Y.
1425*
KAWAKAMI, T.G.
1420*
KAWAMURA, J.
1586*
KEAST, D.
1295*, 1576*
KELLEN, J.A.
1248
KENDALL, B.
1657*, 1728*
KENDE, M.
1476
KENNEDY, B.J.
1670*
KENNY, G.
1647, 1727*
KESSEL, D.
1750*
KETCHAM, A.S.
1306, 1569*
KHOO, S.K.
1465
KIANG, D.T.
1670*
KIKKAWA, J.-I.
1791*
KIKUCHI, K.
1791*
KILLMANN, S.-A.
1608
KIM, H.
1225*
KIMURA, G.
1390
KINGDON, H.S.
1616
KINO, I.
1683*
KIRK, J.
1629
KIRSCH, W.M.
1622
KITHIER, K.
1525*
KLEIHUES, P.
1272
KLEIN, E.
1339, 1341, 1551*
KLEIN, G.
1341, 1367, 1381, 1543*
KLINE, I.
1473
KOCH, J.
1324
KOHN, H.I.
1439
KOIZUMI, A.
1291*
KOLESNICHENKO, T.S.
1232
KOMMINENI, C.
1289*
KONDRAT'YEVA, A.F.
1507*
KONIG, E.
1653
KOPELOVICH, L.
1721*
KOPF, A.W.
1587*
KOPILASHVILI, G.S.
1509*
KORB, F.
1311*
KOSKY, K.
1722*
KOSS, L.G.
1584
KRAIN, L.S.
1591
KROES, R.
1484
KRUEGER, R.G.
1722*
KRUG, U.
1668*
KRUGLYAKOVA, K.YE.
1317*
KRUSH, A.J.
1602*, 1691*
KRYSTAL, G.
1680*
KRYUKOVA, I.N.
1557*
KUDO, H.
1501
KUDO, T.
1791*
KUFE, D.
1410
KULIN, H.E.
1735*
KUPCHIK, H.Z.
1565*
KURAGANO, T.
1650
KWA, H.G.
1442
KYLE, R.A.
1605*
LACH, H.
1318*
LAERUM, O.D.
1241

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

LAMON, E.W.
1339
LAMPEN, N.
1337
LAMPKIN, B.C.
1593
LANCE, E.M.
1451
LARSON, D.L.
1374
LASKOV, R.
1533*
LASZLO, A.
1305
LATNER, A.L.
1633
LATTES, R.
1683*, 1699*
LAUDER, I.
1518*
LAURENCE, D.J.R.
1552*
LEBOWITZ, P.
1352
LECLERC, J.C.
1490
LEE, S.-C.
1628
LEEDHAM, P.W.
1679*
LEFEBVRE, N.
1345
LEGENDRE, S.M.
1457
LEHNINGER, A.L.
1723*
LENGEROVA, A.
1204
LEONARD, E.J.
1445
LEONARD, N.J.
1244
LERNER, M.P.
1554*
LEUNG, B.S.
1758*
LEVAN, G.
1243
LEVENBUK, I.S.
1433*
LEVI, J.A.
1676*, 1778*
LEVINE, G.D.
1715*
LEVINSON, W.
1424*
LEVINSON, W.E.
1411
LEVY, J.P.
1329, 1490
LEVY, N.L.
1362
LEWIS, A.M., JR.
1352
LEWIS, G.K.
1544*, 1563*
LEWIS, M.L.
1359
LEYTEN, R.
1347
LICATA, A.
1426*
LIE, J.T.
1465
LIEBER, M.M.
1322
LIEN, W.M.
1746*
LIJINSKY, W.
1289*
LIKHACHEV, YU.P.
1304
LILLY, F.
1326
LIMACHER, H.
1285*
LIN, J.-K.
1268
LINDSAY, D.B.
1689*
LINELL, F.
1795*
LINIAL, M.
1405
LIPKIN, M.
1270
LIPMAN, M.B.
1321
LIPPMAN, M.M.
1473
LISIEWICZ, J.
1788*
LISS, R.H.
1689*
LITTLE, J.R.
1447
LIU, P.I.
1659*
LIVINGSTON, A.M.
1658*
LIVINGSTON, D.M.
1399
LIVINGSTON, V.W.-C.
1658*
LJUNGBERG, O.
1231*, 1751*
LOBUGLIO, A.F.
1560*
LODMELL, D.L.
1340
LOEB, L.A.
1614
LONG, J.C.
1491
LONGFIELD, A.K.
1685*
LONGSTAFF, E.
1633
LOONEY, W.B.
1619
LOUIS, W.L.
1704*
LOWE, C.R.
1697*
LOWRY, D.R.
1336
LUECHTRATH, H.
1606*
LUECKE, D.H.
1356
LUNDIN, F.E., JR.
1588
LUSTIG, V.
1248
LYARSKII, P.P.
1304
LYNCH, H.T.
1602*, 1691*
MABUCHI, K.
1589, 1596*
MACH, O.
1431*
MACKAY, I.R.
1465
MACKINNON, A.E.
1287*
MACLENNAN, I.C.M.
1562*
MACMAHON, B.
1697*, 1699*
MACPHERSON, I.A.
1368
MAEKAWA, K.
1516*
MAGEE, P.N.
1261, 1272
MAGNER, D.
1226*
MAHER, V.M.
1250
MAIZEL, J.V., JR.
1387
MAKITA, A.
1382
MAKIURA, S.
1271
MALAISE, E.P.
1366
MALKIEWICZ-WASOWICZ, B.
1788*
MALONEY, M.A.
1312*
MALYGIN, A.M.
1548*
MANCINI, L.O.
1376
MANOR, H.
1407
MARCIANI, S.
1316*
MARCUS, P.B.
1656*
MARDINEY, M.R., JR.
1457, 1523*
MARGULIS, A.R.
1312*
MARK, J.
1243, 1609
MARKESBERY, W.R.
1760*
MARKOE, A.M.
1412*, 1419*
MARONCELLI, P.
1780*

MARTIN, J.-P.
   1505*
MARTIN, M.
   1233
MARTIN, M.A.
   1396
MARTIN, W.J.
   1531*
MARTZ, E.
   1492
MARUCCI, A.A.
   1488
MARUCHI, N.
   1589, 1596*
MARUTA, H.
   1467
MARX, J.L.
   1224*
MARZOEV, A.I.
   1310*
MASLOW, D.E.
   1782*
MASON, W.S.
   1401, 1405
MASUDA, T.
   1438
MATHIAS, A.P.
   1272
MATSUHISA, T.
   1650
MATTINGLY, R.F.
   1752*
MAUER, A.M.
   1593
MAVLIGIT, G.
   1456
MAYO, A.A.
   1619
MAYYASI, S.A.
   1374
MCCAIN, B.
   1432*
MCCREDIE, K.B.
   1456
MCDONALD, A.D.
   1226*
MCDONALD, J.J.
   1244
MCGREGOR, D.H.
   1659*
MCINTIRE, K.R.
   1519*
MCKENZIE, I.F.C.
   1556*
MCWILLIAMS, N.B.
   1593
MEDAWAR, P.B.
   1451
MEENAGHAN, M.A.
   1792*
MEHRISHI, J.N.
   1550*
MEIER, H.
   1375
MELCHERS, F.
   1527*
MENDOZA, C.B., JR.
   1597*
MERANZE, D.R.
   1257
MEREKALOVA, Z.I.
   1415*
MERINO, F.
   1546*
METCALF, D.
   1724*, 1725*
MEUNARGIYA, V.V.
   1509*
MEUNIER, M.
   1245
MILGROM, F.
   1546*
MILLER, E.C.
   1235, 1262, 1274
MILLER, G.
   1343
MILLER, J.A.
   1235, 1262, 1274
MILLER, J.F.A.P.
   1468
MILLER, K.
   1418*
MILLER, R.G.
   1499
MINOWADA, J.
   1694*
MITCHELL, H.
   1571*
MITELMAN, F.
   1243
MITTELMAN, F.
   1609
MIYAKAWA, M.
   1247
MIZUNO, D.
   1467
MOMII, A.
   1516*
MONACO, A.P.
   1422*
MONACO, F.
   1695*
MONTES DE OCA, F.
   1733*
MOORE, D.H.
   1361
MOORE, M.
   1281*
MOORE, M.A.S.
   1724*, 1725*
MOQUIN, R.B.
   1677*
MORGAN, A.C.
   1688*
MORGAN, R.K.B.
   1600*
MORIN, O.
   1506*
MORLEY, C.G.D.
   1616
MORRIS, H.P.
   1619, 1636, 1637, 1638,
   1642, 1721*
MORRIS, N.R.
   1665*
MORRISON, A.S.
   1697*, 1699*
MORROW, J.Y.
   1619
MOTT, G.E.
   1646
MOTTA, J.V.
   1335
MOULE, Y.
   1236
MOUSSET, S.
   1358
MCY, P.
   1511*
MOZES, E.
   1498
MUNK, K.
   1398
MUNOZ, N.
   1409
MURAMATSU, M.
   1266
MURPHY, G.
   1647
MURPHY, G.P.
   1692*
MURPHY, S.
   1630
MURPHY, W.M.
   1539*, 1540*
MURTHY, A.S.K.
   1279
MYERS, J.W.
   1747*
NACHKOV, D.
   1521*
NAHMIAS, A.J.
   1393
NAIB, Z.M.
   1393
NAITO, M.
   1469
NAKAYAMA, I.
   1731*
NAKAYASU, M.
   1763*
NARAYAN, O.
   1406
NASTAC, E.
   1429*, 1430*
NATHANS, D.
   1406, 1417*
NATHANSON, L.
   1545*
NECHELES, T.
   1545*
NEIMAN, P.E.
   1391
NELSO   K.
   1437
NEMOTO, N.
   1266
NERI, G.
   1455
NEVILLE, A.M.
   1552*
NEWBERNE, P.M.
   1267
NEWTON, A.A.

1487
NEWTON, K.A.
1623
NGUYEN-HOANG-NAM,
1618
NICHOLS, J.L.
1325
NICHOLSON, G.L.
1478
NICKELS, J.
1621
NICKERSON, P.A.
1731*
NIGAM, V.N.
1207
NIKONOVA, T.V.
1232
NISHIYAMA, R.H.
1705*
NIWA, A.
1340
NIXON, D.W.
1706*
NOBREGA, F.T.
1605*
NOHARA, M.
1763*
NORBACK, D.H.
1240
NORRBY, K.
1462
NORRYD, C.
1609
NOTKINS, A.L.
1340
NUSSBAUM, A.
1647
O'CONNOR, P.J.
1239
O'KEEFE, D.D.
1690*
O'NEILL, P.J.
1463
OBUKH, I.B.
1557*
ODAKA, T.
1330
OEHLERT, W.
1202
OHNISHI, T.
1648
OIKAWA, A.
1763*
OKINO, T.
1291*
OKUNEWICK, J.P.
1412*, 1419*, 1427*
OLIVA, H.
1755*
OLIVER, R.A.M.
1532*
OLIVERIO, V.T.
1297*
OLSON, M.O.J.
1615
OMATA, S.
1648
ONO, M.
1467
ONUIGBO, W.I.B.
1203
ORMEROD, M.G.
1251
ORNSTEIN, L.
1522*
ORRICK, L.R.
1615
OSAFUNE, S.
1469
OSSOWSKI, L.
1333, 1342
OWEN, J.T.
1470
OWEN, L.N.
1228*
OWENS, A.H., JR.
1640
PACE, N.R.
1400
PAN, S.F.
1757*
PAPA, S.
1771*
PAPAS, T.A.
1421*
PARADIES, G.
1771*
PARKER, J.E.
1517*
PARKER, K.
1305
PARKS, R.E., JR.
1797*
PATCHEFSKY, A.
1584
PATT, H.M.
1312*
PATTERSON, D.S.P.
1218
PATTILLO, R.A.
1752*
PAULING, L.
1213
PAYNE, L.N.
1416*
PECHT, I.
1574*
PELLEGRINO, M.A.
1564*
PENA, C.E.
1743*
PENN, I.
1547*
PERDUE, J.F.
1418*
PEREZ-BERCOFF, R.
1524*
PEREZ, C.A.
1673*
PERIYANAYAGAM, W.J.
1784*
PERLIN, E.
1677*
PETER, H.H.
1440
PETER, H.-H.
1444
PETERS, R.L.
1726*
PETERS, W.P.
1410
PETERSEN, H.S.
1278
PETKOVIC, I.
1737*
PETRANYI, G.
1513*
PHILLIPS, B.
1566*
PHILLIPS, E.L.
1412*
PHILLIPS, R.A.
1499
PICHAT, L.
1618
PICKART, L.
1625
PIDOUX, M.
1506*
PIECZYNSKI, W.J.
1331
PIERRE, R.V.
1663*
PIKOVSKI, M.A.
1471
PILCH, B.Z.
1306
PILLAI, W.J.
1784*
PIPER, P.W.
1700*
PIPERNO, J.R.
1635
PISANO, J.C.
1510*
PITOT, H.C.
1646
PLAGEMANN, P.G.W.
1767*
PLATA, E.J.
1686*
PLATA, F.
1490
POCHET, R.
1345
POGOSYANTS, E.E.
1414*
POLANO, M.K.
1745*
POLGAR, P.
1796*
POLLACK, S.
1437
PORTER, E.H.
1448
POULIK, M.D.
1525*, 1572*
PRESSMAN, P.I.
1672*
PRICE, M.R.
1466
PRICE, P.M.
1346
PRICE, R.A.

1717*
PROCTOR, J.W.
1528*
PROFFITT, M.R.
1436
QUIGLEY, J.P.
1333, 1342
RAAM, S.V.
1443
RABINOWITZ, Y.
1535*
RABINOWITZ, Z.
1265, 1332
RABSON, A.S.
1611
RABSTEIN, L.S.
1726*
RADSAK, K.
1423*
RAESAENEN, J.A.
1549*
RAMA RAO, G.V.S.V.
1486
RAMENSKY, E.V.
1777*
RANADIVE, K.J.
1246, 1786*
RAPP, F.
1212, 1403, 1408, 1639
RAPPAPORT, H.
1718*
RASZKOWSKI, R.
1299*
RAWLINSON, D.G.
1693*
RAY, P.K.
1644
REEDER, D.D.
1627
REEDMAN, B.M.
1381
REES, R.J.W.
1354
REHACEK, J.
1356
REICH, E.
1333, 1342, 1413*
REIF, A.E.
1713*
REINISCH, C.L.
1351
REISFELD, R.A.
1564*
REITER, E.O.
1735*
REJTHAR, A.
1541*
RETHY, A.
1761*
REUTER, M.A.
1250
RHEINS, M.S.
1570*
RHIM, J.S.
1263, 1369
RICE, J.M.
1264
RICHARDS, F.F.
1321
RICHART, R.M.
1683*
RIFKIN, D.B.
1333, 1342, 1413*
RITA, G.
1524*
RIVAS, M.C.
1755*
ROACH, W.A.
1292*
ROBBINS, J.
1695*
ROBBINS, J.H.
1301, 1522*
ROBERT, F.
1768*
ROBERTS, J.H.
1654*
ROBERTS, M.M.
1450
ROBERTSON, D.D.
1686*
ROBIN, M.S.
1404
ROBINS, R.A.
1466
ROBINSON, C.M.
1713*
ROCKERBIE, R.A.
1517*
RODIGHIERO, C.
1316*
ROEDER, C.
1765*
ROGERS, A.E.
1267
ROHRSCHNEIDER, L.R.
1214
ROIZMAN, B.
1397
RONGEY, R.W.
1493
ROPARTZ, C.
1505*
ROSAS-URIBE, A.
1718*
ROSE, N.R.
1610
ROSE, S.
1568*
ROSEMOND, G.P.
1269
ROSEN, S.W.
1611
ROSENBERG, S.A.
1225*
ROSHCHUPKIN, D.I.
1310*
ROSS, L.J.N.
1487
ROSS, W.C.J.
1618
ROTHSCHILD, H.
1394
ROTKIN, I.D.
1217
ROUNDS, D.E.
1624
ROWE, W.P.
1326, 1336
RUBIN, B.
1529*
RUBINSTEIN, L.J.
1730*
RUDDLE, N.H.
1321
RUDDY, S.
1558*
RUDENSTAM, C.M.
1528*
RUEMKE, P.
1776*
RUKOVENA, E.
1685*
RUOSLAHTI, E.
1474
RUPRECHT, R.M.
1380
RUSH, R.A.
1704*
RUTENBURG, A.M.
1796*
SABINE, J.R.
1583, 1721*
SACHS, H.
1799*
SACHS, L.
1265, 1332, 1407
SACK, G.H., JR.
1406
SACKSTEDER, M.R.
1360
SADOFF, L.
1626
SAFINA, G.V.
1777*
SAINEROVA, H.
1458
SAITO, T.
1791*
SAKAMOTO, Y.
1650
SAKSELA, E.
1604*
SALIH, H.
1623
SALYER, D.
1714*
SALZBERG, S.
1404
SAMBROOK, J.
1208
SAN, R.H.C.
1260
SANDELIN, K.
1474
SANDER, J.
1259
SANDER, S.
1259
SANDHAUS, L.
1421*
SANEL, F.T.
1323
SANFORD, B.H.

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

1439
SARAVIS, C.A.
1565*
SARDESAI, S.
1525*
SARKAR, K.
1756*
SARKAR, N.H.
1361
SARKAR, S.
1438
SATO, K.
1642
SATO, T.
1337
SCHACHNER, M.
1472
SCHALCH, D.S.
1511*
SCHANTZ, A.
1707*
SCHERBAKOV, V.YA.
1555*
SCHEVING, L.E.
1681*
SCHIDLOVSKY, G.
1374
SCHIFFER, D.
1748*
SCHINITSKY, M.R.
1475
SCHLESINGER, R.M.
1713*
SCHNITZER, B.
1705*, 1719*
SCHOLAR, E.M.
1797*
SCHOLEFIELD, P.G.
1680*
SCHOLM, J.
1392
SCHOPMAN, W.
1749*
SCHULTZ, R.M.
1486
SCHUMACHER, H.R.
1729*
SCHUR, P.H.
1558*
SCHWARTZ, J.P.
1665*
SCHWARTZ, R.D.
1360
SCOLNICK, E.M.
1334
SCRIBNER, J.D.
1264
SCULLY, R.E.
1710*, 1711*
SEAL, U.S.
1712*
SEEGER, R.C.
1470
SEGALOFF, A.
1302
SEILER, J.P.
1285*
SEKIKAWA, M.
1543*
SELL, S.
1479
SEMENKOV, V.F.
1555*
SENINA, E.T.
1414*
SERBAN, G.
1430*
SEREBRYAKOV, V.SH.
1509*
SHABAD, L.M.
1232
SHAMBERGER, R.J.
1685*
SHAMOTO, M.
1425*
SHANKAR NARAKAN, K.
1624
SHAPAS, B.G.
1275
SHAPIRO, H.M.
1519*
SHAPIRO, S.
1464
SHARMA, O.K.
1669*
SHARP, E.A.
1691*
SHEARER, G.M.
1498
SHEIN, H.M.
1620
SHEININ, R.
1357
SHIGEMATSU, T.
1359
SHIMOJO, H.
1382
SHIMOKAWA, K.
1702*
SHIRES, T.K.
1262
SHOHAM, M.
1574*
SHOHAT, B.
1494
SHOPE, T.
1343
SILAEVA, S.A.
1793*
SILVERBERG, E.
1601*
SILVERBERG, S.G.
1709*
SILVERMAN, E.
1545*
SILVERNAIL, P.
1647
SIME, G.C.
1553*
SIMMONS, A.V.
1779*
SIMMONS, R.L.
1644
SIMON, E.
1533*
SIMS, P.
1288*
SINCLAIR, I.P.
1307
SINHA, A.A.
1712*
SINKOVICS, J.G.
1542*
SIRMAKECHIAN, T.
1255
SKINNER, D.G.
1320*
SKIPPER, H.E.
1221*
SKURZAK, H.M.
1339
SLAGA, T.J.
1264, 1275
SLUNGAARD, A.
1536*
SMADJA-JOFFE, F.
1366
SMALL, E.W.
1276
SMITH, B.
1742*
SMITH, B.J.
1328
SMITH, C.A.
1641
SMITH, D.F.
1455
SMITH, G.
1252
SMITH, H.
1703*
SMITH, H.J.
1463
SMITH, J.L.
1534*
SMITH, K.P.
1636
SMITH, M.J.
1536*
SMITH, R.T.
1386
SMULSON, M.
1654*
SNELL, G.D.
1556*
SNODGRASS, M.J.
1482
SOBAJIMA, Y.
1501
SOBIS, H.
1347, 1348
SOEIRO, R.
1387
SOGA, J.
1632
SONENBERG, M.
1249
SOO, S.F.
1439
SORGER, K.
1727*
SPAHN, G.J.
1726*
SPEDEN, J.B.

1676*, 1778*
SPENCE, I.M.
1372
SPIEGELMAN, S.
1380, 1392, 1410
SPIERS, A.S.D.
1779*
SPONZO, R.W.
1297*
SPRECHER-GOLDBERGER, S.
1345
SPRINGGATE, C.F.
1614
SREBRO, Z.
1318*
SRIVASTAVA, B.I.S.
1694*
SRODES, C.H.
1757*
STARK, P.
1654*
STARZL, T.E.
1547*
STEHLIN, J.S.
1688*
STEINER, G.C.
1769*
STELLMAN, J.M.
1293*
STEPHENSON, J.R.
1379
STEVENS, L.C.
1222*
STEVENSON, A.
1450
STEWART, B.W.
1261
STEWART, M.L.
1387
STEWART, T.H.M.
1520*
STICH, H.F.
1260, 1363
STICH, W.
1260
STILLMAN, M.A.
1587*
STOIAN, M.
1430*
STONEHILL, E.H.
1270
STORY, M.T.
1752*
STRAEULI, P.
1765*
STRONG, E.W.
1674*
STRONG, J.A.
1282*
STRUVE, M.YE.
1798*
STULTING, R.D.
1503
SUBAK-SHARPE, J.H.
1395
SUGAR, J.
1254
SUGAWARA, M.
1501
SUGIHARA, S.
1271
SUKONTHAMAN, Y.
1269
SUMMERS, D.F.
1387
SUNDER-PLASSMANN, M.
1781*
SUTHERLAND, J.C.
1519*
SUTHERLAND, R.M.
1732*
SWARTZENDRUBER, D.C.
1684*
SWINTON, N.W.
1690*
SWISLOCKI, N.I.
1249
SYMEONIDIS, A.
1255
SYMON, L.
1728*
SZABO, G.
1513*
SZEGEDI, G.
1513*
SZEKELY, I.E.
1729*
SZENDE, B.
1617
TACHIBANA, Y.
1291*
TAKAHASHI, K.
1253
TAKAYAMA, S.
1266
TALERMAN, A.
1460
TALVALKAR, G.V.
1786*
TANI, E.
1790*
TANNENBAUM, S.R.
1284*
TASHJIAN, A.H., JR.
1611
TAUB, R.N.
1575*
TAYLOR, B.A.
1375
TAYLOR, C.W.
1678*
TAYLOR, H.B.
1699*
TAYLOR, J.F.
1514*
TAZAWA, K.
1632
TEICH, N.
1336
TEMPLE, R.
1666*
TENSER, R.B.
1365
TERAYAMA, H.
1648
TERRACINI, B.
1238
TERRAGNO, N.A.
1752*
TESTA, M.C.
1238
TEZABWALA, B.U.
1246
THALER, M.M.
1625
THIRY, L.
1345
THOMAS, C.B.
1764*
THOMAS, D.B.
1566*
THOMAS, W.
1295*
THOMPSON, J.C.
1627
THOMSON, D.M.P.
1497
THOMSCN, S.
1578
THRASH, C.R.
1701*
THURNHERR, N.
1270
TING, R.C.Y.
1383
TOBIA, A.
1333, 1342
TODARO, G.J.
1322, 1399
TOGA, M.
1775*
TOMA, V.
1430*
TOMASI, T.B., JR.
1559*
TOMASI, V.
1761*
TOMPKINS, W.A.F.
1486
TORLINSKI, L.
1789*
TOTH, J.
1254
TRANBERG, K.-G.
1609
TREVISANI, A.
1761*
TREVISANI, E.T.
1308
TRIPIER, M.F.
1775*
TROY, F.A.
1420*
TSANKCVA, K.
1521*
TSUDA, K.
1291*
TSUKAGOSHI, S.
1483
TURNER, G.A.
1633
TYSON, S.
1626
TYTKO, S.A.

1685*
UEDA, G.
1511*
UNDERWOOD, J.C.E.
1770*
UNGARO, P.C.
1523*
UNKELESS, J.C.
1333, 1342
URSU, E.
1429*
USUBUCHI, I.
1501
UTSUMI, K.R.
1543*
UYEDA, C.K.
1681*
UYEKI, E.M.
1526*
UZUNOV, P.
1620
VAHERI, A.
1474
VAITKEVICIUS, V.K.
1525*
VALLE, A.
1755*
VAN BENTHEM, M.
1776*
VAN BUSKIRK, J.J.
1622
VAN DE MAELE, B.
1347
VAN DER WALT, J.J.
1656*
VAN NOORD, M.J.
1355
VAN ROSSUM, G.D.V.
1636
VAN ROY, M.
1506*
VAN WOERT, M.H.
1311*
VANDEPUTTE, M.
1347, 1348
VARDOSANIDZE, E.SH.
1509*
VARET, B.
1329
VARGHESE, A.J.
1315*
VARIAKOJIS, D.
1718*
VARMUS, H.E.
1411
VASCONCELOS-COSTA, J.
1504
VATHANOPHAS, V.
1269
VAUPEL, P.
1783*
VAWTER, G.F.
1279
VEDALDI, D.
1316*
VEIDENHEIMER, M.C.
1690*
VELAYUDHAN, V.C.
1784*
VENDITTI, J.M.
1473
VERMEIL, C.
1506*
VERNON, M.L.
1369
VERSTRAETEN, A.A.
1442
VESELY, P.
1434*
VIADENA, E.
1590
VIDRINE, J.G.
1374
VIJAY, I.K.
1420*
VIKLICKY, V.
1458
VIKTOROVA, L.N.
1777*
VILLANUEVA, R.
1269
VINARCHUK, M.P.
1631
VINCENT, P.C.
1676*, 1778*
VINCZE, A.
1244
VINOGRAD, J.
1641
VIRAGOS, G.
1280
VISFELDT, J.
1370
VLADIMIROV, YU.A.
1310*
VLADUTIU, G.D.
1610
VOGEL, H.H., JR.
1303
VOGT, P.K.
1401
VON KLEIST, S.
1537*
VON ZEYNEK, E.R.
1592
WADDELL, M.
1325
WAGENBICHLER, P.
1800*
WAGONER, J.K.
1588
WAKEFIELD, J.ST.J.
1242
WAKUI, A.
1791*
WALBORG, E.F., JR.
1455
WALDRON, R.L.
1727*
WALKER, I.R.
1738*
WALKER, J.S.
1461
WALLACE, D.M.
1552*
WALLACE, I.W.J.
1450
WALLIS, K.
1762*
WALTON, I.G.
1282*
WANG, D.Y.
1651
WANG, J.L.
1453
WARNER, D.
1418*
WARNER, N.L.
1465
WARREN, J.
1360
WASON, W.M.
1573*
WASSILEWA, L.
1521*
WATNE, A.L.
1597*
WATSON, E.S.
1544*, 1563*
WAXMAN, S.
1346
WEAVER, D.K.
1705*
WEBSTER, I.
1258
WECHSLER, J.
1329
WEDDERBURN, N.
1530*
WEIGEL, W.O.
1440
WEIGLE, W.O.
1444
WEINER, L.P.
1406
WEINHOUSE, S.
1642
WEINTRAUB, B.D.
1611
WEIR, J.A.
1708*
WEISBURGER, J.H.
1484
WEISS, B.
1620
WEISS, R.A.
1401
WEISS, W.
1299*
WEISSBACH, A.
1667*
WEISSMAN, S.M.
1352
WEKSLER, M.E.
1500
WELLS, S.A., JR.
1569*
WELVAART, K.
1745*
WENDEL, E.J.
1335
WERCHAU, H.
1402
WERTHAMER, S.

1319*
WHELDON, T.E.
1629
WHITCUTT, J.M.
1364, 1371
WHITE, J.S.
1327
WHITTEN, H.D.
1544*, 1563*
WIENER, F.
1341
WIERNIK, P.H.
1519*
WIGGLESWORTH, N.M.
1328
WIGZEIL, H.
1529*
WIGZELL, H.
1339
WILBUR, J.R.
1542*
WILEY, A.L., JR.
1303
WILHOITE, R.
1309*
WILLIAMS, D.E.
1281*
WILLIAMS, E.H.
1435*
WILLIAMS, G.M.
1484
WILLIAMS, J.F.
1385
WILLIAMS, J.G.
1368
WILLIAMS, L.J., JR.
1688*
WILLIAMS, N.
1724*, 1725*
WILLIS, C.E.
1685*
WILLS, E.D.
1273
WINKLEY, J.
1626
WISLOCKI, P.G.
1274
WOLF, P.H.
1698*
WOLFF, J.
1666*
WOLFF, J.S., III
1374
WOLFF, M.
1579
WONG, P.
1535*
WOOD, D.C.
1758*
WOOD, M.L.
1422*
WOTKE, J.
1541*
WRIGHT, E.B.
1283*
WRIGHT, J.T.
1294*
WRIGHT, P.W.
1489
WU, A.M.
1383
WU, C.
1607
WUERTHNER, K.
1799*
WUNDERLICH, J.R.
1531*
WYNDER, E.L.
1589, 1596*
YAHARA, I.
1453
YAKOVLEVA, L.S.
1415*
YAMAMOTO, K.
1648
YAMAMOTO, T.
1265, 1332
YATES, V.J.
1376
YEOMAN, L.C.
1678*
YESNER, R.
1582
YEVREINOVA, T.N.
1798*
YIM, S.O.
1688*
YOSHIDA, O.
1247
YOSHIDA, T.O.
1543*
YOSHIKURA, H.
1350
YUASA, S.
1697*, 1699*
ZAIN, B.S.
1352
ZAKI, S.A.
1393
ZALENSKI, D.
1660*
ZAMCHECK, N.
1565*
ZANELLI, J.
1451
ZATZ, M.M.
1452
ZAVON, M.R.
1277
ZEDECK, M.S.
1249
ZHIZHINA, G.I.
1317*
ZIMMERMAN, H.M.
1759*
ZIMMERMAN, L.E.
1662*, 1671*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

- 2-ACETYLAMINOFLUORENE
  - HEPATOMA
    - CHOLESTEROL SYNTHESIS, PRECANCEROUS LIVER, RAT, 1646
  - URINARY BLADDER TUMOR
    - BETA-GLUCURONIDASE INHIBITOR, RAT, 1247

- ACETYLCHOLINE
  - REGENERATION, LIMB
    - NEWT, 1687*

- ACID PHOSPHATASE
  - ISOZYMES
    - PROSTATIC CARCINOMA PATIENTS, 1713*

- ACTINOMYCIN D
  - ALKALINE PHOSPHATASE ACTIVITY
    - TRANSPLANTED TUMOR CELLS, MOUSE, 1772*
  - DIMETHYLNITROSAMINE
    - TUMOR INDUCTION, RAT, 1261
  - RNA SYNTHESIS
    - FOWL PLAQUE VIRUS REPLICATION, HAMSTER, 1368
    - POLYOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 1368
    - ROUS SARCOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 1368
  - SUCCINATE DEHYDROGENASE ACTIVITY
    - TRANSPLANTED TUMOR CELLS, MOUSE, 1772*

- ADENOAMELOBLASTOMA
  - AMYLOID CONTENT
    - ULTRASTRUCTURE, HUMAN, 1792*

- ADENOCARCINOMA
  - COLON
    - 1,2-DIMETHYLHYDRAZINE, MOUSE, 1270
  - COLON, SIGMOID
    - URETEROSIGMOIDOSTOMY, CASE REPORTS, 1746*, 1747*
  - COLON, TRANSVERSE
    - RADIATION-INDUCED, CASE REPORT, 1309*
  - ILEOCYSTOPLASTY
    - CASE REPORT, 1679*
  - MAMMARY GLAND
    - C-TYPE VIRUS PARTICLES, RAT, 1426*
    - LIPOGENESIS, HIGH CARBOHYDRATE DIET, RAT, 1660*
  - PANCREAS
    - EPIDEMIOLOGY, NEW YORK CITY, 1596*
    - INCIDENCE, U.S.A., 1589
    - MICROSPORIDIOSIS, CASE REPORT, 1656*
  - RING-SHAPED PARTICLES
    - ULTRASTRUCTURE, HUMAN CULTURED CELLS, 1624
  - UTERINE CERVIX
    - CYTOLOGIC, HISTOLOGIC, AND CYTOPHOTOMETRIC STUDIES, 1799*

- ADENOCYSTIC CARCINOMA
  - SALIVARY GLAND
    - NUCLEAR DNA CONTENT, HUMAN, 1683*

- ADENOMA
  - ADRENAL GLAND
    - BLACK PIGMENTED, CASE REPORTS, 1655*
  - LUNG
    - LONG-TERM EFFECT OF DDT, MOUSE, 1232, 1238

- ADENOMA, ADRENOCORTICAL
  - VIRILIZATION
    - ULTRASTRUCTURE, CASE REPORT, 1741*

- ADENOSINE DIPHOSPHORIBOSYLATION
  - DNA SYNTHESIS
    - NUCLEAR PROTEINS, HELA CELLS, 1654*

- ADENOSINE TRIPHOSPHATASE
  - ACTIVITY
    - LYMPHOCYTIC LEUKEMIA, PLATELET KINETICS, MOUSE, 1428*

- ADENYLATE CYCLASE
  - ACTIVITY, SOLUBLE AND MEMBRANE BOUND
    - YOSHIDA ASCITES HEPATOMA CELLS, 1761*

- ADENYLATE KINASE
  - INDUCTION
    - HORMONALLY TREATED RATS, 1637

- ADNEXAL TUMOR, FEMALE
  - PROBABLE WOLFFIAN ORIGIN
    - CLINICOPATHOLOGICAL STUDY, 1711*

- ADRENAL GLAND
  - ADENOMA
    - BLACK PIGMENTED, CASE REPORTS, 1655*

- AFLATOXIN B1
  - ALPHA-FETOPROTEIN
    - SERUM, RAT, 1484
  - METABOLISM
    - TOXICITY, BIRD, MAMMAL, REVIEW, 1218
  - SUBRIBOSOMAL PARTICLE FORMATION
    - LIVER, RAT, 1236

- AGE
  - AZO-DYE CARCINOGENESIS
    - LIVER, RAT, 1233
  - P-DIMETHYLAMINOAZOBENZENE METABOLISM
    - LIVER, RAT, 1233

- AGGLUTINATION
  - CONCANAVALIN A
    - PURIFICATION OF ONCORNA VIRUSES, 1387

- ALKALINE PHOSPHATASE
  - ACTIVITY
    - ACTINOMYCIN D, TRANSPLANTED TUMOR CELLS, MOUSE, 1772*
    - HODGKIN'S DISEASE, CLINICAL STUDY, 1779*
    - MALIGNANT LYMPHOMA, CLINICAL STUDY, 1779*
    - OSTEOSARCOMA, MOUSE, 1702*

        PUROMYCIN, TRANSPLANTED TUMOR CELLS, MOUSE, 1772*
    LEUKEMIA, CHRONIC MYELOCYTIC
        HUMAN, 1612

ALKYLATING AGENT
    DNA SYNTHESIS
        SKIN, MOUSE, 1275
    RNA SYNTHESIS
        SKIN, MOUSE, 1275

ALLERGY
    CANCER RISK
        CLINICAL STUDY, 1464

AMELOBLASTOMA
    AMYLOID CONTENT
        ULTRASTRUCTURE, HUMAN, 1792*

AMINO ACID
    51CR AND 14C-LABELED
        INCORPORATION AND RELEASE IN EHRLICH ASCITES TUMOR CELLS, 1782*

AMYLOID
    CONTENT
        ODONTOGENIC TUMORS, ULTRASTRUCTURE, HUMAN, 1792*

ANDROGEN
    MAMMARY TUMOR
        DEPENDENCE, HUMAN, 1623
    URINARY AND PLASMA LEVELS
        BENIGN BREAST DISEASE, HUMAN, 1651

ANTHRACENE
    HISTONE BINDING, 1276

ANTIBODY
    ANTI-ENHANCING FAB ALLOANTIBODY
        PROLONGED SURVIVAL OF FIBROSARCOMA ALLOTRANSPLANTS, MOUSE, 1563*
    ANTI-THETA
        RECONSTITUTION OF T CELL-DEPLETED SPLEEN CELL POPULATIONS, MOUS, 1499
    ANTIGEN, CARCINOEMBRYONIC
        RABBIT, 1565*
    ANTINUCLEAR
        SERA, NASOPHARYNGEAL CARCINOMA PATIENTS, 1543*
        SERA, SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS, 1543*
    ANTITUMOR
        ASCITES TUMOR, SERUM, RAT, 1507*
    AVIAN GS ANTIGEN
        AVIAN RNA TUMOR VIRUS-INFECTED CHICKENS, 1474
    CELL ORGANOIDS
        SERA, NASOPHARYNGEAL CARCINOMA PATIENTS, 1543*
        SERA, SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS, 1543*
    ELIMINATION
        AUGMENTATION OF IMMUNE RESPONSE, RAT, 1568*
    HEPATOMA
        IMMUNITY, RAT, 1466
    J-CHAIN
        MURINE TUMOR CELLS, 1443
    TUMOR-SPECIFIC
        ISOTOPIC ANTIGLOBULIN ASSAY, 1569*

ANTIGEN
    A
        PURIFICATION, MAREK'S DISEASE VIRUS, 1487
    AUSTRALIA
        INCIDENCE, CANCER PATIENTS, 1525*
    CARCINOEMBRYONIC
        ANTIBODY PRODUCTION, RABBIT, 1565*
        GASTROINTESTINAL CANCER PATIENTS, 1537*
        QUANTITATIVE STUDY OF MALIGNANT NEOPLASMS, HUMAN, 1465
        RADIOIMMUNOASSAY, HUMAN COLON CARCINOMA, 1463
        UROTHELIAL CARCINOMA, CLINICAL STUDY, 1552*
    CELL-SURFACE
        MAREK'S DISEASE VIRUS-INFECTED QUAIL CELLS, 1469
        3'-METHYL-4-DIMETHYLAMINOAZOBENZENE, RAT LIVER CELLS, 1459
        N-METHYL-N-NITROSOUREA, RAT LIVER CELLS, 1459
        3-METHYLCHOLANTHRENE, RAT LIVER CELLS, 1459
        MOVEMENT, HEPATOMA CELLS, GUINEA PIG, 1445
        NEUROBLASTOMA, MOUSE, 1472
        TURKEY HERPESVIRUS-INFECTED QUAIL CELLS, 1469
    CROSS-REACTING EMBRYONIC
        METHYLCHOLANTHRENE-INDUCED SARCOMA, RAT, 1497
    EPSTEIN-BARR ASSOCIATED COMPLEMENT-FIXING
        LOCALIZATION, HUMAN LYMPHOBLASTOID CELL LINES, 1381
    FETAL
        CANCER, MOUSE, 1451
        TRANSPLANT RESISTANCE, HUMAN ADENOVIRUS-INDUCED TUMOR, HAMSTER, 1509*
    GROUP-SPECIFIC
        DETECTION, SERUM, HUMAN, 1502
        TYPE-SPECIFIC, 1508*
        VIRAL LEUKEMIA DEVELOPMENT, MOUSE, 1508*
    H
        DNA SYNTHESIS DEPRESSION, SPLEEN, MOUSE, 1452
    H-2
        GROWTH, MURINE CULTURED TUMOR CELLS, 1564*
        TUMOR GRAFT SYSTEM, MOUSE, 1556*
    HEPATOMA
        IMMUNITY, RAT, 1466
    HISTOCOMPATIBILITY
        TUMOR CELLS, REVIEW, 1204
    HL-A DEFICIENCY
        MALIGNANT MELANOMA PATIENTS, 1545*
    HL-A SYSTEM
        MAMMARY CARCINOMA, HUMAN, 1492
    MEMBRANE

    - DIVIDING LYMPHOID CELLS, HUMAN, 1566*
  - SPLENO-THYMIC
    - FETAL THYMUS, LEUKEMIA, HUMAN, 1505*
  - SURFACE
    - ADENOVIRUS 12-INFECTED KB CELLS, 1504
    - C-TYPE VIRUS, HYBRID MOUSE CELLS, 1341
    - SENDAI VIRUS, HYBRID MOUSE CELLS, 1341
  - TRANSPLANTATICN
    - BO-IV CARCINOMA, SYNGENEIC MICE, 1541*
    - LYMPHOID CELLS, CHIMERIC MICE, 1561*
    - MAMMARY CARCINOMA, SYNGENEIC MICE, 1541*
  - TUMOR
    - STRUCTURAL CONFORMATION, 1571*
  - TUMOR-ASSOCIATED
    - DETECTION, SERUM, HUMAN, 1502
  - TUMCR-SPECIFIC
    - MALIGNANT NEOPLASTIC TISSUES, HUMAN, 1449
    - URINARY BLADDER CARCINCMA CELL LINE, HUMAN, 1458
  - VIRUS, AVIAN LEUKCSIS
    - IMMUNOHISTCCHEMISTRY, 1488
  - VIRUS, EPSTEIN-BARR
    - DNA SYNTHESIS, HUMAN LYMPHOBLASTOID CELLS, 1386
  - VIRUS, MAMMARY TUMOR
    - RADIOIMMUNOASSAY, MOUSE, 1442
  - VIRUS, MURINE LEUKEMIA
    - DETECTION, MOUSE, 1477

- ANTIGEN, THETA
  - CROSS REACTIVITY
    - MOUSE, RAT, 1440
  - CROSS-REACTIVITY
    - MOUSE, RAT, 1444
  - LYMPHOSARCOMA
    - MOUSE, 1546*
  - SPECIES-SPECIFIC
    - MOUSE, RAT, 1444

- ANTISERUM
  - BRAIN-ASSOCIATED THYMUS ANTIGENS
    - MOUSE, RAT, 1440, 1444
  - PASSIVE ADMINISTRATION
    - ANTI-EL4 ACTIVITY, SERUM, MOUSE, 1523*
  - THYMUS EXTRACT SPECIFIC FCR MOUSE THYMUS-DERIVED CELLS
    - RABBIT, 1438

- ARGEMONE OIL
  - CARCINOGENESIS
    - MOUSE, 1246

- ARYL HYDROCARBON HYDROXYLASE
  - INDUCTION
    - POLYCYCLIC HYDROCARBONS, CHICK EMBRYO, 1252

- ASBESTOS
  - CARCINOMA OF HEAD AND NECK
    - HUMAN, 1290*
  - ENVIRONMENTAL HAZARD
    - HUMAN, 1258
  - LUNG CANCER
    - MESOTHELIOMA, HUMAN, REVIEW, 1229*
  - MESOTHELIOMA
    - HUMAN, 1258
  - OCCUPATIONAL HAZARD
    - HUMAN, 1258

- ASCITES TUMOR
  - SEE ALSO EHRLICH
  - CELL CYCLE
    - MOUSE, 1603*
  - CELL PROLIFERATION
    - MOUSE, 1603*
  - ELECTROKINETIC CHARACTERIZATION
    - MOUSE, 1765*
  - JB-1
    - GROWTH, CYTOKINETIC ANALYSIS, 1787*
    - RESTING STAGES, 1739*
  - OXIDATIVE DECARBOXYLATICN OF MALATE
    - MITOCHONDRIA, MOUSE, 1723*
  - SERUM CYTOTOXIN LEVELS
    - RAT, 1507*
  - TUMOR-HOST RELATIONSHIP
    - LYMPHOCYTES, MOUSE, 1471

- ASCORBIC ACID
  - CELL PROLIFERATION
    - REVIEW, 1213

- L-ASPARAGINASE
  - INDUCED ALTERATIONS
    - BURKITT'S LYMPHOMA CELLS, 1524*

- ASTROCYTOMA
  - CYCLIC AMP PHOSPHODIESTERASE
    - CLONED CELLS, RAT, 1620
  - MUCOPOLYSACCHARIDE, ACID
    - MYELIN SHEATH CELLS, 1742*
  - NDPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*
  - TPPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*

- ATPASE
  - ACTIVITY
    - MAMMARY TUMOR, RAT, 1254

- AZOXYMETHANE
  - INTESTINAL CARCINOMA
    - RAT, 1269

- BACILLUS CALMETTE-GUERIN
  - GROWTH, TUMOR
    - MOLONEY SARCOMA VIRUS-INDUCED TUMOR, MOUSE, 1476
  - HISTIOCYTE-TUMOR CELL INTERACTIONS
    - TUMOR REGRESSION, ULTRASTRUCTURE, GUINEA PIG, 1482
  - IMMUNE RESPONSE
    - MOLONEY SARCOMA VIRUS-INDUCED TUMOR, MOUSE, 1476
    - SKIN TUMOR, MOUSE, 1475
  - MELANOMA, MALIGNANT
    - HUMAN, 1575*

- BASAL CELL CARCINOMA
  - FIBROBLAST-LIKE CULTURED CELLS
    - CYTOGENETIC STUDY, 1754*
  - PULMONARY METASTASIS
    - CASE REPORT, 1659*
  - SKIN
    - CLINICAL STUDY, 1784*

- BASOSQUAMOUS CARCINOMA
  - CLINICAL STUDY
    - REVIEW, 1223*

- BENZENE
  - RETICULUM CELL SARCOMA
    - MOUSE, 1241

- BENZIDINE
  - URINARY BLADDER TUMOR
    - OCCUPATIONAL HAZARD, 1277

- BENZIMIDAZOLE
  - MUTAGENESIS
    - SALMONELLA TYPHIMURIUM, 1285*

- BENZO(A)PYRENE
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 1331
  - DNA BINDING
    - CALF THYMUS, 1245
  - TRANSFORMATION
    - EMBRYO CELLS, RAT, 1263

- BENZOYL-L-ARGININEAMIDE
  - MUTAGENESIS
    - E.COLI, 1253
    - S.TYPHIMURIUM, 1253

- 3,4-BENZPYRENE
  - HISTONE BINDING, 1276

- BIS(CHLOROMETHYL)ETHER
  - DNA SYNTHESIS
    - SKIN, MOUSE, 1275
  - RNA SYNTHESIS
    - SKIN, MOUSE, 1275

- BLOOD
  - DEMONSTRATION OF PROGENITOR CRYPTOCIDES
    - NEOPLASTIC DISEASE PATIENTS, 1658*
  - ERYTHROPOIETIC ACTIVITY
    - COLONY FORMING UNITS, LEUKEMIA VIRUS-INFECTED CELLS, MOUSE, 1412*
  - HEMOPOIESIS
    - ACUTE LEUKEMIA DEVELOPMENT, RAT, 1577
  - OXYGEN CONSUMPTION
    - LEUKOCYTES, LEUKEMIA PATIENTS, 1661*
  - PLATELET KINETICS
    - ADENOSINE TRIPHOSPHATASE ACTIVITY, LEUKEMIC MOUSE, 1428*

- BLOOD GROUP
  - BRAIN TUMOR INCIDENCE
    - CORRELATIONS, STATISTICAL STUDY, 1753*

- BONE
  - GIANT CELL TUMORS
    - ULTRASTRUCTURE, CASE REPORTS, 1769*
  - OSTEOSARCOMA
    - ALKALINE PHOSPHATASE ACTIVITY, MOUSE, 1702*
    - CALCITONIN, CASE REPORT, 1282*
  - OSTEOSARCOMA, EXTRASKELETAL
    - RADIATION-INDUCED, CASE REPORT, 1319*
  - VIRUS, MURINE SARCOMA
    - TUMORIGENICITY, RAT, MOUSE, HAMSTER, 1359

- BONE MARROW
  - C-TYPE VIRUS ACTIVATION
    - METHYLNITROSOUREA, MOUSE, 1280
  - CATECHOLAMINES
    - DETECTION, NEUROBLASTOMA CELLS, HUMAN, 1613
  - CELL PROLIFERATION
    - SERUM FACTOR, LEUKEMIC CELLS, HUMAN, 1640
  - CHROMOSOME, Y
    - AGE-ASSOCIATED ANEUPLOIDY, HUMAN LEUKEMIA, 1663*
  - GRANULOCYTIC COLONY FORMATION
    - NORMAL AND LEUKEMIC HUMAN CELLS, 1724*, 1725*
  - HEMOPOIESIS
    - ACUTE LEUKEMIA DEVELOPMENT, RAT, 1577
  - MURAMIDASE
    - LEUKEMIA, CLINICAL STUDY, 1778*

- BRAIN
  - COMPOSITE GLIOMA-SARCOMA
    - ULTRASTRUCTURE, CASE REPORT, 1743*
  - EPENDYMOBLASTOMA
    - ULTRASTRUCTURE, CASE REPORT, 1759*
  - HEMANGIOBLASTOMA, CEREBELLAR
    - ULTRASTRUCTURE, STROMA CELLS, CASE REPORT, 1586*
  - RADIATION, X-RAY
    - GOMORI POSITIVE GLIAL CELLS, MOUSE, 1318*

- BRAIN TUMOR
  - CONNATAL INTRACRANIAL
    - CLINICAL STUDY, 1781*
  - GROWTH, TUMOR
    - ULTRASONIC VISUALIZATION, CASE REPORTS, 1599*
  - INCIDENCE
    - BLOOD GROUP CORRELATIONS, STATISTICAL STUDY, 1753*
  - MAMMARY CARCINOMA
    - FAMILIAL OCCURRENCE, 1691*
  - NDPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*
  - TPPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*

- BREAST
  - SEE MAMMARY

- 5-BROMODEOXYURIDINE
  - C-TYPE VIRUS ACTIVATION

    - EMBRYONIC RAT FIBROBLASTS. 1355
  - CYCLIC AMP
    - GLIAL TUMOR CELLS. 1665*
  - TUMORIGENICITY
    - SV40-TRANSFORMED HAMSTER KIDNEY CELLS. 1394
  - VIRUS. C-TYPE RNA
    - ACTIVATION. TRANSFORMED MOUSE CELLS. 1322
  - VIRUS. MURINE LEUKEMIA
    - INDUCTION. MOUSE. 1336

- 7-BROMOMETHYL-12-METHYLBENZ(A)ANTHRACENE
  - AZAGUANINE-RESISTANT MUTANT INDUCTION
    - HAMSTER CELLS. 1234

- 7-BROMOMETHYLBENZ(A)ANTHRACENE
  - AZAGUANINE-RESISTANT MUTANT INDUCTION
    - HAMSTER CELLS. 1234

- BRONCHOGENIC CARCINOMA
  - GONADOTROPHIN SYNTHESIS
    - HUMAN CELL CULTURE. 1611

- BURKITT'S LYMPHOMA
  - CELL
    - L-ASPARAGINASE-INDUCED ALTERATIONS. 1524*
    - NEOCARZINOSTATIN TREATMENT. ULTRASTRUCTURAL STUDY. 1425*
  - ETIOLOGY
    - UGANDA. 1435*
  - MYCOPLASMA-LIKE ORGANISMS
    - CASE REPORTS. 1372
  - VIRUS. EPSTEIN-BARR
    - ETIOLOGY. REVIEW. 1219
  - VIRUS. HERPES SAIMIRI
    - HUMAN. MONKEY. 1349
  - VIRUS. RNA TUMOR
    - HUMAN. 1410

- CALCITONIN
  - OSTEOSARCOMA
    - CASE REPORT. 1282*
  - SECRETION
    - MEDULLARY CARCINOMA OF THYROID. CASE REPORT. 1749*

- CANCER
  - CYTOLOGY
    - MOUSE. HUMAN. 1798*
  - FREQUENCY VARIATIONS
    - FAMILIAL OCCURRENCE. 1602*
  - HYPOTHESIS
    - VIROLOGICAL AND IMMUNOLOGICAL RELATIONSHIPS. 1346
  - RESEARCH PROGRAMS
    - REVIEW. 1221*

- CARBOHYDRATE
  - DIETARY EFFECT
    - LIPOGENESIS. MAMMARY ADENOCARCINOMA. RAT. 1660*

- CARCINOGEN. CHEMICAL
  - EXPOSURE STANDARDS
    - HUMAN. 1253*

- CARCINOMA
  - 3H-THYMIDINE LABELING
    - AUTORADIOGRAPHY. 1580

- CARRAGEENAN
  - CYTOTOXICITY. SYNERGISTIC
    - MOLONEY SARCOMA CELLS. MOUSE. 1437

- CATECHOLAMINE
  - PHAEOCHROMOCYTOMA
    - RADIOIMMUNOASSAY. 1704*

- CATECHOLAMINES
  - BONE MARROW
    - DETECTION. NEUROBLASTOMA CELLS. HUMAN. 1613

- CECUM
  - CANCER INCIDENCE
    - WEST VIRGINIA. 1597*

- CELL
  - ARGENTAFFIN
    - MEDULLARY THYROID CARCINOMA PATIENTS. 1751*
  - B CELL
    - AUTOIMMUNITY. MOUSE. 1576*
  - BURKITT'S LYMPHOMA
    - L-ASPARAGINASE-INDUCED ALTERATIONS. 1524*
    - NEOCARZINOSTATIN TREATMENT. ULTRASTRUCTURAL STUDY. 1425*
  - C CELL
    - MEDULLARY THYROID CARCINOMA PATIENTS. 1751*
  - COLONY FORMING
    - DETERMINATION OF THE NUMBER OF CLONOGENIC CELLS. 1774*
  - EHRLICH ASCITES TUMOR
    - PLASMA MEMBRANE ISOLATION. 1664*
    - SURFACE MOLECULAR COMPONENTS. 1550*
  - ENHANCED MALIGNANT BEHAVIOR
    - HISTONE TREATMENT. HAMSTER KIDNEY CELLS. 1633
  - EPITHELIUM
    - CHANGES IN THE SURFACE COATS. HUMAN MAMMARY TUMOR. 1645
  - GOMORI POSITIVE GLIA
    - X-IRRADIATION. BRAIN. MOUSE. 1318*
  - GRANULOCYTIC COLONY FORMATION
    - NORMAL AND LEUKEMIC HUMAN HEMATOPOIETIC CELL. 1724*. 1725*
  - HYPOXIC
    - IN VITRO TUMOR MODEL. 1732*
  - INTERCELLULAR LACUNAE
    - ULTRASTRUCTURE. STIMULATED LEUKOCYTE CULTURE. 1522*
  - LYMPHORETICULAR
    - ULTRASTRUCTURE. NON-LYMPHOID HUMAN NEOPLASMS. 1770*
  - MITOTIC AUTOREGULATION
    - NEOPLASIA. MATHEMATICAL MODEL. 1629
  - PLAQUE FORMING
    - EFFECT OF DETERGENTS. IMMUNITY. 1526*
  - REIDER
    - MICROTUBULES. ULTRASTRUCTURE. HUMAN LEUKEMIA. 1794*

- RING-SHAPED PARTICLES
  - ULTRASTRUCTURE, HUMAN MALIGNANT CULTURED CELLS, 1624
- ROSETTE-FORMING
  - SPLEEN, MOUSE, 1533*
  - T CELLS, IMMUNOSUPPRESSION, 1513*
- SURFACE ALTERATION
  - MURINE SARCOMA VIRUS-TRANSFORMED CELL LINE, MOUSE, 1404
- SURFACE MOTILITY
  - CONTACT INHIBITION OF MITOSIS, ROUS SARCOMA CELL LINE, RAT, 1434*
  - PINOCYTOSIS, ROUS SARCOMA CELL LINE, RAT, 1434*
- T AND B CELL
  - ACUTE LYMPHOBLASTIC LEUKEMIA, HUMAN, 1729*
  - HAPTEN-REACTIVE HELPER LYMPHOCYTES, 1529*
  - MAREK'S DISEASE LYMPHOMAS, CHICKEN, 1416*
- T CELL
  - RECONSTITUTION FACTOR, MOUSE, 1499
- T CELL ACTIVATION
  - IMMUNOMANIPULATION OF TUMORS, MOUSE, 1468

CELL CYCLE
- ASCITES TUMOR
  - MOUSE, 1603*
- LEUKEMIA, ACUTE LYMPHOBLASTIC
  - KINETICS, COMPUTER MODEL, HUMAN, 1593
- LINEAR ENERGY TRANSFER
  - RADIATION HEPATOCARCINOGENESIS, MOUSE, 1303

CELL DIFFERENTIATION
- CYTOPLASMIC DNA
  - POLYOMA VIRUS-TRANSFORMED HAMSTER CELLS, 1324
- PLAQUE FORMING CELL PRODUCTION
  - EFFECT OF DETERGENTS, IMMUNITY, 1526*

CELL PROLIFERATION
- ASCITES TUMOR
  - MOUSE, 1603*
- ASCORBIC ACID
  - REVIEW, 1213
- BONE MARROW
  - SERUM FACTOR, LEUKEMIC CELLS, HUMAN, 1640
- CARCINOGENESIS
  - REVIEW, 1202
- PROTEASES
  - LIVER, RAT, MOUSE, 1648
- VIRUS, FRIEND LEUKEMIA
  - KINETICS, MOUSE, 1366

CELL, TUMOR
- MOTILITY INHIBITION
  - SYNGENEIC IMMUNE SERUM, MOUSE, 1553*

CERVICAL CARCINOMA
- EPIDEMIOLOGY
  - TRANSMISSIBLE CARCINOGENIC AGENTS, REVIEW, 1217
- ETIOLOGY
  - REVIEW, 1216
- HYPOGLYCEMIA
  - IMMUNOASSAYABLE INSULIN, CASE REPORT, 1670*

CERVIX
- ANAPLASIA
  - HERPESVIRUS, CYTOHISTOPATHOLOGY, HUMAN, 1393
- TRANSMISSIBLE CARCINOGENIC AGENTS
  - EPIDEMIOLOGY, REVIEW, 1217

1'ACETOXYSAFROLE
- ELECTROPHILIC REACTIVITY
  - SAFROLE METABOLISM, 1274

CHILDREN
- HODGKIN'S DISEASE
  - CLINICAL STUDY, 1675*, 1705*
- LEUKEMIA, ARACHNOID
  - HISTOPATHOLOGICAL STUDY, 1717*

CHIMERISM
- LYMPHOID CELLS
  - TRANSPLANTATION ANTIGENS, MOUSE, 1561*

1-(2-CHLOROETHYL)-3-CYCLOHEXYL-1-NITROSOUREA
- PHYSIOLOGIC DISPOSITION
  - HUMAN, 1297*

1-(2-CHLOROETHYL)-3(4-METHYL CYCLOHEXYL)-1-NITROSOUREA
- PHYSIOLOGIC DISPOSITION
  - HUMAN, 1297*

CHLOROMETHYL METHYL ETHER
- DNA SYNTHESIS
  - SKIN, MOUSE, 1275
- LUNG CANCER
  - OCCUPATIONAL HAZARD, 1299*
- RNA SYNTHESIS
  - SKIN, MOUSE, 1275

CHOLESTEROL
- CONTENT
  - MICROSOMES, MORRIS HEPATOMA, 1789*
- DNA SYNTHESIS
  - CULTURED MOUSE CELLS, 1701*

CHOLESTEROL SYNTHESIS
- 2-ACETYLAMINOFLUORENE-INDUCED HEPATOMAS
  - PRECANCEROUS LIVER, RAT, 1646
- ETHIONINE
  - FEEDBACK CONTROL, PRECANCEROUS LIVER, RAT, 1583
- HYPERPLASTIC LIVER NODULES
  - PRECANCEROUS LIVER, RAT, 1646

CHONDROSARCOMA
- DEVELOPMENT IN AN AREA OF IRRADIATED FIBROUS DYSPLASIA
  - CASE REPORT, 1314*
- EXTRASKELETAL MESENCHYMAL
  - CASE REPORT, 1662*

CHROMATIN
- THERMAL STABILITY
  - MICROFLUOROMETRIC COMPARISONS, NORMAL AND NEOPLASTIC CELLS, 1643

CHROMATIN, SEX
- VARIABLE PATTERN
  - URINARY BLADDER CARCINOMA, CASE REPORT, 1737*

CHROMOSOME
- ADENOVIRUS SA7-TRANSFORMED CELLS
  - HAMSTER, 1364, 1371
- ANALYSIS
  - GARDNER'S SYNDROME, HUMAN COLONIC POLYPS, 1609
- BANDING PATTERN
  - POLYOMA VIRUS-TRANSFORMED HAMSTER CELLS, 1332
- 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED SARCOMA
  - HAMSTER, 1243
- FIBROBLAST-LIKE CULTURED CELLS
  - BASAL CELL CARCINOMA PATIENTS, 1754*
- MALIGNANCY CONTROL
  - IDENTIFICATION, HAMSTER CELLS, 1265

CHROMOSOME ABERRATIONS
- CYCLOHEXYLAMINE
  - CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*
- LEUKEMIA, CHRONIC MYELOCYTIC
  - BLASTIC CRISIS REMISSION, HUMAN, 1757*
- N-METHYL-N'-NITRO-NITROSOGUANIDINE
  - XERODERMA PIGMENTOSUM CELLS, 1260
- 4-NITROQUINOLINE-1-OXIDE
  - ADENOVIRUS INFECTION, HAMSTER CELLS, 1363
  - XERODERMA PIGMENTOSUM CELLS, 1260
- SODIUM CYCLAMATE
  - CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*

CHROMOSOME, Y
- BONE MARROW
  - AGE-ASSOCIATED ANEUPLOIDY, HUMAN LEUKEMIA, 1663*

COCARCINOGENICITY
- EYE LENS EXTRACT
  - FISH, 1256

COLCHICINE
- STEROID SECRETION STIMULATION
  - ADRENAL TUMOR CELLS, MOUSE, 1666*
- THERAPY FOR GOUT
  - RETICULUM CELL SARCOMA DEVELOPMENT, CASE REPORT, 1294*
  - STEATORRHOEA DEVELOPMENT, CASE REPORT, 1294*

COLLAGENASE
- CELL PROLIFERATION
  - LIVER, RAT, MOUSE, 1648
- CELL SURFACE ARCHITECTURE
  - LIVER, RAT, MOUSE, 1648
- DNA SYNTHESIS
  - LIVER, RAT, MOUSE, 1648

COLON
- ADENOCARCINOMA
  - 1,2-DIMETHYLHYDRAZINE, MOUSE, 1270
- COLORECTAL CARCINOMA
  - CLINICAL STUDY, 1690*
- MALIGNANT GIANT-CELL TUMOR
  - CASE REPORT, 1744*

COLON CARCINOMA
- ANTIGEN, CARCINOEMBRYONIC
  - RADIOIMMUNOASSAY, HUMAN, 1463
- 1,2-DIMETHYLHYDRAZINE
  - MOUSE, 1270
- ENVIRONMENTAL FACTORS
  - HUMAN, 1594
- INCIDENCE
  - SOUTH SASKATCHEWAN, 1708*
- MORTALITY
  - U.S.A., 1601*

COLON, SIGMOID
- ADENOCARCINOMA
  - URETEROSIGMOIDOSTOMY, CASE REPORTS, 1746*, 1747*

COLON, TRANSVERSE
- ADENOCARCINOMA
  - RADIATION-INDUCED, CASE REPORT, 1309*

COLORECTAL CARCINOMA
- CLINICAL STUDY, 1690*
- DIET
  - HUMAN, 1734*
- ENVIRONMENTAL FACTORS
  - HUMAN, 1734*

CONCANAVALIN A
- AGGLUTINATION
  - PURIFICATION OF ONCORNA VIRUSES, 1387
- BINDING
  - LYMPHOCYTE TRANSFORMATION, HUMAN, 1668*
- BINDING SITE
  - ISOLATION, CELL-SURFACE GLYCOPEPTIDES, RAT HEPATOMA, 1455
  - TEMPERATURE DEPENDENT MOBILITY, MOUSE CELLS, 1478
  - TUMOR CELL SURFACES, MOUSE, 1478
- DNA SYNTHESIS
  - SPLEEN CELLS, RABBIT, 1467
- LYMPHOCYTE TRANSFORMATION
  - AUTOLOGOUS HUMAN LYMPHOBLASTS, 1500
- MEMBRANE RECEPTOR-CYTOPLASMIC INTERACTION
  - LYMPHOCYTE RECEPTOR MOBILITY, 1453
- METAL ION INTERACTION
  - EQUILIBRIUM DIALYSIS, 1574*
- MITOGENIC ACTIVITY
  - SPLEEN CELLS, RABBIT, 1467
- PROTON RELAXATION ENHANCEMENT PROPERTIES

    - NUCLEAR MAGNETIC RESONANCE, 1495

- COPPER
  - DL-ETHIONINE CARCINOGENESIS INHIBITION
    - LIVER, RAT, 1271

- CORTISOL
  - DNA SYNTHESIS
    - CULTURED MOUSE CELLS, 1701*

- CROTON OIL
  - MITOSIS
    - BLUE-GREEN BACTERIA, 1296*
  - SKIN TUMOR PROMOTION
    - BCG VACCINATION, MOUSE, 1475

- CYCLIC AMP
  - 5-BROMODEOXYURIDINE
    - GLIAL TUMOR CELLS, 1665*
  - EHRLICH ASCITES TUMOR
    - CELL KINETICS, 1703*
  - LEUKEMIA, CHRONIC LYMPHATIC
    - CLINICAL STUDY, 1796*
  - LEUKEMIA, CHRONIC MYELOGENOUS
    - CLINICAL STUDY, 1796*
  - PHOSPHODIESTERASE
    - CLONED ASTROCYTOMA CELLS, RAT, 1620
  - PROTEIN KINASE LEVELS
    - FELINE SARCOMA VIRUS-TRANSFORMED CELLS, 1420*
  - REGENERATION, LIMB
    - NEWT, 1687*

- CYCLOHEXIMIDE
  - DIMETHYLNITROSAMINE
    - TUMOR INDUCTION, RAT, 1261
  - GIANT CELL FORMATION
    - IRRADIATION, OEDOGONIUM CARDIACUM, 1305

- CYCLOHEXYLAMINE
  - CHROMOSOME ABERRATIONS
    - CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*
  - DNA SYNTHESIS
    - CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*

- CYCLOPHOSPHAMIDE
  - IMMUNITY
    - 3-METHYL-CHOLANTHRENE-INDUCED SARCOMA, MOUSE, 1281*

- ALPHA-S-CYSTEINYL-5,6-DIHYDROTHYMINE
  - PROPERTIES, 1315*
  - RADIATION-INDUCED CROSS-LINKING OF DNA AND PROTEIN
    - MODEL, 1315*

- CYSTIC TUMOR
  - SPINAL CORD
    - CYST PUNCTURE, ENDOMYELOGRAPHY, CASE REPORTS, 1728*

- CYTOCHROME P-448
  - INDUCTION
    - POLYCHLORINATED BIPHENYLS, LIVER, RAT, 1298*

- CYTOLOGY
  - CANCER
    - MOUSE, HUMAN, 1798*

- CYTOTOXICITY
  - IMMUNE RESPONSE
    - GROSS VIRUS-INDUCED LYMPHOMA, RAT, 1489
    - MURINE SARCOMA VIRUS-INDUCED TUMORS, EFFECTOR CELL, MOUSE, 1490
  - LYMPH NODE CELL
    - HEPATOMA, IMMUNITY, RAT, 1466

- CYTOTOXICITY, SYNERGISTIC
  - CARRAGEENAN
    - MOLONEY SARCOMA CELLS, MOUSE, 1437

- DDT
  - HEPATOMA
    - LONG-TERM EFFECT, MOUSE, 1238
  - LUNG ADENOMA
    - LONG-TERM EFFECT, MOUSE, 1232, 1238
  - LYMPHOMA, MALIGNANT
    - LONG-TERM EFFECT, MOUSE, 1238

- 2-DEOXY-D-GLUCOSE
  - IMMUNOGLOBULIN IGG
    - BIOSYNTHESIS, INTRACELLULAR TRANSPORT, SECRETION, 1527*

- DEOXYRIBONUCLEOPROTEIN
  - IR SPECTRA ALTERATIONS
    - IONIZING RADIATION, CALF THYMUS, 1317*
    - UV RADIATION, CALF THYMUS, 1317*

- DETERGENTS
  - PLAQUE FORMING CELL PRODUCTION
    - IMMUNITY, 1526*

- DIBENZ(A,C)ANTHRACENE
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 1331

- DIBENZ(A,H)ANTHRACENE
  - ADENOVIRUS TRANSFORMATION
    - HAMSTER CELLS, 1331

- DIBUTYRL CYCLIC AMP
  - VIRUS, POLYOMA
    - TRANSFORMATION, 1328
  - VIRUS, SV40
    - TRANSFORMATION, 1328

- DIBUTYRYL AMP
  - REGENERATION, LIMB
    - NEWT, 1687*

- DIET
  - COLORECTAL CARCINOMA
    - HUMAN, 1734*
  - HIGH CARBOHYDRATE
    - LIPOGENESIS, MAMMARY ADENOCARCINOMA, RAT, 1660*
  - NITRILOTRIACETIC ACID
    - NITROSAMINE FORMATION, RAT, 1289*
  - POLYCHLORINATED BIPHENYLS

    GASTRIC LESIONS, MONKEY, 1240

DIETHYL PYROCARBONATE
  NUCLEIC ACID
    REACTION PRODUCTS, 1244

DIETHYLSTILBESTROL
  MAMMARY TUMOR
    INHIBITION BY PROGESTERONE, RAT, 1302

DIMETHYL SULFOXIDE
  VIRUS, MOLONEY SARCOMA
    ONCOGENICITY STIMULATION, MOUSE, 1360
  VIRUS, ROUS SARCOMA
    ONCOGENICITY STIMULATION, QUAIL, 1360

P-DIMETHYLAMINOAZOBENZENE
  METABOLISM
    AGE, RAT LIVER, 1233
    3-METHYLCHOLANTHRENE, RAT LIVER, 1233

1-(4-DIMETHYLAMINOBENZYLIDENE)INDENE
  TUMORIGENICITY
    ANTI-TUMOR EFFECT, MOUSE, 1255

7,12-DIMETHYLBENZ(A)ANTHRACENE
  ADENOVIRUS TRANSFORMATION
    HAMSTER CELLS, 1331
  EPOXIDES
    PREPARATION AND METABOLISM, 1288*
  MAMMARY TUMOR
    ATPASE ACTIVITY, RAT, 1254
    FRUCTOSE-1,6-DIPHOSPHATASE, RAT, 1248
    HORMONE DEPENDENCE, RAT, 1259
    TRANSPLANTATION, RAT, 1259
    UTPASE ACTIVITY, RAT, 1254
  SARCOMA
    CHROMOSOMES, HAMSTER, 1243
  SKIN TUMOR
    BCG VACCINATION, MOUSE, 1475
    CROTON OIL, MOUSE, 1475
  TRANSFORMATION
    EMBRYO CELLS, RAT, 1263

1,2-DIMETHYLHYDRAZINE
  COLON CARCINOMA
    MOUSE, 1270
  INTESTINAL CARCINOMA
    VITAMIN A, RAT, 1267

DIMETHYLNITROSAMINE
  ACTINOMYCIN D
    TUMOR INDUCTION, RAT, 1261
  CYCLOHEXIMIDE
    TUMOR INDUCTION, RAT, 1261
  DNA SYNTHESIS
    KIDNEY, RAT, 1261
  RNA SYNTHESIS
    KIDNEY, RAT, 1261

N,N-DIMETHYLNITROSAMINE
  DNA METHYLATION
    LIVER, RAT, 1239

DNA
  BINDING
    BENZO(A)PYRENE, CALF THYMUS, 1245
    N-MONOMETHYL-4-AMINOAZOBENZENE, IN VITRO REACTIONS, 1268
  CIRCULAR
    SV40-INFECTED CELLS, POLYACRYLAMIDE GEL ELECTROPHORESIS, 1327
  COMPLEX MITOCHONDRIAL
    HUMAN TUMORS, 1641
  CROSS-LINK MEASUREMENT
    NITROGEN MUSTARD, LYMPHOMA CELLS, MOUSE, 1251
  INTEGRATION OF VIRAL INTO CHROMOSOMAL
    POLYOMA-TRANSFORMED CELLS, RAT, 1407
  IR SPECTRA ALTERATIONS
    IONIZING RADIATION, CALF THYMUS, 1317*
    UV RADIATION, CALF THYMUS, 1317*
  N-METHYL-N-NITROSOUREA
    NEURONAL AND GLIAL CELLS, RAT, 1272
  METHYLATION
    N,N-DIMETHYLNITROSAMINE, RAT LIVER, 1239
  NUCLEAR CONTENT
    ADENOCYSTIC CARCINOMA, HUMAN, 1683*
  PHOTODAMAGE
    FUROCOUMARINS, POST-IRRADIATION DARK RECOVERY, 1316*
  PROTEIN
    RADIATION-INDUCED CROSS-LINKING, MODEL, 1315*
  RADIATION, GAMMA
    ENDONUCLEASE ACTIVITY, HUMAN CELLS, 1313*
  RESTRICTION ENZYMES
    REVIEW, 1224*
  RHABDOMYOSARCOMA CELLS
    CELL VOLUME DISTRIBUTIONS, RADIATION, RAT, 1696*
  VIRUS, AVIAN LEUKOSIS
    RNA-DNA HYBRIDIZATION, CHICK EMBRYO, 1391
  VIRUS, SV40
    INTEGRATION SITE, TRANSFORMED MAMMALIAN CELLS, 1396
    5-IODO-2'-DEOXYURIDINE INCORPORATION, 1402
    RESTRICTION ENDONUCLEASE, E.COLI, 1417*

DNA, CYTOPLASMIC
  NUCLEAR SEQUENCES
    POLYOMA VIRUS-TRANSFORMED HAMSTER CELLS, 1324

DNA METABOLISM
  VIRUS, SV40
    SUPERINFECTION, TRANSFORMED KIDNEY CELLS, 1358

DNA POLYMERASE
  HEPATOMA
    CHARACTERIZATION, RAT, 1638
  HODGKIN'S DISEASE

CHARACTERIZATION, 1635
INHIBITION BY RIFAMYCIN
LEUKEMOGENESIS, MOUSE, 1383
SYNTHETIC RNA TEMPLATE UTILIZATION
CULTURED MOUSE CELLS, 1667*
VIRUS, AVIAN MYELOBLASTOSIS
DNA SYNTHESIS, INCORRECT BASE PAIRING, 1614
INHIBITION, NARCISSUS TAZETTA L., 1421*

DNA REPAIR SYNTHESIS
4-NITROQUINOLINE-1-OXIDE
ADENOVIRUS INFECTION, HAMSTER CELLS, 1363
RADIATION, UV
XERODERMA PIGMENTOSUM PATIENTS, 1301

DNA REPLICATION
VIRUS, POLYOMA
PHYSICOCHEMICAL PROPERTIES, 1357

DNA, SATELLITE
METHYLATION
NORMAL AND NEOPLASTIC CELL CULTURES, MOUSE, 1733*

DNA SYNTHESIS
ADENOSINE DIPHOSPHORIBOSYLATION
NUCLEAR PROTEINS, HELA CELLS, 1654*
ALKYLATING AGENT
SKIN, MOUSE, 1275
ANTIGEN-INDUCED DEPRESSION
SPLEEN, MOUSE, 1452
BIS(CHLOROMETHYL)ETHER
SKIN, MOUSE, 1275
CHLOROMETHYL METHYL ETHER
SKIN, MOUSE, 1275
CHOLESTEROL
CULTURED MOUSE CELLS, 1701*
CONCANAVALIN A
SPLEEN CELLS, RABBIT, 1467
CORTISOL
CULTURED MOUSE CELLS, 1701*
CYCLOHEXYLAMINE
CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*
DIMETHYLNITROSAMINE
KIDNEY, RAT, 1261
ENHANCEMENT OF REPLICATION OF HUMAN ADENOVIRUSES BY SV40
SIMIAN CELLS, 1408
EPSTEIN-BARR VIRUS ANTIGEN
HUMAN LYMPHOBLASTOID CELLS, 1386
INHIBITION
SHOPE FIBROMA VIRUS, 1373
LEUKEMIA, ACUTE
ERYTHROBLASTS, HUMAN, 1608
LEUKEMIA, CHRONIC MYELOID
ERYTHROBLASTS, HUMAN, 1608
PROGESTERONE
CULTURED MOUSE CELLS, 1701*
GAMMA-PROPANE SULTONE
SKIN, MOUSE, 1275
BETA-PROPIOLACTONE
SKIN, MOUSE, 1275
PROTEASES
LIVER, RAT, MOUSE, 1648
REVERSE TRANSCRIPTASE
INCORRECT BASE PAIRING, 1614
SODIUM CYCLAMATE
CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*
STIMULATING FACTOR
CELL GROWTH, SERUM, PARTIALLY HEPATECTOMIZED RATS, 1616
URETHANE
SKIN, MOUSE, 1275
VIRUS, ROUS SARCOMA
RNA TEMPLATE CHARACTERIZATION, 1411

DNA TRANSCRIPTION
N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
TRANSFORMED CELLS, HAMSTER, 1266
VIRUS, HERPES SIMPLEX
CERVICAL CANCER TISSUE, HUMAN, 1397

DNASE
ACTIVITY
TRANSPLANTABLE TUMORS, HISTOCHEMISTRY, RAT, 1631

DOPAMINE BETA-HYDROXYLASE
PHAEOCHROMOCYTOMA
RADIOIMMUNOASSAY, 1704*

DUST
METALS, RARE EARTH
RETICULUM CELL SARCOMA OF LUNG, RAT, 1304

EHRLICH ASCITES TUMOR
CELL
PLASMA MEMBRANE ISOLATION, 1664*
SURFACE MOLECULAR COMPONENTS, 1550*
DISTRIBUTION OF RIBOSOMAL CISTRONS
NUCLEI, HEPATIC CELLS, MOUSE, 1650
GROWTH, TUMOR
CELL-FREE ASCITES FLUID, CELL KINETICS, 1703*
CYCLIC AMP, CELL KINETICS, 1703*
INCORPORATION AND RELEASE OF 51CR AND 14C-LABELED AMINO ACIDS, 1782*
LIPID CONTENT
FLUCTUATION DURING THE GROWTH PERIOD, 1791*
REPRESSION OF PROPAGATION
ATTENUATED TUMOR CELLS, 1516*
RIBONUCLEOTIDE REDUCTASE
INHIBITION BY RNA, 1649
URIDINE KINASE
PARTIAL PURIFICATION, 1680*

EHRLICH'S REAGENT
COLORIMETRIC RESPONSE
PLASMA, NORMAL AND CANCER PATIENTS, 1706*

ENDONUCLEASE
ACTIVITY
GAMMA-IRRADIATED DNA, HUMAN CELLS, 1313*
RESTRICTION

    - CONVERSION OF CIRCULAR TO LINEAR SV40 DNA, E.COLI, 1417*

ENDOPLASMIC RETICULUM
- INTERCISTERNAL STRUCTURES
    - ULTRASTRUCTURE, HUMAN MENINGIOMA, 1790*
- PHOSPHOLIPID COMPOSITION CHANGES
    - 20-METHYLCHOLANTHRENE, LIVER, RAT, 1273
    - PHENOBARBITAL, LIVER, RAT, 1273

ENVIRONMENTAL HAZARD
- ASBESTOS
    - HUMAN, 1258

ENZYME
- SEE ALSO NAMES OF
- ACID PHOSPHATASE ISOZYMES
    - PROSTATIC CARCINOMA PATIENTS, 1713*
- LYSOSOMAL
    - ACTIVITY, CHRONIC LYMPHOCYTIC LEUKEMIA LYMPHOCYTES, 1653
- RESTRICTION
    - DNA, REVIEW, 1224*
- TRANSACETYLASE
    - N-HYDROXY-N-ACETYLAMINOFLUORENE, RAT, 1235
- TRANSFER RNA SYNTHETASE ACTIVITY
    - VIRUS, AVIAN MYELOBLASTOSIS, 1388

EPENDYMOBLASTOMA
- BRAIN
    - ULTRASTRUCTURE, CASE REPORT, 1759*

EPIDEMIOLOGY
- ADENOCARCINOMA OF THE PANCREAS
    - NEW YORK CITY, 1596*
- CERVICAL CARCINOMA
    - TRANSMISSIBLE CARCINOGENIC AGENTS, REVIEW, 1217
- PANCREAS CARCINOMA
    - U.S.A., 1589

EPOXIDES
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - PREPARATION AND METABOLISM, 1288*
- HYDROCARBON, POLYCYCLIC AROMATIC
    - PREPARATION AND METABOLISM, 1288*

ERYTHROBLAST
- DNA SYNTHESIS
    - BIOCHEMICAL ABNORMALITY, LEUKEMIA PATIENTS, 1608

ERYTHROLEUKEMIA
- MELPHALAN TREATMENT
    - PRIMARY MACROGLOBULINEMIA PATIENT, CASE REPORT, 1278

ERYTHROPOIESIS
- COLONY FORMING UNITS
    - GROWTH, LEUKEMIA VIRUS-INFECTED TRANSPLANTED CELLS, MOUSE, 1412*

ESOPHAGEAL CARCINOMA
- CAUSE AND PREVENTION
    - S. AFRICA, 1292*
- NEOPLASMS, MALIGNANT
    - INCIDENCE, S.AFRICA, 1592

ESTERASE
- PRIMATE-SPECIFIC
    - QUANTITATIVE DETERMINATION, NORMAL AND MALIGNANT TISSUES, HUMAN, 1610

H3-ESTRADIOL
- LOCALIZATION
    - HUMAN PROSTATIC CARCINOMA, 1712*

ESTROGEN
- MELANOMA, MALIGNANT
    - DEPENDENCE, HUMAN, 1626

ETHIDIUM BROMIDE
- HERPES SIMPLEX VIRUS MULTIPLICATION
    - HELA CELLS, 1423*

ETHIONINE
- CHOLESTEROL SYNTHESIS
    - FEEDBACK CONTROL, PRECANCEROUS LIVER, RAT, 1583

DL-ETHIONINE
- CARCINOGENESIS
    - INHIBITION BY COPPER, LIVER, RAT, 1271

EYE
- LENS EXTRACT
    - COCARCINOGENICITY, FISH, 1256
- RETINOBLASTOMA
    - ULTRASTRUCTURE, CASE REPORT, 1775*

FATTY ACID
- PHORBOL ESTER
    - TUMOR PROMOTION, REVIEW, 1214

FERRITIN
- CARCINO-FETAL
    - HUMAN LIVER, 1682*

ALPHA-FETOPROTEIN
- AFLATOXIN B1
    - SERUM, RAT, 1484
- N-2-FLUORENYLACETAMIDE
    - SERUM, RAT, 1484
- N-HYDROXY-N-2-FLUORENYLACETAMIDE
    - SERUM, RAT, 1484
- 2'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    - SERUM, RAT, 1484
- 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    - SERUM, RAT, 1484
- RADIOIMMUNOASSAY
    - RAT, 1479
- SERUM CONCENTRATION
    - OVARIAN TERATOCARCINOMA PATIENT, CASE REPORT, 1519*

FIBRINOLYSIN
- AVIAN SARCOMA VIRUS TRANSFORMATION
    - RAT EMBRYO CELLS, 1342
- ROUS SARCOMA VIRUS TRANSFORMATION
    - CHICK EMBRYO FIBROBLASTS, 1333
- SV40 TRANSFORMATION
    - HAMSTER EMBRYO CELLS, 1342

- FIBROSARCOMA
  - DEVELOPMENT IN LOWER ABDOMINAL WALL
    - RADIATION-INDUCED, CASE REPORT, 1320*
  - PROLONGED SURVIVAL OF ALLOTRANSPLANTS
    - ANTI-ENHANCING FAB ALLOANTIBODY, MOUSE, 1563*
  - SKIN
    - CASE REPORT, 1745*

- N-2-FLUORENYLACETAMIDE
  - ALPHA-FETOPROTEIN
    - SERUM, RAT, 1484

- N,N'-2-7-FLUORENYLENEBISACETAMIDE
  - HEPATOMA
    - MASTOMYS, 1632

- FRUCTOSE-1,6-DIPHOSPHATASE
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMOR
    - RAT, 1248

- FUROCOUMARINS
  - DNA PHOTODAMAGE
    - POST-IRRADIATION DARK RECOVERY, 1316*

- GALLIUM-67
  - LABELED ASCITES TUMOR CELLS
    - METASTASIS, MOUSE, 1684*

- GARDNER'S SYNDROME
  - POLYPS, COLONIC
    - CHROMOSOME ANALYSIS, HUMAN, 1609

- GASTRIN
  - BASAL RELEASE
    - ANTRAL PH EFFECT, DOG, 1627

- GASTROINTESTINAL CARCINOMA
  - ANTIGEN, CARCINOEMBRYONIC
    - HUMAN, 1537*
  - DETECTION
    - BLOOD SELENIUM, 1685*

- GENE
  - LEUKOSIS-TYPE VIRUS
    - PHEASANT EMBRYO CELLS, 1338

- GENETICS
  - CANCER FREQUENCY VARIATIONS
    - FAMILIAL OCCURRENCE, 1602*
  - GENETIC RECOMBINATION
    - MUTATION, REVIEW, 1230*
  - HOST-GENE CONTROL
    - C-TYPE RNA VIRUS EXPRESSION, TUMORIGENESIS, MOUSE, 1375
  - HOST RANGE RECOMBINENTS
    - ISOLATION, AVIAN RNA TUMOR VIRUSES, 1401
  - INHERITED SUSCEPTIBILITY
    - FRIEND LEUKEMIA VIRUS, MOUSE, 1330
    - LYMPHATIC LEUKEMIA VIRUS, MOUSE, 1330
    - SPLEEN FOCUS-FORMING VIRUS, MOUSE, 1330
  - POLYOMA VIRUS TRANSFORMATION
    - HAMSTER CELLS, 1332
  - RESISTANCE
    - AVIAN SARCOMA VIRUS, CHICKEN, 1335
  - TERATOCARCINOGENESIS
    - REVIEW, 1222*
  - VIRUS, HERPES SIMPLEX
    - TEMPERATURE SENSITIVE MUTANTS, 1395

- GLIOBLASTOMA
  - INCIDENCE
    - BLOOD GROUP CORRELATIONS, STATISTICAL STUDY, 1753*
  - NDPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*
  - ORGAN CULTURE SYSTEMS
    - IN VITRO CHARACTERISTICS, HUMAN, 1730*
  - TPPASE ACTIVITY
    - HISTOCHEMISTRY, HUMAN, 1748*

- GLOMERULONEPHRITIS
  - C-TYPE VIRUS LOCALIZATION
    - KIDNEY, MOUSE, 1377

- GLYCOGEN PHOSPHORYLASE
  - HEPATOMA
    - CHARACTERIZATION, RAT, 1642

- GLYCOGEN PHOSPHORYLASE B
  - ACTIVITY
    - HUMAN MYOMA OF THE UTERUS, 1777*

- GLYCOGEN SYNTHETASE
  - HEPATOMA
    - CHARACTERIZATION, RAT, 1642

- GLYCOPEPTIDE
  - CELL-SURFACE
    - ISOLATION, RAT HEPATOMA, 1455

- GLYCOPROTEIN
  - CONTENT
    - SV40-TRANSFORMED KIDNEY CELLS, MONKEY, 1382

- GONADOTROPHIN
  - HEPATOMA
    - CASE REPORT, 1634
  - HUMAN CHORIONIC
    - SYNTHESIS, BRONCHOGENIC CARCINOMA CELLS, HUMAN, 1611

- GRAFT-VS-HOST REACTION
  - GROWTH, TUMOR
    - SYNGENIC SARCOMA, MOUSE, 1555*
  - VIRAL LYMPHOMA INDUCTION
    - MOUSE, 1485
  - VIRUS, MURINE LEUKEMIA
    - TUMOR INDUCTION, MOUSE, 1321

- GRANULAR CELL TUMOR
  - NERVOUS SYSTEM, CENTRAL
    - CASE REPORTS, 1760*

- GRANULOCYTE
  - LEUKEMIA, CHRONIC MYELOGENOUS
    - THROMBOPLASTIC ACTIVITY, 1788*

- GROUND-NUT OIL

  - CARCINOGENESIS
    - MOUSE, 1246

- GROWTH
  - ABNORMAL CELLS
    - MATHEMATICAL MODEL, 1600*
  - COLONY FORMING UNITS
    - ERYTHROPOIESIS, LEUKEMIA VIRUS-INFECTED TRANSPLANTED CELL, MOUS, 1412*
  - HEPATOMA, MORRIS
    - HUMAN SERUM, RAT, 1625
  - LIVER REGENERATION
    - DNA SYNTHESIS STIMULATING FACTOR, RAT, 1616
  - LYMPHOMA
    - GRAFFI LEUKEMIA VIRUS, MOUSE, 1329
  - MENINGEOMA
    - SKULL, CASE REPORTS, 1604*
  - RHABDOMYOSARCOMA
    - MURINE SARCOMA VIRUS, MOUSE, 1329
  - TRANSPLANTATION KINETICS
    - MOUSE TUMOR CELLS, MATHEMATICAL ANALYSIS, 1448
  - TUMOR CELL
    - H-2 ANTIGEN EXPRESSION, MOUSE, 1564*

- GROWTH, TUMOR
  - BACILLUS CALMETTE-GUERIN
    - MOLONEY SARCOMA VIRUS-INDUCED TUMOR, MOUSE, 1476
    - SKIN, MOUSE, 1475
  - BRAIN TUMOR
    - ULTRASONIC VISUALIZATION, CASE REPORTS, 1599*
  - EHRLICH ASCITES TUMOR
    - CELL-FREE ASCITES FLUID, CELL KINETICS, 1703*
    - CYCLIC AMP, CELL KINETICS, 1703*
  - GRAFT-VS-HOST REACTION
    - SYNGENIC SARCOMA, MOUSE, 1555*
  - HEPATOMA
    - HORMONALLY TREATED RATS, 1637
    - MATHEMATICAL ANALYSIS, RAT, 1619
  - JB-1 ASCITES TUMOR
    - CYTOKINETIC ANALYSIS, 1787*
  - MACROPHAGE
    - REJECTION OF ALLOGENEIC LYMPHOMA, RAT, 1562*
  - MELANOMA B16 CELLS
    - TRANSPLANTABILITY, 1462
  - MUCOPOLYSACCHARIDE
    - MOUSE, 1494
  - PROTEIN CALORIE DEFICIENCY
    - IMMUNE RESPONSE, MOUSE, 1481
  - RENAL CELL CARCINOMA
    - MOUSE, 1692*
  - SYNGENEIC
    - MOUSE, 1441
  - TUMOR-HOST RELATIONSHIP
    - ASCITES CELL, MOUSE, 1471

- HEAD
  - CARCINOMA
    - ASBESTOS EXPOSURE, HUMAN, 1290*

- HEMANGIOBLASTOMA
  - CEREBELLAR
    - ULTRASTRUCTURE, STROMA CELLS, CASE REPORT, 1586*

- HEMOGLOBIN
  - ABNORMALITIES
    - NEOPLASTIC HEMATOLOGICAL DISORDERS, CLINICAL STUDY, 1738*
  - MALMCE
    - FIBROSIS AND LUNG CANCER, FAMILIAL OCCURRENCE, CASE REPORTS, 1795*

- HEMOPOIESIS
  - BONE MARROW
    - ACUTE LEUKEMIA DEVELOPMENT, RAT, 1577

- HEPATOCARCINOGENESIS
  - N,N-DIMETHYLNITROSAMINE
    - DNA METHYLATION, RAT LIVER, 1239
  - RADIATION
    - CELL CYCLE, MOUSE, 1303
    - LINEAR ENERGY TRANSFER, MOUSE, 1303
  - SAFROLE
    - METABOLISM, 1274
  - SEQUENTIAL MORPHOLOGICAL AND HISTOLOGICAL CHANGES
    - RECIPROCAL CROSS MOUSE STRAINS, 1786*

- HEPATOCARCINOMA
  - HISTIOCYTE-TUMOR CELL INTERACTIONS
    - BCG, TUMOR REGRESSION, ULTRASTRUCTURE, GUINEA PIG, 1482

- HEPATOCELLULAR CARCINOMA
  - GROWTH WITHIN BILE DUCTS
    - CASE REPORTS, 1727*
  - 1'-HYDROXYSAFROLE
    - RAT, 1262
  - SAFROLE
    - RAT, 1262

- HEPATOCYTE
  - TUMOR CELL INVASION
    - PLASMA CELL TUMOR, MOUSE, 1756*

- HEPATOMA
  - SEE ALSO LIVER
  - 2-ACETYLAMINOFLUORENE
    - CHOLESTEROL SYNTHESIS, PRECANCEROUS LIVER, RAT, 1646
  - ADENYLATE CYCLASE ACTIVITY
    - YOSHIDA ASCITES CELLS, 1761*
  - ANTIBODY
    - IMMUNITY, RAT, 1466
  - ANTIGEN
    - IMMUNITY, RAT, 1466
  - ANTIGEN, CELL SURFACE
    - MOVEMENT, GUINEA PIG, 1445
  - DDT
    - LONG-TERM EFFECT, MOUSE, 1238
  - DNA POLYMERASE
    - CHARACTERIZATION, RAT, 1638
  - N,N'-2-7-FLUORENYLENEBISACETAMIDE
    - MASTOMYS, 1632
  - GLYCOGEN PHOSPHORYLASE
    - CHARACTERIZATION, RAT, 1642
  - GLYCOGEN SYNTHETASE

    CHARACTERIZATION, RAT, 1642
  GONADOTROPHIN-PRODUCING
    CASE REPORT, 1634
  GROWTH, TUMOR
    HORMONALLY TREATED RATS, 1637
    MATHEMATICAL ANALYSIS, RAT, 1619
  ISOLATION OF CELL-SURFACE
   GLYCOPEPTIDES
    RAT, 1455
  KETONE BODY OXIDATION
    MITOCHONDRIA, MOUSE, 1762*
  PRIMATE-SPECIFIC ESTERASE
    QUANTITATIVE DETERMINATION, 1610
  RNA, RIBOSOMAL
    WARFARIN THERAPY, MOUSE, 1622
  RNA, TRANSFER
    CHROMATOGRAPHIC STUDIES, 1639

HEPATOMA, MORRIS
  GROWTH
    HUMAN SERUM, RAT, 1625
  ION-TRANSPORTING ACTIVITY
    LIVER, RAT, 1636
  LIPID METABOLISM
    RAT, 1721*
  MICROSOME
    CHOLESTEROL CONTENT, 1789*
    PHOSPHOLIPID COMPOSITION, 1789*

HEPATOMA, NOVIKOFF
  NUCLEOTIDE POOLS
    CULTURED CELLS, RAT, 1767*
  PROTEIN, NUCLEAR
    LIVER, POLYACRYLAMIDE GEL
     ELECTROPHORESIS, RAT, 1678*
  PROTEIN, NUCLEOLAR
    COMPARISON WITH NORMAL LIVER,
     POLYACRYLAMIDE GEL
     ELECTROPHORESI, 1615
  TRANSFER RNA METHYLTRANSFERASE
    LIVER, RAT, 1669*

HISTONE
  BINDING
    ANTHRACENE, 1276
    3,4-BENZYPYRENE, 1276
    9-METHYLANTHRACENE, 1276
    PYRENE, 1276
  RAT LIVER HISTONE TREATMENT
    ENHANCED MALIGNANT BEHAVIOR,
     HAMSTER KIDNEY CELLS, 1633

HODGKIN' DISEASE
  LYMPHOMA, MALIGNANT
    INCIDENCE, MINNESOTA, 1605*

HODGKIN'S DISEASE
  ALKALINE PHOSPHATASE ACTIVITY
    CLINICAL STUDY, 1779*
  CHILDREN
    CLINICAL STUDY, 1675*, 1705*
  DETECTION
    BLOOD SELENIUM, 1685*
  DNA POLYMERASE
    CHARACTERIZATION, 1635
  HETEROGENEITY
    MOLECULAR EVIDENCE, 1635
  INTRANUCLEAR MITOCHONDRIA
    ULTRASTRUCTURE, CLINICAL STUDY,
     1755*
  LONG TERM CULTURES
    MORPHOLOGIC AND RADIOAUTOGRAPHIC
     STUDY, 1720*
  THYMOMA
    THYMUS, CLINICOPATHOLOGICAL STUDY,
     1621

HORMONE
  SEE ALSO NAMES OF
  HEPATOMA GROWTH
    ADENYLATE KINASE INDUCTION, RAT,
     1637
    PYRUVATE KINASE INDUCTION, RAT,
     1637

HORMONE, ADRENOCORTICOTROPIC
  PRODUCTION
    ISLET CELL CARCINOMA, CASE REPORT,
     1693*
  SECRETION
    MEDULLARY CARCINOMA OF THYROID,
     CASE REPORT, 1749*

HORMONE, FOLLICLE STIMULATING
  SUPPRESSION
    GESTATIONAL TROPHOBLASTIC
     NEOPLASMS, WOMEN, 1735*

HORMONE, GROWTH
  PITUITARY TUMOR
    SUPPRESSION OF ANTERIOR PITUITARY,
     RAT, 1731*

HORMONE, STEROID
  SECRETION
    ANTIMICROTUBULAR AGENTS, MOUSE
     TUMOR CELLS, 1666*
  SULFURYLATION
    MAMMARY TUMOR, MICRO ASSAY, HUMAN,
     1758*

HYBRIDIZATION
  DNA-RNA
    AVIAN LEUKOSIS VIRUS DNA, CHICK
     EMBRYO, 1391
    MOLECULAR RELATEDNESS OF C-TYPE
     RNA VIRUSES, 1378
    MYELOMAS, MOUSE, 1722*
  MOLECULAR
    NUCLEIC ACIDS, MAEDI VIRUS, 1392
    NUCLEIC ACIDS, RNA TUMOR VIRUSES,
     1392
    NUCLEIC ACIDS, VISNA VIRUS, 1392
    RNA TUMOR VIRUSES, TAXONOMY, 1380

HYDROCARBON, POLYCYCLIC
  ARYL HYDROCARBON HYDROXYLASE INDUCTION
    CHICK EMBRYO, 1252

HYDROCARBON, POLYCYCLIC AROMATIC
  EPOXIDES
    PREPARATION AND METABOLISM, 1288*

N-HYDROXY-2-AMINOFLUORENE
  O-GLUCURONIDE CONJUGATE
    MUTAGENESIS, B.SUBTILIS, 1250
    TRANSFORMING DNA, B.SUBTILIS, 1250

N-HYDROXY-N-ACETYLAMINOFLUORENE
  TRANSACETYLASE
    RAT, 1235

N-HYDROXY-N-2-FLUORENYLACETAMIDE
  ALPHA-FETOPROTEIN
    SERUM, RAT, 1484

1'-HYDROXYSAFROLE
  HEPATOCELLULAR CARCINOMA
    RAT, 1262
  SAFROLE METABOLISM
    HEPATOCARCINOGENESIS, 1274

HYPOGLYCEMIA
  CERVICAL CARCINOMA
    IMMUNOASSAYABLE INSULIN, CASE REPORT, 1670*

IMIDAZOLE
  REGENERATION, LIMB
    NEWT, 1687*

IMMUNE RESPONSE
  ANTI-THYMOCYTE SERUM
    PARADOXICAL EFFECT, THYMUS, MOUSE, 1515*
  ANTI-TUMOR REACTIVITY
    MOUSE, 1454
  ANTIBODY-MEDIATED REGULATION
    IMMUNOGLOBULIN IGG, MOUSE, 1573*
  AUGMENTATION
    ANTIBODY ELIMINATION, RAT, 1568*
  AUTOIMMUNIZATION
    ACUTE LEUKEMIA PATIENTS, 1456
  BACILLUS CALMETTE-GUERIN
    MOLONEY SARCOMA VIRUS-INDUCED TUMOR, MOUSE, 1476
    SKIN TUMOR, MOUSE, 1475
  CANCER ETIOLOGY
    GENETICS, HUMAN, 1512*
  CARCINOGENESIS
    REVIEW, 1205
  CYTOTOXICITY
    GROSS VIRUS-INDUCED LYMPHOMA, RAT, 1489
    MURINE SARCOMA VIRUS-INDUCED TUMORS, EFFECTOR CELL, MOUSE, 1490
  IODOACETATE
    LEUKEMIC AKR MICE, 1570*
  LYMPHOCYTE
    CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1536*
  MAMMARY ADENOCARCINOMA CELLS
    SURVIVAL TIME, MOUSE, 1473
  NITROGEN MUSTARD
    RESISTANCE, YOSHIDA SARCOMA CELLS, RAT, 1483
  PHYTOHEMAGGLUTININ
    CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1535*
  PROTEIN CALORIE DEFICIENCY
    TUMOR GROWTH, MOUSE, 1481
  RADIATION, X-RAY
    LEUKEMIA CELLS, MOUSE, 1531*
  TOBACCO SMOKING
    MOUSE, 1295*
  TUMOR TRANSFER
    COMPLEMENT ACTIVITY, MOUSE, 1457
  VIRUS, MOLONEY LEUKEMIA
    DEPRESSION BY MALARIAL INFECTION, MOUSE, 1530*
  VIRUS, MOLONEY MURINE LEUKEMIA
    MOUSE, 1436

IMMUNE SURVEILLANCE
  CARCINOGENESIS
    REVIEW, 1205
  IMMUNOLOGY
    REVIEW, 1201
  INHERITED SELECTED DEFECT
    CANCER ETIOLOGY, HUMAN, 1512*

IMMUNITY
  AUTOIMMUNITY
    B CELLS, MOUSE, 1576*
  CELL-MEDIATED
    FIBROMA VIRUS-INDUCED TUMORS, RABBIT, 1486
    MOLONEY MURINE LEUKEMIA VIRUS, MOUSE, 1436
  CROSS-IMMUNITY
    SYNGENEIC TUMORS, MOUSE, 1501
  CYCLOPHOSPHAMIDE THERAPY
    3-METHYL-CHOLANTHRENE-INDUCED SARCOMA, MOUSE, 1281*
  IN VITRO ASSAY
    125I-IODODEOXYURIDINE-LABELED TARGET CELLS, MOUSE, 1470
  LEUKEMIA, ACUTE
    HUMAN, 1532*
  MAMMARY ADENOCARCINOMA CELLS
    SURVIVAL TIME, MOUSE, 1473
  PLAQUE FORMING CELL PRODUCTION
    EFFECT OF DETERGENTS, 1526*
  T CELL ACTIVATION
    IMMUNOMANIPULATION OF TUMORS, MOUSE, 1468
  TRANSPLANTATION
    HERPES-SIMPLEX VIRUS-TRANSFORMED CELLS, HAMSTER, 1403
  VIRUS, ROUS SARCOMA
    AVIAN LEUKOSIS VIRUSES, CHICKEN, 1474

IMMUNITY, CELLULAR
  LYMPHOCYTE SENSITIVITY
    PHYTOHEMAGGLUTININ, CLINICAL STUDY, 1560*
  NEPHROBLASTOMA
    CLINICAL STUDY, 1538*
  TUMOR CELL DESTRUCTION
    MACROPHAGES, RAT, 1551*

IMMUNOABSORPTION
  CELLULAR
    LYMPHOCYTE-TUMOR INTERACTION, MOUSE, 1503

IMMUNODEFICIENCY
  LEUKEMIA, ACUTE LYMPHOBLASTIC
    CHILDREN, 1517*

IMMUNODEPRESSION
  ANTILYMPHOMA ALLOGRAFT REACTIVITY
    TUMOR-ASSOCIATED FACTORS, MOUSE, 1480

- IMMUNOGENICITY
  - ENHANCEMENT
    - NORMAL AND TUMOR TISSUE, MOUSE, 1556*

- IMMUNOGLOBULIN
  - CLASS CHARACTERISTICS
    - MULTIPLE MYELOMA, CLINICAL STUDY, 1539*
  - M-COMPONENTS
    - SERUM, SOLID MALIGNANT NEOPLASM PATIENTS, 1460
  - LIGHT CHAINS
    - MULTIPLE MYELOMA, CLINICAL STUDY, 1540*
  - LOCAL PRODUCTION
    - MAMMARY CARCINOMA, HUMAN, 1450
  - MONOCLONAL F(AB)2 FRAGMENT
    - MALIGNANT LYMPHOMA, CASE REPORT, 1559*
  - TUBULAR STRUCTURES
    - HUMAN LYMPH NODE GERMINAL CENTERS, 1549*

- IMMUNOGLOBULIN IGA
  - INTRACELLULAR CRYSTALS
    - CHRONIC LYMPHOCYTIC LEUKEMIA PATIENT, 1534*
  - J-CHAIN
    - MURINE TUMOR CELLS, 1443

- IMMUNOGLOBULIN IGG
  - ANTIBODY-MEDIATED REGULATION
    - IMMUNE RESPONSE, MOUSE, 1573*
  - BIOSYNTHESIS
    - EFFECT OF 2-DEOXY-D-GLUCOSE, 1527*
  - DISTRIBUTION OF ANTIGEN FRAGMENTS
    - RNA NUCLEOTIDES, 1521*
  - INTRACELLULAR TRANSPORT
    - EFFECT OF 2-DEOXY-D-GLUCOSE, 1527*
  - SECRETION
    - EFFECT OF 2-DEOXY-D-GLUCOSE, 1527*

- IMMUNOGLOBULIN IGM
  - J-CHAIN
    - MURINE TUMOR CELLS, 1443

- IMMUNOLOGY
  - ANTITUMOR VACCINATION
    - YEAST, MOUSE, 1506*
  - CANCER HYPOTHESIS
    - VIROLOGICAL AND IMMUNOLOGICAL RELATIONSHIPS, 1346
  - IMMUNE SURVEILLANCE
    - REVIEW, 1201, 1205
  - IMMUNIZATION WITH NORMAL SYNGENEIC TISSUES
    - TUMOR INDUCTION, MOUSE, 1548*
  - INTERCELLULAR LACUNAE
    - ULTRASTRUCTURE, STIMULATED LEUKOCYTE CULTURE, 1522*
  - LONG-TERM SPONTANEOUS TUMOR INCIDENCE
    - NEONATALLY THYMECTOMIZED MICE, 1439
  - OPSONIC ACTIVITY
    - PLASMA, CARCINOMA PATIENTS, 1510*
  - TRANSPLANTATION RESISTANCE
    - FETAL ANTIGENS, HUMAN ADENOVIRUS-INDUCED TUMORS, HAMSTER, 1509*
  - TUMOR TOLERANCE
    - BLOCKING FACTORS, REVIEW, 1210
  - VIROLOGY
    - CHEMOTHERAPY, RADIOTHERAPY, REVIEW, 1220*
  - VIRUS, ROUS
    - TUMOR INDUCTION, MOUSE, 1557*

- IMMUNOSUPPRESSION
  - CANCER
    - HUMAN, 1547*
  - NEOPLASIA
    - REVIEW, 1205
  - PAPOVA VIRUS DETECTION
    - KIDNEY TRANSPLANTATION, HUMAN, 1496
  - ROSETTE FORMATION
    - T CELLS, 1513*
  - SYNTHETIC POLYPEPTIDE IMMUNOGENS
    - MURINE LEUKEMIA VIRUS, MOUSE, 1498
  - VIRUS, MURINE LEUKEMIA
    - MOUSE, 1498
  - VIRUS, POLYOMA
    - ONCOGENESIS, MOUSE, 1354

- INSULIN
  - HYPOGLYCEMIA
    - IMMUNOASSAY, CERVICAL CARCINOMA PATIENT, CASE REPORT, 1670*

- INTESTINAL CARCINOMA
  - AZOXYMETHANE
    - RAT, 1269
  - 1,2-DIMETHYLHYDRAZINE
    - VITAMIN A, RAT, 1267

- IODINE
  - RADIOACTIVE IODINE UPTAKE STUDY
    - THYROID CARCINOMA, CASE REPORT, 1306

- 5-IODO-2'-DEOXYURIDINE
  - INCORPORATION
    - SV40 DNA, 1402

- IODOACETATE
  - IMMUNE RESPONSE
    - LEUKEMIC AKR MICE, 1570*
  - SPLENIC PLAQUE FORMATION
    - LEUKEMIC AKR MICE, 1570*
  - SURVIVAL RATE
    - LEUKEMIC AKR MICE, 1570*

- 5-IODODEOXYURIDINE
  - VIRUS, MURINE LEUKEMIA
    - INDUCTION, MOUSE, 1336

- 125I-IODODEOXYURIDINE
  - LABELED ASCITES TUMOR CELLS
    - METASTASIS, MOUSE, 1684*
  - LABELED SARCOMA CELLS
    - MEASUREMENT OF TUMOR IMMUNITY, MOUSE, 1470

- IRON DEXTRAN
  - SARCOMA
    - CASE REPORTS, 1287*

- ISLET CELL CARCINOMA

- ACTH-SECRETING
  - ULTRASTRUCTURE, CASE REPORT, 1693*

KAPOSI'S SARCOMA
- LYMPHOCYTE TRANSFORMATION
  - PHYTOHEMAGGLUTININ, 1514*

KIDNEY
- C-TYPE VIRUS LOCALIZATION
  - GLOMERULONEPHRITIS, MOUSE, 1377
- METABOLISM
  - DIMETHYLNITROSAMINE-INDUCED CHANGES, RAT, 1261
- RENAL CARCINOMA
  - LIVING KIDNEY GRAFT DONOR, CASE REPORT, 1652
- RENAL CELL CARCINOMA
  - MOUSE, 1692*
- TRANSPLANTATION
  - PAPOVA VIRUS, IMMUNOSUPPRESSION, HUMAN, 1496

KIDNEY TUMOR
- N-METHYL-N-NITROSOUREA
  - RAT, 1279

LACTIC DEHYDROGENASE
- LEUKEMIA
  - SERUM, CLINICAL STUDY, 1676*

LARYNGEAL PAPILLOMA
- VIRUS ISOLATION
  - HUMAN, 1430*

LEUKEMIA
- ANTIGEN, SPLENO-THYMIC
  - FETAL THYMUS, HUMAN, 1505*
- BONE MARROW CELLS
  - PROLIFERATION, SERUM FACTOR, HUMAN, 1640
- DEVELOPMENT
  - FRIEND VIRUS COMPLEX, MOUSE, 1578
- EL-4 CELLS
  - X-IRRADIATION, IMMUNE RESPONSE, MOUSE, 1531*
- LACTIC DEHYDROGENASE
  - SERUM, CLINICAL STUDY, 1676*
- MAMMARY CARCINOMA
  - FAMILIAL OCCURRENCE, 1691*
- MISSING Y CHROMOSOME
  - HUMAN, 1663*
- MORTALITY
  - U.S.A., 1601*
- MURAMIDASE
  - SERUM, CLINICAL STUDY, 1676*
- MURAMIDASE, SERUM AND MARROW
  - CLINICAL STUDY, 1778*
- OCCUPATIONAL HAZARD
  - U.S.A., 1590
- OXYGEN CONSUMPTION
  - HUMAN LEUKOCYTES, 1661*
- PURINE METABOLISM
  - LYMPHOCYTES, HUMAN, 1797*
- REGRESSION
  - FRIEND VIRUS COMPLEX, MOUSE, 1578
- SPLENIC PLAQUE FORMATION
  - IODOACETATE, AKR MICE, 1570*
- SURVIVAL RATE
  - IODOACETATE, AKR MICE, 1570*

LEUKEMIA, ACUTE
- AUTOIMMUNITY
  - IMMUNE RESPONSE, HUMAN, 1456
- DNA SYNTHESIS
  - ERYTHROBLASTS, HUMAN, 1608
- HEMOPOIESIS
  - BONE MARROW, RAT, 1577
- IMMUNITY
  - HUMAN, 1532*
- LYMPHOCYTE BLASTOGENIC RESPONSE
  - SERUM, HUMAN, 1456

LEUKEMIA, ACUTE LYMPHOBLASTIC
- CELL CYCLE
  - KINETICS, COMPUTER MODEL, HUMAN, 1593
- IMMUNODEFICIENCY
  - CHILDREN, 1517*
- T AND B CELL
  - HUMAN, 1729*
- TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE ACTIVITY
  - HUMAN CELL LINE, 1694*

LEUKEMIA, ACUTE MYELOID
- PHYTOHEMAGGLUTININ RESPONSIVENESS
  - IMMUNOLOGICAL STUDIES, 1461
- SERUM INHIBITORY FACTORS
  - IMMUNOLOGICAL STUDIES, 1461

LEUKEMIA, ARACHNOID
- NERVOUS SYSTEM, CENTRAL
  - HISTOPATHOLOGICAL STUDY, CHILDREN, 1717*

LEUKEMIA, CHRONIC GRANULOCYTIC
- CENTRAL HYPOVENTILATION
  - CASE REPORT, 1677*

LEUKEMIA, CHRONIC LYMPHATIC
- ANTIGEN, TUMOR-SPECIFIC
  - HUMAN, 1449
- CYCLIC AMP
  - CLINICAL STUDY, 1796*
- LYMPHOCYTES
  - IMMUNE RESPONSE, HUMAN, 1536*
- PHYTOHEMAGGLUTININ
  - LYMPHOCYTE RESPONSE, HUMAN, 1535*
- REIDER CELL MICROTUBULES
  - ULTRASTRUCTURE, 1794*

LEUKEMIA, CHRONIC LYMPHOCYTIC
- ANTITHYMOCYTE ANTISERUM REACTIVITY
  - LYMPHOCYTES, HUMAN, 1491
- IMMUNOGLOBULIN IGA
  - INTRACELLULAR CRYSTALS, CASE REPORT, 1534*
- LYMPHOCYTE LYSOSOMES
  - ULTRASTRUCTURE, CYTOCHEMISTRY, HUMAN, 1653
- LYSOSOMAL ENZYMES
  - ULTRASTRUCTURE, CYTOCHEMISTRY, HUMAN, 1653

LEUKEMIA, CHRONIC MYELOCYTIC
- ALKALINE PHOSPHATASE
  - HUMAN, 1612
- BLASTIC CRISIS REMISSION
  - CYTOGENETIC STUDIES, HUMAN, 1757*

- LEUKEMIA, CHRONIC MYELOGENOUS
  - CYCLIC AMP
    - CLINICAL STUDY, 1796*
  - GRANULOCYTE
    - THROMBOPLASTIC ACTIVITY, 1788*

- LEUKEMIA, CHRONIC MYELOID
  - DNA SYNTHESIS
    - ERYTHROBLASTS, HUMAN, 1608

- LEUKEMIA, LYMPHOBLASTIC
  - PHOSPHATE DEPLETION
    - GLUCOCORTICOID-INDUCED HYPERPHOSPHATEMIA, CASE REPORT, 1773*

- LEUKEMIA, LYMPHOCYTIC
  - PLATELET KINETICS
    - ADENOSINE TRIPHOSPHATASE ACTIVITY, MOUSE, 1428*

- LEUKEMIA, MYELOID
  - INCIDENCE
    - BLOOD LOSS, X-IRRADIATED RATS, 1312*

- LEUKEMIA, MYELOMONOCYTIC
  - VIRUS, C-TYPE RNA
    - ULTRASTRUCTURE, MOUSE TUMOR CELLS, 1323

- LEUKEMOGENESIS
  - VIRUS, RAUSCHER LEUKEMIA
    - RIFAMYCIN, DNA POLYMERASE, MOUSE, 1383

- LEUKOCYTE
  - HETEROPHILE RESPONSE
    - EPSTEIN-BARR VIRUS, MONKEY, 1343
  - INTERCELLULAR LACUNAE
    - ULTRASTRUCTURE, STIMULATED CELL CULTURES, 1522*
  - OXYGEN CONSUMPTION
    - BLOOD, LYMPH NODES, LEUKEMIA PATIENTS, 1661*
  - PERMEABILITY TO WATER
    - HEATS OF ACTIVATION, LEUKEMIC CELLS, 1766*
  - PREVENTION OF CELL-TO-CELL SPREAD
    - HERPES SIMPLEX VIRUS, RABBIT, 1340

- LICHEN PLANUS
  - SQUAMOUS CELL CARCINOMA
    - TONGUE, CASE REPORT, 1587*

- LIGAND
  - BINDING SITE
    - MYELOMA PROTEIN, MOUSE, 1447

- LIPID
  - CONTENT FLUCTUATION
    - EHRLICH CARCINOMA CELLS, 1791*
  - METABOLISM
    - HEPATOMA, RAT, 1721*

- LIPOGENESIS
  - HIGH CARBOHYDRATE DIET
    - MAMMARY ADENOCARCINOMA, RAT, 1660*

- LIVER
  - CYTOCHROME P-448 INDUCTION
    - POLYCHLORINATED BIPHENYLS, RAT, 1298*
  - DNA-DEPENDENT DNA POLYMERASE
    - HEPATOMA, CHARACTERIZATION, RAT, 1638
  - DNA METHYLATION
    - N,N-DIMETHYLNITROSAMINE, RAT, 1239
  - HYPERPLASTIC NODULES
    - CHOLESTEROL SYNTHESIS, RAT, 1646
  - ION-TRANSPORTING ACTIVITY
    - HEPATOMA, RAT, 1636
  - METABOLISM
    - FEEDBACK CONTROL, ETHIONINE-FED RATS, 1583
  - METASTASIS
    - LYMPHOMA, ULTRASTRUCTURE, SURFACTANT-TREATED HAMSTERS, 1581
  - PROTEIN, NUCLEAR
    - HEPATOMA, POLYACRYLAMIDE GEL ELECTROPHORESIS, RAT, 1678*
  - REGENERATION
    - DNA SYNTHESIS STIMULATING FACTOR, RAT, 1616
  - SPONTANEOUS TUMOR DEVELOPMENT
    - MASTOMYS, 1632
  - TRANSFER RNA METHYLTRANSFERASE
    - NOVIKOFF HEPATOMA, RAT, 1669*

- LUNG
  - ADENOMA
    - LONG-TERM EFFECT OF DDT, MOUSE, 1232, 1238
  - METASTASIS
    - BASAL CELL CARCINOMA, CASE REPORT, 1659*
    - RETICULUM CELL SARCOMA, CASE REPORT, 1659*
    - THYROID CARCINOMA, CASE REPORT, 1659*
  - METASTASIS PREVENTION
    - PLASMA FACTOR, RAT, 1528*
  - RETICULUM CELL SARCOMA
    - RARE EARTH METAL DUST, RAT, 1304

- LUNG CANCER
  - ASBESTOS
    - MESOTHELIOMA, HUMAN, REVIEW, 1229*
  - CHLOROMETHYL METHYL ETHER
    - OCCUPATIONAL HAZARD, 1299*
  - HEMOGLOBIN MALMOE
    - FIBROSIS, FAMILIAL OCCURRENCE, CASE REPORTS, 1795*
  - MORTALITY
    - U.S.A., 1601*
  - TOBACCO SMOKING
    - HISTOPATHOLOGY, HUMAN, 1582
    - HUMAN, 1598*

- LUPUS ERYTHEMATOSUS
  - ANTIBODIES TO CELL ORGANOIDS
    - SERA, PATIENTS, 1543*

- LYMPH NODE
  - GERMINAL CENTERS
    - TUBULAR STRUCTURES, IMMUNOGLOBULINS, HUMAN, 1549*

- OXYGEN CONSUMPTION
  - LEUKOCYTES, LEUKEMIA PATIENTS, 1661*

- LYMPH NODE CELL
  - CYTOTOXICITY
    - HEPATOMA, IMMUNITY, RAT, 1466

- LYMPH NODE, CERVICAL
  - METASTASIS
    - UNKNOWN PRIMARY CANCER, CLINICAL STUDY, 1673*

- LYMPH NODE, REGIONAL
  - METHYLCHOLANTHRENE-INDUCED SARCOMA
    - IMMUNE RESPONSE, MOUSE, 1454

- LYMPHANGIOMYOMA
  - HISTOGENESIS
    - ULTRASTRUCTURE, CASE REPORTS, 1579

- LYMPHATIC SYSTEM
  - PERMEATION
    - REVIEW, 1203

- LYMPHCYTE
  - PHYTOHEMAGGLUTININ
    - CELLULAR IMMUNITY, CLINICAL STUDY, 1560*

- LYMPHOBLAST
  - VIRUS, EPSTEIN-BARR
    - SUPERINFECTION, HUMAN CELL LINES, 1367

- LYMPHOCYTE
  - ANTITHYMOCYTE ANTISERUM REACTIVITY
    - CHRONIC LYMPHOCYTIC LEUKEMIA, HUMAN, 1491
  - B CELL
    - AUTOIMMUNITY, MOUSE, 1576*
  - BLASTOGENIC RESPONSE
    - AUTOIMMUNIZATION, ACUTE LEUKEMIA PATIENTS, 1456
  - CYTOTOXIC
    - SERUM BLOCKING FACTORS, HUMAN, 1542*
  - IMMUNE RESPONSE
    - CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1536*
  - IN VITRO CULTURE
    - PLASMA-FREE MEDIUM, HUMAN, 1520*
  - LEUKEMIA, CHRONIC LYMPHOCYTIC
    - LYSOSOMAL ENZYME ACTIVITY, HUMAN, 1653
    - ULTRASTRUCTURE, CYTOCHEMISTRY, HUMAN, 1653
  - MEMBRANE RECEPTOR-CYTOPLASMIC INTERACTION
    - CONCANAVALIN A, 1453
  - BETA2-MICROGLOBULIN PRODUCTION
    - HUMAN CELL LINE, 1572*
  - PURINE METABOLISM
    - LEUKEMIC CELLS, HUMAN, 1797*
  - T AND B CELL
    - ACUTE LYMPHOBLASTIC LEUKEMIA, HUMAN, 1729*
    - HAPTEN-REACTIVE, 1529*
    - MAREK'S DISEASE LYMPHOMAS, CHICKEN, 1416*
  - T CELL
    - RECONSTITUTION FACTOR, MOUSE, 1499
  - T CELL ACTIVATION
    - IMMUNOMANIPULATION OF TUMORS, MOUSE, 1468
  - TRANSFORMATION
    - AUTOLOGOUS HUMAN LYMPHOBLASTS, 1500
    - CONCANAVALIN A BINDING, HUMAN, 1668*
    - PHYTOHEMAGGLUTININ, KAPOSI'S SARCOMA, 1514*
    - PHYTOHEMAGGLUTININ, MALIGNANT DISEASE PATIENTS, 1518*
    - PHYTOHEMAGGLUTININ, PLASMA-FREE MEDIUM, HUMAN, 1520*
    - PHYTOHEMAGGLUTININ, SERUM INHIBITORY FACTORS, LEUKEMIA PATIENTS, 1461
    - WHEAT GERM AGGLUTININ BINDING, HUMAN, 1668*
  - TUMOR-HOST RELATIONSHIP
    - ASCITES CELL, MOUSE, 1471
  - TUMOR INTERACTION
    - CELLULAR IMMUNOABSORPTION, MOUSE, 1503

- LYMPHOCYTE RESPONSE
  - VIRUS, MOLONEY SARCOMA
    - TUMOR, MOUSE, 1339

- LYMPHOMA
  - C-TYPE RNA VIRUS EXPRESSION
    - SPONTANEOUS TUMORS, MOUSE, 1493
  - GROWTH
    - GRAFFI LEUKEMIA VIRUS, MOUSE, 1329
  - 3H-THYMIDINE LABELING
    - AUTORADIOGRAPHY, 1580
  - HERPESVIRUS SAIMIRI
    - BURKITT'S LYMPHOMA, HUMAN, MONKEY, 1349
  - MAREK'S DISEASE
    - T AND B CELL ANALYSIS, CHICKEN, 1416*
  - METASTASIS
    - LIVER, ULTRASTRUCTURE, SURFACTANT-TREATED HAMSTERS, 1581
  - N-METHYL-N-NITROSOUREA
    - RAT, 1279
  - NON-HODGKIN'S
    - CLINICOPATHOLOGIC STUDY, REVIEW, 1225*
  - REJECTION
    - MACROPHAGES, TUMOR GROWTH, RAT, 1562*
  - VIRAL INDUCTION
    - GRAFT-VS-HOST REACTION, MOUSE, 1485
  - VIRUS, GROSS
    - IMMUNE CYTOTOXICITY, RAT, 1489
  - VIRUS, RNA TUMOR
    - HUMAN, 1410

- LYMPHOMA, HISTIOCYTIC
  - LEUKEMIC PHASE
    - ULTRASTRUCTURE, CLINICAL STUDY, 1719*

- LYMPHOMA, MALIGNANT
  - ALKALINE PHOSPHATASE ACTIVITY
    - CLINICAL STUDY, 1779*
  - DDT
    - LONG-TERM EFFECT, MOUSE, 1238
  - HODGKIN' DISEASE
    - INCIDENCE, MINNESOTA, 1605*
  - MONOCLONAL IMMUNOGLOBULIN F(AB)2 FRAGMENT
    - CASE REPORT, 1559*
- LYMPHOMA, NODULAR
  - PROTEINACEOUS PRECIPITATE
    - CLINICAL STUDY, 1718*
- LYMPHORETICULAR CELL
  - ULTRASTRUCTURE
    - NON-LYMPHOID HUMAN NEOPLASMS, 1770*
- LYMPHOSARCOMA
  - ACQUIRED C1 INHIBITOR DEFICIENCY
    - HUMAN, 1558*
  - ANTIGEN, THETA
    - MOUSE, 1546*
  - PORCINE AND OVINE
    - REVIEW, 1228*
- LYSOZYME
  - ACTIVITY
    - NITROBLUE-TETRAZOLIUM REDUCTION, LEUKEMIC CELLS, HUMAN, 1736*
- MACROGLOBULINEMIA
  - MELPHALAN TREATMENT
    - ERYTHROLEUKEMIA DEVELOPMENT, CASE REPORT, 1278
- MACROPHAGE
  - ALLOGRAFT REJECTION
    - FIBROSARCOMA CELLS, MOUSE, 1544*
  - GROWTH, TUMOR
    - REJECTION OF ALLOGENEIC LYMPHOMA, RAT, 1562*
  - NONIMMUNOLOGIC RECOGNITION
    - TARGET CELL FACTORS, CONTACT INHIBITION, MOUSE, 1446
  - TUMOR CELL DESTRUCTION
    - IMMUNITY, RAT, 1551*
- MALATE
  - OXIDATIVE DECARBOXYLATION
    - MITOCHONDRIA, ASCITES TUMOR CELLS, MOUSE, 1723*
- MAMMARY CARCINOMA
  - ANTIGEN, HL-A
    - HUMAN, 1492
  - ANTIGEN, TRANSPLANTATION
    - SYNGENEIC MICE, 1541*
  - BRAIN TUMOR
    - FAMILIAL OCCURRENCE, 1691*
  - CULTIVATION IN VITRO
    - HUMAN CELL LINE, 1686*
  - ENVIRONMENTAL FACTORS
    - HUMAN, 1594
  - HISTOLOGY
    - BOSTON AND TOKYO, 1699*
    - INTERNATIONAL DIFFERENCES, 1697*
  - IMMUNGLOBULIN PRODUCTION
    - HUMAN, 1450
  - LEUKEMIA
    - FAMILIAL OCCURRENCE, 1691*
  - METASTASIS
    - EYELID, CLINICAL STUDY, 1671*
  - PAPILLOMATOSIS, FLORID SUBAREOLAR DUCT
    - CASE REPORTS, 1584
  - SARCOMA, SOFT TISSUE
    - FAMILIAL OCCURRENCE, 1691*
  - SEQUENTIAL MORPHOLOGICAL AND HISTOLOGICAL CHANGES
    - RECIPROCAL CROSS MOUSE STRAINS, 1786*
  - SPONTANEOUS
    - SEQUENTIAL ENZYME CHANGES, RECIPROCAL CROSS MOUSE STRAINS, 1785*
  - STEROID SULFURYLATION
    - MICRO ASSAY, HUMAN, 1758*
  - SURVIVAL
    - INTERNATIONAL DIFFERENCES, 1697*
- MAMMARY GLAND
  - ADENOCARCINOMA
    - C-TYPE VIRUS PARTICLES, RAT, 1426*
    - LIPOGENESIS, HIGH CARBOHYDRATE DIET, RAT, 1660*
  - ADENOCARCINOMA CELLS
    - IMMUNE RESPONSE, MOUSE, 1473
  - MELANOMA, MALIGNANT
    - METASTASIS, CASE REPORTS, 1672*
- MAMMARY TUMOR
  - ANDROGEN
    - DEPENDENCE, HUMAN, 1623
  - BENIGN BREAST LESIONS
    - ANDROGEN LEVELS, HUMAN, 1651
  - DIETHYLSTILBESTROL
    - INHIBITION BY PROGESTERONE, RAT, 1302
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - ATPASE ACTIVITY, RAT, 1254
    - FRUCTOSE-1,6-DIPHOSPHATASE, RAT, 1248
    - HORMONE DEPENDENCE, RAT, 1259
    - TRANSPLANTATION, RAT, 1259
    - UTPASE ACTIVITY, RAT, 1254
  - EPITHELIAL CELLS
    - CHANGES IN THE SURFACE COATS, HUMAN, 1645
  - N-METHYL-N-NITROSOUREA
    - RAT, 1237
  - N-NITROQUINOLINE-N-OXIDE
    - RAT, 1237
  - RADIATION, GAMMA
    - INHIBITION BY PROGESTERONE, RAT, 1302
- MAREK'S DISEASE
  - LYMPHOMA
    - T AND B CELL ANALYSIS, CHICKEN, 1416*
- MECKEL'S DIVERTICULUM
  - CARCINOMA
    - CASE REPORT, 1716*
- MEDULLARY CARCINOMA

  - PARATHYROID
    - ARGENTAFFIN CELLS, C-CELLS, HUMAN, 1751*
  - THYROID
    - ACTH AND CALCITONIN SECRETION, CASE REPORT, 1749*
    - ARGENTAFFIN CELLS, C-CELLS, HUMAN, 1751*
    - CLINICOPATHOLOGIC STUDY, 1674*
    - HUMAN, REVIEW, 1231*

- MEDULLOBLASTOMA
  - INCIDENCE
    - BLOOD GROUP CORRELATIONS, STATISTICAL STUDY, 1753*

- MELANOMA
  - B16 CELLS
    - TRANSPLANTIBILITY, GROWTH PATTERNS, 1462
  - RADIATION, X-RAY
    - MELANOSOMAL TYROSINASE ACTIVITY, MOUSE, 1311*
  - TYROSINASE ACTIVITY
    - CULTURED CELLS, MOUSE, 1763*

- MELANOMA, HUMAN
  - TRANSPLANTATION
    - METASTASIS, THYMUS-DEFICIENT MOUSE, 1688*

- MELANOMA, MALIGNANT
  - BACILLUS CALMETTE-GUERIN
    - HUMAN, 1575*
  - ESTROGEN
    - DEPENDENCE, HUMAN, 1626
  - HL-A DEFICIENCY
    - CLINICAL STUDY, 1545*
  - METASTASIS
    - MAMMARY GLAND, CASE REPORTS, 1672*
  - SKIN
    - CLINICAL STUDY, 1784*
  - VIRUS, RADIAL-TYPE
    - ULTRASTRUCTURE, HAMSTER CELLS, 1344

- MELANOSOMAL TYROSINASE
  - ACTIVITY
    - X-IRRADIATION, MOUSE MELANOMAS, 1311*

- MELPHALAN
  - ERYTHROLEUKEMIA DEVELOPMENT
    - PRIMARY MACROGLOBULINEMIA PATIENT, CASE REPORT, 1278

- MENINGEOMA
  - GROWTH
    - SKULL, CASE REPORTS, 1604*

- MENINGIOMA
  - ENDOPLASMIC RETICULUM
    - INTERCISTERNAL STRUCTURES, ULTRASTRUCTURE, HUMAN, 1790*
  - INCIDENCE
    - BLOOD GROUP CORRELATIONS, STATISTICAL STUDY, 1753*
  - INVASION OF THE FACIAL BONES
    - CASE REPORTS, 1657*

- MESOTHELIOMA
  - ASBESTOS
    - HUMAN, 1258
  - LUNG CANCER
    - ASBESTOS, HUMAN, REVIEW, 1229*
  - PRIMARY MALIGNANT
    - PATHOLOGIC STUDY, REVIEW, 1226*

- METABOLISM
  - AFLATOXIN B1
    - TOXICITY, BIRD, MAMMAL, REVIEW, 1218
  - P-DIMETHYLAMINOAZOBENZENE
    - AGE, RAT LIVER, 1233
    - 3-METHYLCHOLANTHRENE, RAT LIVER, 1233
  - KIDNEY
    - DIMETHYLNITROSAMINE-INDUCED CHANGES, RAT, 1261
  - LIPID
    - HEPATOMA, RAT, 1721*
  - LIVER
    - FEEDBACK CONTROL, ETHIONINE-FED RATS, 1583
  - NUCLEIC ACID
    - DIMETHYLNITROSAMINE, KIDNEY, RAT, 1261
  - PROTEIN
    - DIMETHYLNITROSAMINE, KIDNEY, RAT, 1261
  - PURINE
    - HUMAN LYMPHOCYTES AND LEUKEMIC CELLS, 1797*
  - SAFROLE
    - HEPATOCARCINOGENESIS, 1274
  - TRYPTOPHAN
    - BLADDER CANCER PATIENTS, CLINICAL STUDY, 1647

- METALS
  - ION INTERACTION
    - CONCANAVALIN A, 1574*
  - ZINC
    - MALIGNANT DISEASE PATIENTS, 1283*

- METALS, RARE EARTH
  - DUST
    - RETICULUM CELL SARCOMA OF LUNG, RAT, 1304

- METASTASIS
  - GALLIUM-67 LABELED ASCITES TUMOR CELLS
    - MOUSE, 1684*
  - 125I-IODODEOXYURIDINE LABELED ASCITES TUMOR CELLS
    - MOUSE, 1684*
  - LUNG
    - BASAL CELL CARCINOMA, CASE REPORT, 1659*
    - RETICULUM CELL SARCOMA, CASE REPORT, 1659*
    - THYROID CARCINOMA, CASE REPORT, 1659*
  - LYMPH NODE, CERVICAL
    - UNKNOWN PRIMARY CANCER, CLINICAL STUDY, 1673*
  - LYMPHATIC SYSTEM
    - REVIEW, 1203
  - LYMPHOMA

- LIVER, ULTRASTRUCTURE, SURFACTANT-TREATED HAMSTERS, 1581
- MAMMARY CARCINOMA
  - EYELID, CLINICAL STUDY, 1671*
- MECHANISMS
  - SYNGENEIC AND AUTOCHTHONOUS TUMOR-HOST SYSTEMS, 1567*
- MELANOMA, MALIGNANT
  - MAMMARY GLAND, CASE REPORTS, 1672*
- PLATELET-TUMOR CELL INTERACTIONS
  - MALIGNANT TUMORS, MOUSE, 1630
- PREVENTION OF LUNG METASTASIS
  - PLASMA FACTOR, RAT, 1528*
- THROMBOCYTOPENIA
  - MALIGNANT TUMORS, MOUSE, 1630
- TRANSPLANTED HUMAN MELANOMAS
  - THYMUS-DEFICIENT MOUSE, 1688*

3-METHYL-CHOLANTHRENE
- SARCOMA
  - IMMUNITY, CYCLOPHOSPHAMIDE THERAPY, MOUSE, 1281*

2'-METHYL-4-DIMETHYLAMINOAZOBENZENE
- ALPHA-FETOPROTEIN
  - SERUM, RAT, 1484

3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
- ANTIGEN, CELL SURFACE
  - LIVER CELLS, RAT, 1459
- ALPHA-FETOPROTEIN
  - SERUM, RAT, 1484

N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
- DNA TRANSCRIPTION
  - TRANSFORMED CELLS, HAMSTER, 1266
- STOMACH CANCER
  - RESISTANCE, RAT, 1257

N-METHYL-N'-NITRO-NITROSOGUANIDINE
- CHROMOSOME ABERRATIONS
  - XERODERMA PIGMENTOSUM CELLS, 1260

N-METHYL-N-NITROSOUREA
- ANTIGEN, CELL SURFACE
  - LIVER CELLS, RAT, 1459
- DNA
  - NEURONAL AND GLIAL CELLS, RAT, 1272
- INJECTION SITE TUMORS
  - RAT, 1237
- KIDNEY TUMOR
  - RAT, 1279
- LYMPHOMA
  - RAT, 1279
- MAMMARY TUMOR
  - RAT, 1237
- SACCHARIN
  - BLADDER CARCINOMA, RAT, 1242
- TUMORIGENESIS
  - RAT, 1279

9-METHYLANTHRACENE
- HISTONE BINDING, 1276

METHYLATION
- DNA
  - N,N-DIMETHYLNITROSAMINE, RAT LIVER, 1239
- DNA, SATELLITE
  - NORMAL AND NEOPLASTIC CELL CULTURES, MOUSE, 1733*

METHYLAZOXYMETHANOL ACETATE
- RNA SYNTHESIS INHIBITION
  - RAT, 1249

METHYLCHOLANTHRENE
- INDUCED TRANSPLANTED SARCOMA
  - HOST IMMUNITY, MOUSE, 1454
- SARCOMA
  - CROSS-REACTING EMBRYONIC ANTIGEN, RAT, 1497

3-METHYLCHOLANTHRENE
- ADENOVIRUS TRANSFORMATION
  - HAMSTER CELLS, 1331
- ANTIGEN, CELL SURFACE
  - LIVER CELLS, RAT, 1459
- AZO-DYE CARCINOGENESIS
  - LIVER, RAT, 1233
- P-DIMETHYLAMINOAZOBENZENE METABOLISM
  - LIVER, RAT, 1233
- TRANSFORMATION
  - EMBRYO CELLS, RAT, 1263

20-METHYLCHOLANTHRENE
- BINDING
  - EPIDERMAL SUBCELLULAR FRACTIONS, MOUSE, 1264
- PHOSPHOLIPID COMPOSITION CHANGES
  - ENDOPLASMIC RETICULUM, LIVER, RAT, 1273
- TUMOR INDUCTION
  - IMMUNIZATION WITH NORMAL SYNGENEIC TISSUES, MOUSE, 1548*

METHYLENE DIMETHANESULPHONATE
- RESISTANCE
  - TRANSFERABLE RESISTANCE FACTOR, YOSHIDA SARCOMA CELLS, 1617

METHYLNITROSOUREA
- C-TYPE VIRUS ACTIVATION
  - BONE MARROW, MOUSE, 1280
  - THYMUS, MOUSE, 1280

METHYLTRANSFERASE
- RNA, TRANSFER
  - NORMAL LIVER AND NOVIKOFF HEPATOMA, RAT, 1669*

BETA2-MICROGLOBULIN
- PRODUCTION
  - HUMAN LYMPHOCYTE LINES, 1572*

MICROSOME
- HEPATOMA, MORRIS
  - CHOLESTEROL CONTENT, 1789*
  - PHOSPHOLIPID COMPOSITION, 1789*

MICROSPORIDIOSIS
- ADENOCARCINOMA OF THE PANCREAS
  - CASE REPORT, 1656*

MILK
- MAMMARY TUMOR VIRUS DEGRADATION
  - MOUSE, 1361

- MITOCHONDRIA
  - DNA
    - HUMAN TUMORS, 1641
  - INTRANUCLEAR
    - ULTRASTRUCTURE, HODGKIN'S DISEASE PATIENTS, 1755*
  - ISOLATION OF VIROSOMES
    - ULTRASTRUCTURE, ROUS SARCOMA CELLS, CHICKEN, 1431*
  - KETONE BODY OXIDATION
    - HEPATOMA, MOUSE, 1762*
  - OXIDATIVE DECARBOXYLATION OF MALATE
    - ASCITES TUMOR CELLS, MOUSE, 1723*
  - ALPHA-OXOGLUTARATE TRANSPORT
    - ASCITES TUMOR CELLS, 1771*
  - RADIATION, UV
    - ALTERATION OF OXIDATIVE PHOSPORYLATION, RAT LIVER, 1310*
    - PROTEIN STATE CHANGES, RAT LIVER, 1310*
  - SUBVIRAL ONCOGENIC PARTICLES
    - ULTRASTRUCTURE, ROUS SARCOMA CELLS, CHICKEN, 1431*

- MITOSIS
  - AUTOREGULATION
    - NEOPLASIA, MATHEMATICAL MODEL, 1629
  - CROTON OIL
    - BLUE-GREEN BACTERIA, 1296*
  - MITOTIC NESTS
    - AMPHIBIAN TISSUE CULTURE, 1681*
    - HUMAN VAGINAL SMEARS, 1681*

- N-MONOMETHYL-4-AMINOAZOBENZENE
  - DNA BINDING
    - IN VITRO REACTIONS, 1268
  - RNA BINDING
    - IN VITRO REACTIONS, 1268

- MORPHOLINE
  - NITROSATION
    - N-NITROSOMORPHOLINE FORMATION, 1284*

- MORTALITY
  - CANCER
    - URANIUM MILL WORKERS, U.S.A., 1588
  - COLON CARCINOMA
    - U.S.A., 1601*
  - LEUKEMIA
    - U.S.A., 1601*
  - LUNG CANCER
    - U.S.A., 1601*
  - NASOPHARYNGEAL CARCINOMA
    - CALIFORNIA, 1595*
  - OVARIAN CARCINOMA
    - U.S.A., 1601*
  - PANCREAS CARCINOMA
    - U.S.A., 1601*
  - PROSTATE CARCINOMA
    - U.S.A., 1601*
  - RECTAL CARCINOMA
    - U.S.A., 1601*
  - STOMACH CANCER
    - U.S.A., 1601*
  - UTERINE CANCER
    - U.S.A., 1601*

- MUCOPOLYSACCHARIDE
  - CARCINOGENESIS
    - REVIEW, 1207
  - CONTENT
    - SV40-TRANSFORMED KIDNEY CELLS, MONKEY, 1382
  - GROWTH, TUMOR
    - MOUSE, 1494

- MUCOPOLYSACCHARIDE, ACID
  - ASTROCYTOMA
    - MYELIN SHEATH CELLS, 1742*
  - NEURILEMMOMA
    - MYELIN SHEATH CELLS, 1742*
  - OLIGODENDROGLIOMA
    - MYELIN SHEATH CELLS, 1742*

- MURAMIDASE
  - BONE MARROW
    - LEUKEMIA, CLINICAL STUDY, 1778*
  - LEUKEMIA
    - SERUM, CLINICAL STUDY, 1676*
  - SERUM
    - LEUKEMIA, CLINICAL STUDY, 1778*

- MUSTARD OIL
  - CARCINOGENESIS
    - MOUSE, 1246

- MUTAGENESIS
  - BENZIMIDAZOLE
    - SALMONELLA TYPHIMURIUM, 1285*
  - BENZOYL-L-ARGININEAMIDE
    - E.COLI, 1253
    - S.TYPHIMURIUM, 1253
  - 7-BROMOMETHYL-12-METHYLBENZ(A) ANTHRACENE
    - HAMSTER CELLS, 1234
  - 7-BROMOMETHYLBENZ(A)ANTHRACENE
    - HAMSTER CELLS, 1234
  - GENETIC RECOMBINATION
    - REVIEW, 1230*
  - O-GLUCURONIDE CONJUGATE OF N-HYDROXY-2-AMINOFLUORENE, 1250

- MUTANT
  - VIRUS, ROUS SARCOMA
    - BIOLOGICAL AND PHYSICAL PROPERTIES, 1405

- MYELOMA
  - NUCLEIC ACID
    - HYBRIDIZATION STUDIES, MOUSE, 1722*
  - PROTEIN
    - LIGAND BINDING SITES, MOUSE, 1447

- MYELOMA, MULTIPLE
  - IMMUNOCHEMISTRY
    - THERAPEUTICAL FEATURES, 1780*
  - IMMUNOGLOBULIN CLASS CHARACTERISTICS
    - CLINICAL STUDY, 1539*
  - LIGHT CHAINS
    - CLINICAL STUDY, 1540*

- MYOMA
  - UTERUS
    - GLYCOGEN PHOSPHORYLASE B ACTIVITY, HUMAN, 1777*

- NASOPHARYNGEAL CARCINOMA
  - ANTIBODIES TO CELL ORGANOIDS
    - SERA, PATIENTS, 1543*
  - MORTALITY
    - CALIFORNIA, 1595*
  - VIRUS, EPSTEIN-BARR
    - ETIOLOGY, REVIEW, 1219

- NDPASE
  - ACTIVITY
    - CEREBRAL TUMORS, HUMAN, 1748*

- NECK
  - CARCINOMA
    - ASBESTOS EXPOSURE, HUMAN, 1290*
  - CERVICAL LYMPH NODE METASTASIS
    - UNKNOWN PRIMARY CANCER, CLINICAL STUDY, 1673*

- NEOCARZINOSTATIN
  - VIRUS, EPSTEIN-BARR
    - ULTRASTRUCTURE, BURKITT'S LYMPHOMA CELLS, 1425*

- NEOPLASIA
  - IMMUNOSUPPRESSION
    - REVIEW, 1205
  - MITOTIC AUTOREGULATION
    - MATHEMATIC MODEL, 1629

- NEOPLASMS
  - NON-LYMPHOID HUMAN
    - LYMPHORETICULAR CELLS, ULTRASTRUCTURE, 1770*

- NEOPLASMS, MALIGNANT
  - ANTIGEN, CARCINOEMBRYONIC
    - QUANTITATIVE STUDY, HUMAN, 1465
  - ESOPHAGEAL CARCINOMA
    - INCIDENCE, S.AFRICA, 1592
  - IMMUNOGLOBULINS, MONOCLONAL
    - HUMAN, 1460
  - SKIN
    - CLINICAL STUDY, 1784*

- NEPHROBLASTOMA
  - IMMUNITY, CELLULAR
    - CLINICAL STUDY, 1538*

- NERVOUS SYSTEM
  - SPECIFIC PROTEIN PRODUCTION
    - HUMAN NEUROBLASTOMA CELL CULTURE, 1554*

- NERVOUS SYSTEM, CENTRAL
  - GRANULAR CELL TUMOR
    - CASE REPORTS, 1760*
  - LEUKEMIA, ARACHNOID
    - HISTOPATHOLOGICAL STUDY, CHILDREN, 1717*

- NEURAMINIDASE
  - SIALIC ACID RELEASE
    - NORMAL AND MALIGNANT CELLS, MOUSE, 1644

- NEURILEMMOMA
  - MUCOPOLYSACCHARIDE, ACID
    - MYELIN SHEATH CELLS, 1742*

- NEUROBLASTOMA
  - ANTIGEN, CELL-SURFACE
    - MOUSE, 1472
  - NERVOUS SYSTEM-SPECIFIC PROTEIN PRODUCTION
    - HUMAN CELL CULTURE, 1554*

- NITRILOTRIACETIC ACID
  - DIET
    - NITROSAMINE FORMATION, RAT, 1289*

- NITROGEN MUSTARD
  - DNA CROSS-LINK MEASUREMENT
    - LYMPHOMA CELLS, MOUSE, 1251
  - RESISTANCE
    - IMMUNOSENSITIVITY, YOSHIDA SARCOMA CELLS, RAT, 1483

- N-NITROQUINOLINE-N-OXIDE
  - INJECTION SITE TUMORS
    - RAT, 1237
  - MAMMARY TUMOR
    - RAT, 1237

- 4-NITROQUINOLINE-1-OXIDE
  - CHROMOSOME ABERRATIONS
    - XERODERMA PIGMENTOSUM CELLS, 1260
  - VIRUS, ADENO 12
    - CHROMOSOME ABERRATIONS, HAMSTER CELLS, 1363
    - DNA REPAIR SYNTHESIS, HAMSTER CELLS, 1363
    - TRANSFORMATION, HAMSTER CELLS, 1363
  - VIRUS, ADENO SA7
    - CHROMOSOME ABERRATIONS, HAMSTER CELLS, 1363
    - DNA REPAIR SYNTHESIS, HAMSTER CELLS, 1363
    - TRANSFORMATION, HAMSTER CELLS, 1363

- NITROSAMINES
  - FORMATION
    - NITRILOTRIACETIC ACID, RAT, 1289*

- N-NITROSOMORPHOLINE
  - FORMATION
    - NITROSATION OF MORPHOLINE, 1284*

- NUCLEIC ACID
  - DIETHYL PYROCARBONATE
    - REACTION PRODUCTS, 1244
  - METABOLISM
    - DIMETHYLNITROSAMINE, KIDNEY, RAT, 1261
  - MYELOMA
    - HYBRIDIZATION STUDIES, MOUSE, 1722*
  - VIRUS, SV40-LIKE
    - ISOLATION, PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY PATIENT, 1406

- OCCUPATIONAL HAZARD
  - ASBESTOS
    - HUMAN, 1258
  - BENZIDINE
    - URINARY BLADDER TUMOR, 1277

  CANCER MORTALITY
    URANIUM MILL WORKERS, U.S.A., 1588
  LEUKEMIA
    U.S.A., 1590
  LUNG CANCER
    CHLOROMETHYL METHYL ETHER WORKERS, 1299*

ODONTOGENIC TUMOR
  AMYLOID CONTENT
    ULTRASTRUCTURE, HUMAN, 1792*

OIL
  ARGEMONE
    CARCINOGENESIS, MOUSE, 1246
  CROTON
    MITOSIS, BLUE-GREEN BACTERIA, 1296*
  GROUND-NUT
    CARCINOGENESIS, MOUSE, 1246
  MUSTARD
    CARCINOGENESIS, MOUSE, 1246
  PARAFFIN
    EFFECT OF INSTALLATION IN HUMAN TISSUES, 1286*

OLIGODENDROGLIOMA
  MUCOPOLYSACCHARIDE, ACID
    MYELIN SHEATH CELLS, 1742*
  NDPASE ACTIVITY
    HISTOCHEMISTRY, HUMAN, 1748*
  TTPASE ACTIVITY
    HISTOCHEMISTRY, HUMAN, 1748*

ONCOGENESIS
  ONTOGENESIS
    BIOLOGICAL CORRELATION, 1607

ONTOGENESIS
  ONCOGENESIS
    BIOLOGICAL CORRELATION, 1607

OSTEOSARCOMA
  ALKALINE PHOSPHATASE ACTIVITY
    MOUSE, 1702*
  CALCITONIN
    CASE REPORT, 1282*
  EXTRASKELETAL
    RADIATION-INDUCED, CASE REPORT, 1319*

OVARIAN CARCINOMA
  HYSTERECTOMY
    UNILATERAL OVARECTOMY, CLINICAL STUDY, 1800*
  MORTALITY
    U.S.A., 1601*

OVARIAN TERATOCARCINOMA
  SERUM ALPHA-FETOPROTEIN CONCENTRATION
    CASE REPORT, 1519*

OVARY
  SCLEROSING STROMAL TUMORS
    CLINICAL STUDY, 1710*

ALPHA-OXOGLUTARATE
  TRANSPORT IN MITOCHONDRIA
    ASCITES TUMOR CELLS, 1771*

PANCREAS
  ADENOCARCINOMA
    EPIDEMIOLOGY, NEW YORK CITY, 1596*
    INCIDENCE, U.S.A., 1589
    MICROSPORIDIOSIS, CASE REPORT, 1656*

PANCREAS CARCINOMA
  EPIDEMIOLOGY
    U.S.A., 1589
  MORTALITY
    U.S.A., 1601*

PAPAIN
  CELL PROLIFERATION
    LIVER, RAT, MOUSE, 1648
  CELL SURFACE ARCHITECTURE
    LIVER, RAT, MOUSE, 1648
  DNA SYNTHESIS
    LIVER, RAT, MOUSE, 1648

PAPILLOMA
  LARYNGEAL
    VIRUS ISOLATION, HUMAN, 1430*

PAPILLOMATOSIS
  FLORID SUBAREOLAR DUCT
    MAMMARY CARCINOMA, CASE REPORTS, 1584

PARAFFIN OIL
  EFFECT OF INSTALLATION IN TISSUES
    HUMAN, 1286*

PARATHYROID
  MEDULLARY CARCINOMA
    ARGENTAFFIN CELLS, C-CELLS, HUMAN, 1751*

PARATHYROID CARCINOMA
  PATHOLOGY
    CLINICAL STUDY, 1707*

PERICARDIUM
  MULTIPLE TUMORS
    OYSTER, 1698*

PERMEATION
  LYMPHATIC SYSTEM
    REVIEW, 1203

PHAEOCHROMOCYTOMA
  DOPAMINE BETA-HYDROXYLASE
    RADIOIMMUNOASSAY, 1704*
  PLASMA CATECHOLAMINE
    RADIOIMMUNOASSAY, 1704*

PHENOBARBITAL
  PHOSPHOLIPID COMPOSITION CHANGES
    ENDOPLASMIC RETICULUM, LIVER, RAT, 1273

PHORBOL ESTER
  FATTY ACID
    TUMOR PROMOTION, REVIEW, 1214

PHOSPHODIESTERASE
  CYCLIC AMP
    CLONED ASTROCYTOMA CELLS, RAT,

1620

PHOSPHOLIPID
- COMPOSITION
  - MICROSOMES, MORRIS HEPATOMA, 1789*
- SYNTHESIS
  - 20-METHYLCHOLANTHRENE, LIVER, RAT, 1273
  - PHENOBARBITAL, LIVER, RAT, 1273

PHYTOHEMAGGLUTININ
- IMMUNE RESPONSE
  - CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 1535*
- LYMPHOCYTE BLASTOGENIC RESPONSE
  - AUTOIMMUNIZATION, ACUTE LEUKEMIA PATIENTS, 1456
- LYMPHOCYTE SENSITIVITY
  - CELLULAR IMMUNITY, CLINICAL STUDY, 1560*
- LYMPHOCYTE TRANSFORMATION
  - AUTOLOGOUS HUMAN LYMPHOBLASTS, 1500
  - KAPOSI'S SARCOMA, 1514*
  - MALIGNANT DISEASE PATIENTS, 1518*
  - PLASMA-FREE MEDIUM, HUMAN, 1520*
  - SERUM INHIBITORY FACTORS, LEUKEMIA PATIENTS, 1461

PINDBORG TUMOR
- AMYLOID CONTENT
  - ULTRASTRUCTURE, HUMAN, 1792*

PINOCYTOSIS
- CELL SURFACE MOTILITY
  - CONTACT INHIBITION OF MITOSIS, ROUS SARCOMA CELL LINE, RAT, 1434*

PITUITARY
- THYROTROPIC TUMORS
  - GONADOTROPIC ACTIVITIES, MOUSE, 1511*

PITUITARY GLAND
- SQUAMOUS CELL CARCINOMA
  - CASE REPORT, 1714*

PITUITARY TUMOR
- HORMONAL CHARACTERIZATION
  - HISTOLOGY, ULTRASTRUCTURE, CASE REPORT, 1768*
- HORMONE, GROWTH
  - SUPPRESSION OF ANTERIOR PITUITARY, RAT, 1731*
- PROLACTIN
  - SUPPRESSION OF ANTERIOR PITUITARY, RAT, 1731*

PLANT TUMORS
- DEVELOPMENT
  - ULTRASTRUCTURE, PEA POD, 1585*

PLASMA
- COLORIMETRIC RESPONSE TO EHRLICH'S REAGENT
  - NORMAL AND CANCER PATIENTS, 1706*
- OPSONIC ACTIVITY
  - CARCINOMA PATIENTS, 1510*

PLASMA CELL TUMOR
- TUMOR CELL INVASION OF HEPATOCYTES
  - MOUSE, 1756*

PLASMA MEMBRANE
- ISOLATION
  - EHRLICH ASCITES TUMOR CELLS, 1664*
  - VIRUS-TRANSFORMED CELLS, CHICKEN, 1418*

PODOPHYLLOTOXIN
- STEROID SECRETION STIMULATION
  - ADRENAL TUMOR CELLS, MOUSE, 1666*

POLYCHLORINATED BIPHENYLS
- CYTOCHROME P-448 INDUCTION
  - LIVER, RAT, 1298*
- DIET
  - GASTRIC LESIONS, MONKEY, 1240

POLYP, ADENOMATOUS
- SUBMUCOSAL CYSTS
  - CASE REPORT, 1740*

POLYPS, COLONIC
- GARDNER'S SYNDROME
  - CHROMOSOME ANALYSIS, HUMAN, 1609

POLYSACCHARIDE
- CARCINOGENESIS
  - REVIEW, 1207
- CONTENT
  - SV40-TRANSFORMED KIDNEY CELLS, MONKEY, 1382

POTASSIUM CYANIDE
- NUCLEOTIDE DEPLETION
  - NUCLEOSIDE TRANSPORT, RAT HEPATOMA CELLS, 1767*

PROGENITOR CRYPTOCIDES
- DEMONSTRATION IN BLOOD
  - NEOPLASTIC DISEASE PATIENTS, 1658*

PROGESTERONE
- DNA SYNTHESIS
  - CULTURED MOUSE CELLS, 1701*
- MAMMARY TUMOR INHIBITION
  - RAT, 1302

PROLACTIN
- PITUITARY TUMOR
  - SUPPRESSION OF ANTERIOR PITUITARY, RAT, 1731*

PRONASE E
- CELL PROLIFERATION
  - LIVER, RAT, MOUSE, 1648
- CELL SURFACE ARCHITECTURE
  - LIVER, RAT, MOUSE, 1648
- DNA SYNTHESIS
  - LIVER, RAT, MOUSE, 1648

GAMMA-PROPANE SULTONE
- DNA SYNTHESIS
  - SKIN, MOUSE, 1275
- RNA SYNTHESIS
  - SKIN, MOUSE, 1275

- BETA-PROPIOLACTONE
  - DNA SYNTHESIS
    - SKIN, MOUSE, 1275
  - RNA SYNTHESIS
    - SKIN, MOUSE, 1275

- PROSTAGLANDIN
  - SYNTHESIS, ABSENCE OF
    - MALIGNANT HUMAN TROPHOBLASTS, 1752*

- PROSTATE
  - CARCINOMA
    - ACID PHOSPHATASE ISOZYMES, 1713*
    - H3-ESTRADIOL LOCALIZATION, HUMAN, 1712*
    - INCIDENCE, BIOPSY STUDY, 1606*
    - MORTALITY, U.S.A., 1601*

- PROTEASES
  - CELL PROLIFERATION
    - LIVER, RAT, MOUSE, 1648
  - CELL SURFACE ARCHITECTURE
    - LIVER, RAT, MOUSE, 1648
  - DNA SYNTHESIS
    - LIVER, RAT, MOUSE, 1648

- PROTEIN
  - CALORIE DEFICIENCY
    - IMMUNE RESPONSE, MOUSE, 1481
  - DNA
    - RADIATION-INDUCED CROSS-LINKING, MODEL, 1315*
  - METABOLISM
    - DIMETHYLNITROSAMINE, KIDNEY, RAT, 1261
  - MYELOMA
    - LIGAND BINDING SITES, MOUSE, 1447
  - NERVOUS SYSTEM-SPECIFIC
    - HUMAN NEUROBLASTOMA CELL CULTURE, 1554*

- PROTEIN KINASE
  - LEVELS
    - FELINE SARCOMA VIRUS-TRANSFORMED CELLS, 1420*

- PROTEIN, NUCLEAR
  - DNA SYNTHESIS
    - ADENOSINE DIPHOSPHORIBOSYLATION, HELA CELLS, 1654*
  - HEPATOMA, NOVIKOFF
    - LIVER, POLYACRYLAMIDE GEL ELECTROPHORESIS, RAT, 1678*

- PROTEIN, NUCLEOLAR
  - HEPATOMA, NOVIKOFF
    - COMPARISON WITH NORMAL LIVER, POLYACRYLAMIDE GEL ELECTROPHORESI, 1615

- PSORALEN
  - DNA PHOTODAMAGE
    - POST-IRRADIATION DARK RECOVERY, 1316*

- PURINE
  - METABOLISM
    - HUMAN LYMPHOCYTES AND LEUKEMIC CELLS, 1797*

- PUROMYCIN
  - ALKALINE PHOSPHATASE ACTIVITY
    - TRANSPLANTED TUMOR CELLS, MOUSE, 1772*
  - SUCCINATE DEHYDROGENASE ACTIVITY
    - TRANSPLANTED TUMOR CELLS, MOUSE, 1772*

- PYRENE
  - HISTONE BINDING, 1276

- PYRIMIDINE PHOSPHORIBOSYLTRANSFERASE
  - PROPERTIES
    - MURINE LEUKEMIA CELLS, 1750*

- PYRUVATE KINASE
  - INDUCTION
    - HORMONALLY TREATED RATS, 1637

- RADIATION
  - ADENOCARCINOMA OF THE TRANSVERSE COLON
    - CASE REPORT, 1309*
  - CARCINOGENESIS
    - HUMAN, REVIEW, 1209
  - HEPATOCARCINOGENESIS
    - CELL CYCLE, MOUSE, 1303
    - LINEAR ENERGY TRANSFER, MOUSE, 1303
  - RHABDOMYOSARCOMA
    - CELL VOLUME DISTRIBUTIONS, RAT, 1696*
    - DNA CONTENT, RAT CELLS, 1696*

- RADIATION, GAMMA
  - DNA
    - ENDONUCLEASE ACTIVITY, HUMAN CELLS, 1313*
  - MAMMARY TUMOR
    - INHIBITION BY PROGESTERONE, RAT, 1302

- RADIATION, IONIZING
  - DEOXYRIBONUCLEOPROTEIN
    - IR SPECTRA ALTERATIONS, CALF THYMUS, 1317*
  - DNA
    - IR SPECTRA ALTERATIONS, CALF THYMUS, 1317*

- RADIATION, UV
  - DEOXYRIBONUCLEOPROTEIN
    - IR SPECTRA ALTERATIONS, CALF THYMUS, 1317*
  - DNA
    - IR SPECTRA ALTERATIONS, CALF THYMUS, 1317*
  - DNA AND PROTEIN CROSS-LINKING
    - MODEL, 1315*
  - DNA REPAIR SYNTHESIS
    - XERODERMA PIGMENTOSUM PATIENTS, 1301
  - GIANT CELL FORMATION
    - HYPOXIA, CYCLOHEXIMIDE, OEDOGONIUM CARDIACUM, 1305
  - MITOCHONDRIA
    - ALTERATION OF OXIDATIVE PHOSPORYLATION, RAT LIVER, 1310*

    - PROTEIN STATE CHANGES, RAT LIVER, 1310*
  - VIRUS, MURINE LEUKEMIA
    - INACTIVATION, PERMISSIVE AND NON-PERMISSIVE CELLS, 1350
    - TITRATION PATTERNS, PERMISSIVE AND NON-PERMISSIVE CELLS, 1350

- RADIATION, X-RAY
  - BRAIN
    - GOMORI POSITIVE GLIAL CELLS, MOUSE, 1318*
  - GIANT CELL FORMATION
    - HYPOXIA, CYCLOHEXIMIDE, OEDOGONIUM CARDIACUM, 1305
  - IMMUNE RESPONSE
    - LEUKEMIA CELLS, MOUSE, 1531*
  - LEUKEMIA, MYELOID
    - INCIDENCE, BLOOD LOSS, RATS, 1312*
  - MELANOMA
    - MELANOSOMAL TYROSINASE ACTIVITY, MOUSE, 1311*
  - RESISTANCE
    - RAUSCHER LEUKEMIA VIRUS-INFECTED SPLEEN CELLS, MOUSE, 1427*
  - SKIN TUMOR
    - RAT, 1307
  - TRANSFORMATION
    - EMBRYO CELLS, HAMSTER, 1300

- RADIOAUTOGRAPHY
  - RAPID GENERATION OF WHOLE-BODY RADIOAUTOGRAMS
    - MICROCHANNEL PLATE ELECTRON IMAGE AMPLIFIERS, 1689*

- RADIORESISTANCE
  - RAUSCHER LEUKEMIA VIRUS-INFECTED SPLEEN CELLS
    - MOUSE, 1427*
  - SPLENOMEGALY
    - RAUSCHER LEUKEMIA VIRUS-INFECTED MOUSE, 1419*

- RECTAL CARCINOMA
  - MORTALITY
    - U.S.A., 1601*

- RECTUM
  - COLORECTAL CARCINOMA
    - CLINICAL STUDY, 1690*

- REGENERATION, LIMB
  - ACETYLCHOLINE
    - NEWT, 1687*
  - CYCLIC AMP
    - NEWT, 1687*
  - DIBUTYRYL AMP
    - NEWT, 1687*
  - IMIDAZOLE
    - NEWT, 1687*
  - THEOPHYLLINE
    - NEWT, 1687*

- REGRESSION, TUMOR
  - HISTIOCYTE-TUMOR CELL INTERACTIONS
    - BCG, ULTRASTRUCTURE, GUINEA PIG, 1482

- REIDER CELL
  - MICROTUBULES
    - ULTRASTRUCTURE, HUMAN LEUKEMIA, 1794*

- RENAL CARCINOMA
  - LIVING KIDNEY GRAFT DONOR
    - CASE REPORT, 1652

- RENAL CELL CARCINOMA
  - GROWTH, TUMOR
    - MOUSE, 1692*

- RESISTANCE
  - METHYLENE DIMETHANESULPHONATE
    - TRANSFERABLE RESISTANCE FACTOR, YOSHIDA SARCOMA CELLS, 1617
  - NITROGEN MUSTARD
    - IMMUNOSENSITIVITY, YOSHIDA SARCOMA CELLS, RAT, 1483
  - RADIATION, X-RAY
    - RAUSCHER LEUKEMIA VIRUS-INFECTED SPLEEN CELLS, MOUSE, 1427*
  - STOMACH CANCER
    - N-METHYL-N'-NITRO-N-NITROSOGUANIDINE, RAT, 1257
  - TRANSPLANTATION
    - FETAL ANTIGENS, HUMAN ADENOVIRUS-INDUCED TUMORS, HAMSTER, 1509*
  - VIRUS, AVIAN SARCOMA
    - GENE INTERACTION, CHICKEN, 1335
  - VIRUS, HERPES SIMPLEX
    - THERMOSENSITIVE EVENTS, ADENOVIRUS TRANSFORMED HAMSTER CELLS, 1345

- RETICULOENDOTHELIAL SYSTEM
  - IMMUNITY, CELL-MEDIATED
    - FIBROMA VIRUS-INDUCED TUMORS, RABBIT, 1486

- RETICULUM CELL CARCINOMA
  - PULMONARY METASTASIS
    - BASAL CELL CARCINOMA, CASE REPORT, 1659*

- RETICULUM CELL SARCOMA
  - BENZENE
    - MOUSE, 1241
  - DEVELOPMENT
    - COLCHICINE THERAPY FOR GOUT, CASE REPORT, 1294*
  - LEUKEMIC PHASE
    - ULTRASTRUCTURE, CLINICAL STUDY, 1719*
  - LUNG
    - RARE EARTH METAL DUST, RAT, 1304

- RETINOBLASTOMA
  - ULTRASTRUCTURE
    - CASE REPORT, 1775*

- REVERSE TRANSCRIPTASE
  - DNA SYNTHESIS
    - INCORRECT BASE PAIRING, 1614
  - VIRUS, AVIAN MYELOBLASTOSIS
    - INHIBITION, NARCISSUS TAZETTA L., 1421*

- RHABDOMYOSARCOMA

- GROWTH
  - MURINE SARCOMA VIRUS, MOUSE, 1329
- RADIATION
  - CELL VOLUME DISTRIBUTIONS, RAT, 1696*
  - DNA CONTENT, RAT CELLS, 1696*

RIBONUCLEOTIDE REDUCTASE
- ACTIVITY
  - SARCOMA CELLS, RAT, 1793*
- INHIBITION BY RNA
  - EHRLICH TUMOR CELLS, 1649

RIFAMYCIN
- INHIBITION OF DNA POLYMERASE
  - LEUKEMOGENESIS, MOUSE, 1383
- INHIBITION OF RAUSCHER VIRUS-INDUCED LEUKEMIA
  - MOUSE, 1383

RNA
- BINDING
  - N-MONOMETHYL-4-AMINOAZOBENZENE, IN VITRO REACTIONS, 1268
- DISTRIBUTION OF RIBOSOMAL CISTRONS
  - NUCLEI, EHRLICH ASCITES TUMOR, MOUSE, 1650
  - NUCLEI, HEPATIC CELLS, MOUSE, 1650
- NUCLEOTIDES
  - DISTRIBUTION OF ANTIGEN, FRAGMENTS OF IGG, 1521*
- RIBONUCLEOTIDE REDUCTASE INHIBITION
  - EHRLICH TUMOR CELLS, 1649
- 80S-ASSOCIATED
  - L-CELL VIRIONS, MOUSE, 1325
- SUBRIBOSOMAL PARTICLE FORMATION
  - AFLATOXIN B1, LIVER, RAT, 1236
- VIRUS, AVIAN MYELOBLASTOSIS
  - OLIGONUCLEOTIDES, IDENTIFICATION, 1400
- VIRUS, MURINE LEUKEMIA
  - NONPRODUCER CELL, RAT, MOUSE, 1334
- VIRUS, MURINE SARCOMA
  - NONPRODUCER CELL, RAT, MOUSE, 1334
  - OLIGONUCLEOTIDES, IDENTIFICATION, 1400
- VIRUS, MURINE SARCOMA-LEUKEMIA
  - SUBUNITS, 1432*
- VIRUS, ROUS SARCOMA
  - OLIGONUCLEOTIDES, IDENTIFICATION, 1400
- VIRUS, SIMIAM SARCOMA
  - BIOCHEMICAL AND MORPHOLOGICAL STUDIES, 1374

RNA POLYMERASE
- DNA DEPENDENT
  - STIMULATION BY PROTEIN FACTORS, NOVIKOFF ASCITES CELL, 1628

RNA, RIBOSOMAL
- HEPATOMA
  - WARFARIN THERAPY, MOUSE, 1622

RNA SYNTHESIS
- ACTINOMYCIN D
  - FOWL PLAQUE VIRUS REPLICATION, HAMSTER, 1368
  - POLYOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 1368
  - ROUS SARCOMA VIRUS-TRANSFORMED CELLS, HAMSTER, 1368
- ALKYLATING AGENT
  - SKIN, MOUSE, 1275
- BIS(CHLOROMETHYL)ETHER
  - SKIN, MOUSE, 1275
- CHLOROMETHYL METHYL ETHER
  - SKIN, MOUSE, 1275
- DIMETHYLNITROSAMINE
  - KIDNEY, RAT, 1261
- INHIBITION
  - METHYLAZOXYMETHANOL ACETATE-TREATED RATS, 1249
- GAMMA-PROPANE SULTONE
  - SKIN, MOUSE, 1275
- BETA-PROPIOLACTONE
  - SKIN, MOUSE, 1275
- URETHANE
  - SKIN, MOUSE, 1275
- VIRUS, MURINE SARCOMA
  - TRANSFORMED CELL LINE, MOUSE, 1404
- VIRUS, ROUS SARCOMA
  - NUCLEOSIDE ANALOGUES, 1413*

RNA TRANSCRIPTION
- ADENOVIRUS 2-SV40 HYBRID
  - PREFERRED INITIATION SITE, 1352
- VIRUS, HERPES SIMPLEX
  - CERVICAL CANCER TISSUE, HUMAN, 1397
- VIRUS, MURINE SARCOMA
  - TRANSFORMED CELL LINE, MOUSE, 1404
- VIRUS, ROUS SARCOMA
  - RNA TEMPLATE CHARACTERIZATION, 1411

RNA, TRANSFER
- EUKARYOTIC INITIATOR
  - NUCLEOTIDE SEQUENCE, MOUSE MYELOMA CELLS, 1700*
- HEPATOMA
  - CHROMATOGRAPHIC STUDIES, 1639
- METHYLTRANSFERASE
  - NORMAL LIVER AND NOVIKOFF HEPATOMA, RAT, 1669*
- SYNTHETASE ACTIVITY
  - VIRUS, AVIAN MYELOBLASTOSIS, 1388

RNASE
- ACTIVITY
  - TRANSPLANTABLE TUMORS, HISTOCHEMISTRY, RAT, 1631
- CHARGE TRANSFER
  - BIO-MOLECULAR DAMAGE BY LOW ENERGY PROTONS, 1308

SACCHARIN
- N-METHYL-N-NITROSOUREA
  - BLADDER CARCINOMA, RAT, 1242

SAFROLE
- HEPATOCELLULAR CARCINOMA
  - RAT, 1262
- METABOLISM
  - HEPATOCARCINOGENESIS, 1274

SALIVARY GLAND
- ADENOCYSTIC CARCINOMA

    NUCLEAR DNA CONTENT, HUMAN, 1683*

SARCOMA
  7,12-DIMETHYLBENZ(A)ANTHRACENE
    CHROMOSOMES, HAMSTER, 1243
  GROWTH, TUMOR
    GRAFT-VS-HOST REACTION, MOUSE, 1555*
  3H-THYMIDINE LABELING
    AUTORADIOGRAPHY, 1580
  IRON DEXTRAN
    CASE REPORTS, 1287*
  LUNG METASTASIS PREVENTION
    PLASMA FACTOR, RAT, 1528*
  3-METHYL-CHOLANTHRENE
    IMMUNITY, CYCLOPHOSPHAMIDE THERAPY, MOUSE, 1281*
  METHYLCHOLANTHRENE
    CROSS-REACTING EMBRYONIC ANTIGEN, RAT, 1497
    IMMUNE RESPONSE, MOUSE, 1454
  RIBONUCLEOTIDE REDUCTASE ACTIVITY
    RAT CELLS, 1793*
  VIRUS, ECTROMELIA
    MOUSE, 1415*
  YOSHIDA CELLS
    METHYLENE DIMETHANESULPHONATE, TRANSFERABLE RESISTANCE FACTOR, 1617

SARCOMA, SOFT TISSUE
  MAMMARY CARCINOMA
    FAMILIAL OCCURRENCE, 1691*

SELENIUM, BLOOD
  NORMAL AND CANCER PATIENTS
    DETECTION OF GASTROINTESTINAL CANCER AND HODGKIN'S DISEASE, 1685*

SEMINOMA
  PRIMARY THYMIC
    ULTRASTRUCTURE, CASE REPORT, 1715*

SERUM
  ANTIGEN, GROUP-SPECIFIC
    DETECTION, HUMAN, 1502
  ANTIGEN, TUMOR-ASSOCIATED
    DETECTION, HUMAN, 1502
  CELL PROLIFERATION FACTORS
    LEUKEMIC CELLS, HUMAN, 1640
  CYTOTOXIN LEVELS
    ASCITES TUMOR DEVELOPMENT, RAT, 1507*
  DNA SYNTHESIS STIMULATING FACTOR
    CELL GROWTH, PARTIALLY HEPATECTOMIZED RATS, 1616
  MURAMIDASE
    LEUKEMIA, CLINICAL STUDY, 1778*

SIALIC ACID
  ENZYME DIGESTION
    TUMOR CELLS, MOUSE, 1644

SIALYLTRANSFERASE
  THYROGLOBULIN SYNTHESIS
    THYROID CARCINOMA, RAT, 1695*

SKIN
  BASAL CELL CARCINOMA
    CLINICAL STUDY, 1784*
  BASOSQUAMOUS CARCINOMA
    CLINICAL STUDY, REVIEW, 1223*
  FIBROSARCOMA
    CASE REPORT, 1745*
  MELANOMA, MALIGNANT
    CLINICAL STUDY, 1784*
  NEOPLASMS, MALIGNANT
    CLINICAL STUDY, 1784*
  SQUAMOUS CELL CARCINOMA
    CLINICAL STUDY, 1784*

SKIN TUMOR
  7,12-DIMETHYLBENZ(A)ANTHRACENE
    BCG VACCINATION, MOUSE, 1475
    CROTON OIL, MOUSE, 1475
  RADIATION, X-RAY
    RAT, 1307

SKULL
  MENINGEOMA
    GROWTH, CASE REPORTS, 1604*

SODIUM CYCLAMATE
  CHROMOSOME ABERRATIONS
    CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*
  DNA SYNTHESIS
    CYTOKINETIC STUDY, CULTURED HUMAN CELLS, 1291*

SPINAL CORD
  CYSTIC TUMOR
    CYST PUNCTURE, ENDOMYELOGRAPHY, CASE REPORTS, 1728*

SPLEEN
  ANTIGEN-INDUCED DEPRESSION OF DNA SYNTHESIS
    MOUSE, 1452

SPLENOMEGALY
  RADIORESISTANCE
    RAUSCHER LEUKEMIA VIRUS-INFECTED MOUSE, 1419*

SQUAMOUS CELL CARCINOMA
  LICHEN PLANUS
    TONGUE, CASE REPORT, 1587*
  PITUITARY GLAND
    CASE REPORT, 1714*
  SKIN
    CLINICAL STUDY, 1784*

STEATORRHOEA
  DEVELOPMENT
    COLCHICINE THERAPY FOR GOUT, CASE REPORT, 1294*

STOMACH
  GASTRIC LESIONS
    POLYCHLORINATED BIPHENYLS, MONKEY, 1240

STOMACH CANCER
  ENVIRONMENTAL FACTORS
    HUMAN, 1594
  N-METHYL-N'-NITRO-N-NITROSOGUANIDINE

RESISTANCE, RAT, 1257
MORTALITY
U.S.A., 1601*

SUCCINATE DEHYDROGENASE
ACTIVITY
ACTINOMYCIN D, TRANSPLANTED TUMOR CELLS, MOUSE, 1772*
PUROMYCIN, TRANSPLANTED TUMOR CELLS, MOUSE, 1772*

SULPHADIAZINE
CONCENTRATION
MALIGNANT TISSUE, MOUSE, RAT, 1618

SYNCYTIA
FORMATION
C-TYPE VIRUS, XC-PLAGUE ASSAY, 1370

TERATOCARCINOGENESIS
GENETICS
REVIEW, 1222*

TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE
ACTIVITY
CELL LINE, ACUTE LYMPHOBLASTIC LEUKEMIA PATI, 1694*

TESTIS
CANCER INCIDENCE
CALIFORNIA, 1591

12-O-TETRADECANOYLPHORBOL-13-ACETATE
TUMOR PROMOTION
FATTY ACIDS, REVIEW, 1214

THEOPHYLLINE
REGENERATION, LIMB
NEWT, 1687*

THIOSEMICARBAZONE
VIRUS, MAEDI
INHIBITION, 1424*
VIRUS, PROGRESSIVE PNEUMONIA
INHIBITION, 1424*
VIRUS, VISNA
INHIBITION, 1424*

THROMBOCYTOPENIA
METASTASIS
MALIGNANT TUMORS, MOUSE, 1630

THYMOMA
HODGKIN'S DISEASE
THYMUS, CLINICOPATHOLOGICAL STUDY, 1621

THYMUS
ANTI-THYMOCYTE SERUM
PARADOXICAL EFFECT, MOUSE, 1515*
ANTIGEN, SPLENO-THYMIC
LEUKEMIA, HUMAN, 1505*
C-TYPE VIRUS ACTIVATION
BONE MARROW, MOUSE, 1280
HODGKIN'S DISEASE
CLINICOPATHOLOGICAL STUDY, 1621
SEMINOMA
ULTRASTRUCTURE, CASE REPORT, 1715*
THYMOMA
CLINICOPATHOLOGICAL STUDY, 1621

THYROGLOBULIN SYNTHESIS
THYROID CARCINOMA
SIALYLTRANSFERASE ACTIVITY, RAT, 1695*

THYROID
MEDULLARY CARCINOMA
ACTH AND CALCITONIN SECRETION, CASE REPORT, 1749*
ARGENTAFFIN CELLS, C-CELLS, HUMAN, 1751*
CLINICOPATHOLOGIC STUDY, 1674*
HUMAN, REVIEW, 1231*
OSTEOCLASTOMA-LIKE GIANT CELL TUMOR
RADIOTHERAPY, CASE REPORT, 1709*

THYROID CARCINOMA
PULMONARY METASTASIS
BASAL CELL CARCINOMA, CASE REPORT, 1659*
RADIOACTIVE IODINE UPTAKE STUDY
CASE REPORT, 1306
THYROGLOBULIN SYNTHESIS
SIALYLTRANSFERASE ACTIVITY, RAT, 1695*

TOBACCO SMOKING
IMMUNE RESPONSE
MOUSE, 1295*
LUNG CANCER
HISTOPATHOLOGY, HUMAN, 1582
HUMAN, 1598*

TONGUE
LICHEN PLANUS
SQUAMOUS CELL CARCINOMA, CASE REPORT, 1587*

TPPASE
ACTIVITY
CEREBRAL TUMORS, HUMAN, 1748*

TRANSACETYLASE
N-HYDROXY-N-ACETYLAMINOFLUORENE
RAT, 1235

TRANSFORMATION
BENZO(A)PYRENE
EMBRYO CELLS, RAT, 1263
7,12-DIMETHYLBENZ(A)ANTHRACENE
EMBRYO CELLS, RAT, 1263
LYMPHOCYTE
AUTOLOGOUS HUMAN LYMPHOBLASTS, 1500
CONCANAVALIN A BINDING, HUMAN, 1668*
PHYTOHEMAGGLUTININ, KAPOSI'S SARCOMA, 1514*
PHYTOHEMAGGLUTININ, MALIGNANT DISEASE PATIENTS, 1518*
PHYTOHEMAGGLUTININ, PLASMA-FREE MEDIUM, HUMAN, 1520*
PHYTOHEMAGGLUTININ, SERUM INHIBITORY FACTORS, LEUKEMIA PATIENTS, 1461
WHEAT GERM AGGLUTININ BINDING,

HUMAN, 1668*
- 3-METHYLCHOLANTHRENE
  - EMBRYO CELLS, RAT, 1263
- 4-NITROQUINOLINE-1-OXIDE
  - ADENOVIRUS INFECTION, HAMSTER CELLS, 1363
- RADIATION, X-RAY
  - EMBRYO CELLS, HAMSTER, 1300
- VIRUS, ADENO
  - POLYCYCLIC HYDROCARBONS, HAMSTER CELLS, 1331
- VIRUS, ADENO 5
  - EMBRYO CELLS, HAMSTER, 1385
- VIRUS, ADENO 12
  - HERPES SIMPLEX RESISTANCE, THERMOSENSITIVITY, HAMSTER CELLS, 1345
- VIRUS, ADENO SA7
  - CHROMOSOMES, HAMSTER CELLS, 1364, 1371
- VIRUS, AVIAN SARCOMA
  - FIBRINOLYSIN, RAT EMBRYO CELLS, 1342
- VIRUS, HERPES SIMPLEX
  - EMBRYO FIBROBLASTS, HAMSTER, 1403
  - EMBRYONIC LUNG CELLS, HUMAN, 1398
- VIRUS, MURINE SARCOMA
  - CANINE EMBRYO CELLS, 1369
  - SUGAR TRANSPORT, MOUSE EMBRYO FIBROBLASTS, 1384
- VIRUS, POLYOMA
  - CYTOPLASMIC DNA, HAMSTER CELLS, 1324
  - DIBUTYRL CYCLIC AMP, 1328
  - GENETIC CONTROL, HAMSTER CELLS, 1332
  - PROTEIN CHARACTERIZATION, REVIEW, 1208
- VIRUS, ROUS SARCOMA
  - FIBRINOLYSIN, CHICK EMBRYO FIBROBLASTS, 1333
  - NONDIVIDING CHICK EMBRYO CELLS, 1389
- VIRUS, SV40
  - DIBUTYRL CYCLIC AMP, 1328
  - DNA INTEGRATION SITE, MAMMALIAN CELLS, 1396
  - FIBRINOLYSIN, HAMSTER EMBRYO CELLS, 1342
  - PROTEIN CHARACTERIZATION, REVIEW, 1208
  - TEMPERATURE-SENSITIVE MUTANT, RAT CELL, 1390

TRANSPLANTATION
- ANTIGEN
  - BO-IV CARCINOMA, SYNGENEIC MICE, 1541*
  - LYMPHOID CELLS, CHIMERIC MICE, 1561*
  - MAMMARY CARCINOMA, SYNGENEIC MICE, 1541*
- COLONY FORMING UNITS
  - GROWTH, ERYTHROPOIESIS, LEUKEMIA-INFECTED CELLS, MOUSE, 1412*
- IMMUNITY
  - HERPES-SIMPLEX VIRUS-TRANSFORMED CELLS, HAMSTER, 1403
- KIDNEY
  - PAPOVA VIRUS, IMMUNOSUPPRESSION, HUMAN, 1496
- KINETICS
  - TUMOR CELLS, MOUSE, 1448
- MAMMARY TUMOR
  - HORMONE DEPENDENCE, RAT, 1259
- MELANOMA B16 CELLS
  - CULTIVATION, TRYPSINIZATION AND AGGREGATION, 1462
- MELANOMA, HUMAN
  - METASTASIS, THYMUS-DEFICIENT MOUSE, 1688*
- RESISTANCE
  - FETAL ANTIGENS, HUMAN ADENOVIRUS-INDUCED TUMORS, HAMSTER, 1509*
- TUMOR
  - COMPLEMENT ACTIVITY, MOUSE, 1457
  - IMMUNE RESPONSE, MOUSE, 1457

TRYPSIN
- CELL PROLIFERATION
  - LIVER, RAT, MOUSE, 1648
- CELL SURFACE ARCHITECTURE
  - LIVER, RAT, MOUSE, 1648
- DNA SYNTHESIS
  - LIVER, RAT, MOUSE, 1648

TRYPTOPHAN
- METABOLISM
  - BLADDER CANCER PATIENTS, CLINICAL STUDY, 1647

TUMOR
- 3H-THYMIDINE LABELING
  - AUTORADIOGRAPHY, 1580
- LONG-TERM SPONTANEOUS INCIDENCE
  - NEONATALLY THYMECTOMIZED MICE, 1439
- SPONTANEOUS DEVELOPMENT
  - PATHOLOGICAL LESIONS, MOUSE, 1726*
- VASCULARIZATION
  - MORPHOLOGY, RAT, 1783*

TUMOR CELL
- SEPARATION OF VIABLE FROM NONVIABLE
  - FLOTATION ON A FICOLL-TRIOSIL MIXTURE, 1776*

TUMOR VIRUS
- SEE VIRUS

TUMORIGENESIS
- IMMUNIZATION WITH NORMAL SYNGENEIC TISSUES
  - MOUSE, 1548*
- PSYCHOBIOLOGICAL FACTORS
  - YOUNG ADULTS, 1764*

TYROSINASE
- ACTIVITY
  - CULTURED MELANOMA CELLS, MOUSE, 1763*

URANIUM
- OCCUPATIONAL HAZARD
  - CANCER MORTALITY, URANIUM MILL WORKERS, U.S.A., 1588

URETHANE

- DNA SYNTHESIS
    - SKIN, MOUSE, 1275
- RNA SYNTHESIS
    - SKIN, MOUSE, 1275

URIDINE KINASE
- PARTIAL PURIFICATION
    - EHRLICH ASCITES TUMOR CELLS, 1680*

URINARY BLADDER CARCINOMA
- ANTIGEN, TUMOR-SPECIFIC
    - HUMAN CELL LINE, 1458
- N-METHYL-N-NITROSOUREA
    - RAT, 1242
- SACCHARIN
    - RAT, 1242
- TRYPTOPHAN METABOLISM
    - CLINICAL STUDY, 1647
- VARIABLE SEX CHROMATIN PATTERN
    - CASE REPORT, 1737*

URINARY BLADDER TUMOR
- 2-ACETYLAMINOFLUORENE
    - BETA-GLUCURONIDASE INHIBITOR, RAT, 1247
- BENZIDINE
    - OCCUPATIONAL HAZARD, 1277

UROTHELIAL CARCINOMA
- ANTIGEN, CARCINOEMBRYONIC
    - CLINICAL STUDY, 1552*

UTERINE CERVIX
- ADENOCARCINOMA
    - CYTOLOGIC, HISTOLOGIC, AND CYTOPHOTOMETRIC STUDIES, 1799*
- VIRUS, HERPES 2
    - HORMONAL IMBALANCE, MOUSE, 1409

UTERUS
- CANCER
    - MORTALITY, U.S.A., 1601*
- MYOMA
    - GLYCOGEN PHOSPHORYLASE B ACTIVITY, HUMAN, 1777*
- TUMOR INDUCTION
    - MURINE SARCOMA VIRUS, PREGNANT RAT, 1347
    - MURINE SARCOMA VIRUS, ULTRASTRUCTURE, PREGNANT RAT, 1348

UTPASE
- ACTIVITY
    - MAMMARY TUMOR, RAT, 1254

VACCINATION
- ANTITUMOR
    - YEAST, MOUSE, 1506*

VINBLASTINE
- STEROID SECRETION STIMULATION
    - ADRENAL TUMOR CELLS, MOUSE, 1666*

VIROLOGY
- CANCER HYPOTHESIS
    - VIROLOGICAL AND IMMUNOLOGICAL RELATIONSHIPS, 1346
- IMMUNOLOGY
    - CHEMOTHERAPY, RADIOTHERAPY, REVIEW, 1220*

VIROSOMES
- ISOLATION
    - ULTRASTRUCTURE, MITOCHONDRIA, ROUS SARCOMA CELLS, CHICKEN, 1431*

VIRUS
- ADENOVIRUS 2-SV40 HYBRID
    - RNA TRANSCRIPTION INITIATION SITE, 1352
- HUMAN CANCER
    - REVIEW, 1227*
- ISOLATION
    - HUMAN LARYNGEAL PAPILLOMA, 1430*
- L-CELL VIRION
    - 80S-ASSOCIATED RNA, MOUSE, 1325
- SUBVIRAL ONCOGENIC PARTICLES
    - ULTRASTRUCTURE, MITOCHONDRIA, ROUS SARCOMA CELLS, CHICKEN, 1431*

VIRUS, ADENO
- TRANSFORMATION
    - POLYCYCLIC HYDROCARBONS, HAMSTER CELLS, 1331

VIRUS, ADENO 5
- TRANSFORMATION
    - EMBRYO CELLS, HAMSTER, 1385

VIRUS, ADENO 7
- ENHANCEMENT OF REPLICATION OF HUMAN ADENOVIRUSES BY SV40
    - DNA SYNTHESIS, SIMIAN CELLS, 1408

VIRUS, ADENO 12
- ANTIGEN, SURFACE
    - IMMUNOFLUORESCENCE, INFECTED KB CELLS, 1504
- INDUCED TUMORS
    - FETAL ANTIGEN-INDUCED TRANSPLANTATION RESISTANCE, HAMSTER, 1509*
- 4-NITROQUINOLINE-1-OXIDE
    - CHROMOSOME ABERRATIONS, HAMSTER CELLS, 1363
    - DNA REPAIR SYNTHESIS, HAMSTER CELLS, 1363
    - TRANSFORMATION, HAMSTER CELLS, 1363
- TRANSFORMATION
    - HERPES SIMPLEX RESISTANCE, THERMOSENSITIVITY, HAMSTER CELLS, 1345

VIRUS, ADENO SA7
- 4-NITROQUINOLINE-1-OXIDE
    - CHROMOSOME ABERRATIONS, HAMSTER CELLS, 1363
    - DNA REPAIR SYNTHESIS, HAMSTER CELLS, 1363
    - TRANSFORMATION, HAMSTER CELLS, 1363
- TRANSFORMATION
    - CHROMOSOMES, HAMSTER CELLS, 1364, 1371
- TUMOR INDUCTION
    - HAMSTER, 1433*

- VIRUS, AVIAN LEUKOSIS
  - ANTIGEN
    - IMMUNOHISTOCHEMISTRY, 1488
  - DNA
    - RNA-DNA HYBRIDIZATION, CHICK EMBRYO, 1391

- VIRUS, AVIAN MYELOBLASTOSIS
  - DNA POLYMERASE
    - DNA SYNTHESIS, INCORRECT BASE PAIRING, 1614
    - INHIBITION, NARCISSUS TAZETTA L., 1421*
  - REVERSE TRANSCRIPTASE
    - INHIBITION, NARCISSUS TAZETTA L., 1421*
  - RNA
    - OLIGONUCLEOTIDES, IDENTIFICATION, 1400
  - TRANSFER RNA SYNTHETASE ACTIVITY, 1388

- VIRUS, AVIAN RNA TUMOR
  - HOST RANGE RECOMBINENTS
    - ISOLATION, 1401

- VIRUS, AVIAN SARCOMA
  - RESISTANCE
    - GENE INTERACTION, CHICKEN, 1335
  - TRANSFORMATION
    - FIBRINOLYSIN, RAT EMBRYO CELLS, 1342
  - TRANSFORMED CELLS
    - PLASMA MEMBRANE ISOLATION, CHICKEN, 1418*

- VIRUS, C-TYPE PARTICLE
  - ACTIVATION
    - 5-BROMODEOXYURIDINE, EMBRYONIC RAT FIBROBLASTS, 1355
    - METHYLNITROSOUREA, BONE MARROW, MOUSE, 1280
    - METHYLNITROSOUREA, THYMUS, MOUSE, 1280
  - MAMMARY ADENOCARCINOMA
    - RAT, 1426*
  - SURFACE ANTIGEN
    - HYBRID CELLS, MOUSE, 1341
  - SYNCYTIA FORMATION
    - XC-PLAQUE ASSAY, 1370

- VIRUS, C-TYPE RNA
  - ACTIVATION
    - 5-BROMODEOXYURIDINE, TRANSFORMED MOUSE CELLS, 1322
  - EXPERSSION
    - SPONTANEOUS TUMORS, MOUSE, 1493
  - HOST-GENE CONTROL OF EXPERSSION
    - TUMORIGENESIS, MOUSE, 1375
  - INDUCTION
    - GENETIC LOCI, MOUSE, 1379
  - ISOLATION
    - KIDNEY CELL LINE, CAT, 1399
  - LEUKEMIA, MYELOMONOCYTIC
    - ULTRASTRUCTURE, MOUSE TUMOR CELLS, 1323
  - LOCALIZATION
    - GLOMERULAR BASEMENT MEMBRANE, MOUSE, 1377
  - MOLECULAR RELATEDNESS
    - DNA-RNA HYBRIDIZATION, 1378

- VIRUS, CELO
  - TUMORIGENESIS
    - MOUSE, 1376

- VIRUS, ECTROMELIA
  - SARCOMA
    - MOUSE, 1415*

- VIRUS, EPSTEIN-BARR
  - ANTIGEN
    - DNA SYNTHESIS, HUMAN LYMPHOBLASTOID CELLS, 1386
  - BURKITT'S LYMPHOMA
    - ETIOLOGY, REVIEW, 1219
  - COMPLEMENT-FIXING ANTIGEN
    - LOCALIZATION, HUMAN LYMPHOBLASTOID CELL LINES, 1381
  - HETEROPHILE RESPONSE
    - LEUKOCYTES, MONKEY, 1343
  - NASOPHARYNGEAL CARCINOMA
    - ETIOLOGY, REVIEW, 1219
  - NEOCARZINOSTATIN
    - ULTRASTRUCTURE, BURKITT'S LYMPHOMA CELLS, 1425*
  - SUPERINFECTION
    - LYMPHOBLASTOID CELL LINES, HUMAN, 1367

- VIRUS, FELINE SARCOMA
  - TRANSFORMED CELLS
    - PROTEIN KINASE LEVELS, 1420*

- VIRUS, FIBROMA
  - IMMUNITY, CELL-MEDIATED
    - RABBIT, 1486

- VIRUS, FRIEND LEUKEMIA
  - ANTIGENS, GROUP- AND TYPE-SPECIFIC
    - LEUKEMIA DEVELOPMENT, MOUSE, 1508*
  - CELL PROLIFERATION
    - KINETICS, MOUSE, 1366
  - ELIMINATION IN FECES
    - FLY, FLEA, 1356
  - ERYTHROLEUKEMIA CELLS
    - ULTRASTRUCTURE, 1337
  - INHERITED SUSCEPTIBILITY
    - GENETIC CONTROL, MOUSE, 1330
  - LEUKEMIA DEVELOPMENT
    - MOUSE, 1578
  - LEUKEMIA REGRESSION
    - MOUSE, 1578
  - PURIFICATION
    - AGGLUTINATION WITH CONCANAVALIN A, 1387

- VIRUS, GRAFFI LEUKEMIA
  - TUMOR INDUCTION ENHANCEMENT
    - SPLEEN CELL INJECTION, MOUSE, 1329

- VIRUS, GROSS
  - LYMPHOMA
    - IMMUNE CYTOTOXICITY, RAT, 1489

- VIRUS, HERPES
  - ANTIGEN, CELL-SURFACE
    - QUAIL CELLS, 1469
  - CARCINOGENESIS

    - REVIEW. 1212
  - CERVICAL ANAPLASIA
    - CYTOHISTOPATHOLOGY. HUMAN. 1393
  - VIRUS. SV40
    - CONCOMITANT INFECTION. HAMSTER. 1429*

- VIRUS. HERPES 2
  - UTERINE CERVIX
    - HORMONAL IMBALANCE. MOUSE. 1409

- VIRUS. HERPES-LIKE
  - ENVELOPMENT AND RELEASE
    - ULTRASTRUCTURE. GUINEA PIG CELLS. 1365
  - INFECTION
    - ULTRASTRUCTURE. GUINEA PIG CELLS. 1365

- VIRUS. HERPES SAIMIRI
  - BURKITT'S LYMPHOMA
    - HUMAN. MONKEY. 1349

- VIRUS. HERPES SIMPLEX
  - DNA TRANSCRIPTION
    - CERVICAL CANCER TISSUE. HUMAN. 1397
  - GENETICS
    - TEMPERATURE SENSITIVE MUTANTS. 1395
  - MULTIPLICATION
    - ETHIDIUM BROMIDE. HELA CELLS. 1423*
    - MITOCHONDRIAL GENETICAL INFORMATION. HELA CELLS. 1423*
  - PREVENTION OF CELL-TO-CELL SPREAD
    - LEUKOCYTES. RABBIT. 1340
  - RESISTANCE
    - THERMOSENSITIVE EVENTS. ADENOVIRUS TRANSFORMED HAMSTER CELLS. 1345
  - RNA TRANSCRIPTION
    - CERVICAL CANCER TISSUE. HUMAN. 1397
  - TRANSFORMATION
    - EMBRYO FIBROBLASTS. HAMSTER. 1403
    - EMBRYONIC LUNG CELLS. HUMAN. 1398

- VIRUS. LEUKEMIA
  - ACTIVATION
    - HOMOGRAFT REJECTION. MOUSE. 1422*

- VIRUS. LEUKOSIS-TYPE
  - ISOLATION
    - PHEASANT EMBRYO CELLS. 1338

- VIRUS. LYMPHATIC LEUKEMIA
  - INHERITED SUSCEPTIBILITY
    - GENETIC CONTROL. MOUSE. 1330

- VIRUS. MAEDI
  - NUCLEIC ACID SEQUENCES
    - MOLECULAR HYBRIDIZATION. 1392
  - THIOSEMICARBAZONE
    - INHIBITION. 1424*

- VIRUS. MAMMARY TUMOR
  - ANTIGEN
    - RADIOIMMUNOASSAY. MOUSE. 1442
  - DEGRADATION BY HUMAN MILK
    - MOUSE. 1361
  - HUMAN
    - REVIEW. 1215

- VIRUS. MAREK'S DISEASE
  - ANTIGEN. A
    - PURIFICATION. 1487
  - ANTIGEN. CELL-SURFACE
    - QUAIL CELLS. 1469

- VIRUS. MAZURENKO
  - ANTIGENS. GROUP- AND TYPE-SPECIFIC
    - LEUKEMIA DEVELOPMENT. MOUSE. 1508*

- VIRUS. MOLONEY LEUKEMIA
  - IMMUNE RESPONSE
    - DEPRESSION BY MALARIAL INFECTION. MOUSE. 1530*

- VIRUS. MOLONEY MURINE LEUKEMIA
  - IMMUNE RESPONSE
    - MOUSE. 1436
  - IMMUNITY. CELL-MEDIATED
    - MOUSE. 1436

- VIRUS. MOLONEY SARCOMA
  - DIMETHYL SULFOXIDE
    - ONCOGENICITY STIMULATION. MOUSE. 1360
  - LYMPHOCYTE RESPONSE
    - TUMOR. MOUSE. 1339
  - TUMORIGENESIS
    - BCG. IMMUNE RESPONSE. MOUSE. 1476

- VIRUS. MURINE LEUKEMIA
  - ANTIGEN
    - DETECTION. MOUSE. 1477
  - GENE LINKAGE
    - FV-1 LOCUS. MOUSE. 1326
  - GENETIC CONTROL
    - MOUSE. 1477
  - GRAFT-VS-HOST REACTION
    - TUMOR INDUCTION. MOUSE. 1321
  - IMMUNOSUPPRESSION
    - MOUSE. 1498
  - INDUCTION
    - 5-BROMODEOXYURIDINE. MOUSE. 1336
    - 5-IODODEOXYURIDINE. MOUSE. 1336
  - RADIATION. UV
    - INACTIVATION. PERMISSIVE AND NON-PERMISSIVE CELLS. 1350
    - TITRATION PATTERNS. PERMISSIVE AND NON-PERMISSIVE CELLS. 1350
  - RNA
    - NONPRODUCER CELL. RAT. MOUSE. 1334
  - VIRUS. MURINE SARCOMA
    - HOMOLOGOUS RNA. NONPRODUCER CELL. RAT. MOUSE. 1334

- VIRUS. MURINE SARCOMA
  - IMMUNE CYTOTOXICITY
    - EFFECTOR CELL. MOUSE. 1490
  - RNA
    - NONPRODUCER CELL. RAT. MOUSE. 1334
    - OLIGONUCLEOTIDES. IDENTIFICATION. 1400
  - RNA SYNTHESIS
    - TRANSFORMED CELL LINE. MOUSE. 1404
  - RNA TRANSCRIPTION

    - TRANSFORMED CELL LINE, MOUSE, 1404
  - TRANSFORMATION
    - CANINE EMBRYO CELLS, 1369
    - SUGAR TRANSPORT, MOUSE EMBRYO FIBROBLASTS, 1384
  - TUMOR INDUCTION
    - UTERUS, PREGNANT RAT, 1347
    - UTERUS, ULTRASTRUCTURE, PREGNANT RAT, 1348
  - TUMOR INDUCTION ENHANCEMENT
    - SPLEEN CELL INJECTION, MOUSE, 1329
  - TUMORIGENICITY
    - BONE, RAT, MOUSE, HAMSTER, 1359
  - VIRUS, MURINE LEUKEMIA
    - HOMOLOGOUS RNA, NONPRODUCER CELL, RAT, MOUSE, 1334

- VIRUS, MURINE SARCOMA-LEUKEMIA
  - RNA
    - SUBUNITS, 1432*

- VIRUS, ONCORNA
  - CARCINOGENESIS
    - REVIEW, 1211
  - PURIFICATION
    - AGGLUTINATION WITH CONCANAVALIN A, 1387

- VIRUS, PAPOVA
  - KIDNEY TRANSPLANTATION
    - URINE, HUMAN, 1496

- VIRUS, POLYOMA
  - ACTINOMYCIN D
    - FOWL PLAQUE VIRUS REPLICATION, HAMSTER, 1368
    - RNA SYNTHESIS, TRANSFORMED HAMSTER CELLS, 1368
  - DNA REPLICATION
    - PHYSICOCHEMICAL PROPERTIES, 1357
  - IMMUNOSUPPRESSION
    - ONCOGENESIS, MOUSE, 1354
  - REPLICATION INTERFERENCE
    - HOMOLOGOUS INCOMPLETE VIRUS, 1353
  - TRANSFORMATION
    - CYTOPLASMIC DNA, HAMSTER CELLS, 1324
    - DIBUTYRL CYCLIC AMP, 1328
    - GENETIC CONTROL, HAMSTER CELLS, 1332
    - PROTEIN CHARACTERIZATION, REVIEW, 1208
  - TRANSFORMED CELLS
    - INTEGRATION OF VIRAL INTO CHROMOSOMAL DNA, RAT, 1407
  - VIRUS, SENDAI
    - SYNERGISM, HUMAN CELLS, 1362

- VIRUS, PROGRESSIVE PNEUMONIA
  - THIOSEMICARBAZONE
    - INHIBITION, 1424*

- VIRUS, RACIAL-TYPE
  - MELANOMA, MALIGNANT
    - ULTRASTRUCTURE, HAMSTER CELLS, 1344

- VIRUS, RAUSCHER LEUKEMIA
  - ANTIGENS, GROUP- AND TYPE-SPECIFIC
    - LEUKEMIA DEVELOPMENT, MOUSE, 1508*
  - INFECTED SPLEEN CELLS
    - COLONY FORMING UNITS, GROWTH, ERYTHROPOIESIS, MOUSE, 1412*
  - INFECTION
    - RADIORESISTANCE, SPLEEN CELLS, MOUSE, 1427*
    - RADIORESISTANCE, SPLENOMEGALY, MOUSE, 1419*
  - LEUKEMOGENESIS
    - RIFAMYCIN, DNA POLYMERASE, MOUSE, 1383

- VIRUS, RNA TUMOR
  - BURKITT'S LYMPHOMA
    - HUMAN, 1410
  - LYMPHOMA
    - HUMAN, 1410
  - MOLECULAR HYBRIDIZATION
    - TAXONOMY, 1380
  - NUCLEIC ACID SEQUENCES
    - MOLECULAR HYBRIDIZATION, 1392

- VIRUS, ROUS
  - TUMOR INDUCTION
    - IMMUNOLOGICAL STUDY, MOUSE, 1557*

- VIRUS, ROUS SARCOMA
  - ACTINOMYCIN D
    - FOWL PLAQUE VIRUS REPLICATION, HAMSTER, 1368
    - RNA SYNTHESIS, TRANSFORMED HAMSTER CELLS, 1368
  - CYTOPATHIC EFFECT
    - TRANSFORMED CELL LINE, RAT, 1351
  - DIMETHYL SULFOXIDE
    - ONCOGENICITY STIMULATION, QUAIL, 1360
  - DNA SYNTHESIS
    - RNA TEMPLATE CHARACTERIZATION, 1411
  - IMMUNITY
    - AVIAN LEUKOSIS VIRUSES, CHICKEN, 1474
  - MUTANT
    - BIOLOGICAL AND PHYSICAL PROPERTIES, 1405
  - RNA
    - OLIGONUCLEOTIDES, IDENTIFICATION, 1400
  - RNA SYNTHESIS
    - NUCLEOSIDE ANALOGUES, 1413*
  - RNA TRANSCRIPTION
    - RNA TEMPLATE CHARACTERIZATION, 1411
  - SYNTHESIS
    - NUCLEOSIDE ANALOGUES, 1413*
  - TRANSFORMATION
    - FIBRINOLYSIN, CHICK EMBRYO FIBROBLASTS, 1333
    - NONDIVIDING CHICK EMBRYO CELLS, 1389
  - TUMORIGENESIS
    - HAMSTER, 1414*

- VIRUS, SENDAI
  - SURFACE ANTIGEN
    - HYBRID CELLS, MOUSE, 1341
  - VIRUS, POLYOMA

    SYNERGISM, HUMAN CELLS, 1362

VIRUS, SHOPE FIBROMA
  DNA SYNTHESIS INHIBITION
    MECHANISM, 1373

VIRUS, SIMIAN SARCOMA
  RNA
    BIOCHEMICAL AND MORPHOLOGICAL STUDIES, 1374

VIRUS, SPLEEN FOCUS-FORMING
  INHERITED SUSCEPTIBILITY
    GENETIC CONTROL, MOUSE, 1330

VIRUS, SV40
  DNA
    INTEGRATION SITE, TRANSFORMED MAMMALIAN CELLS, 1396
    5-IODO-2'-DEOXYURIDINE INCORPORATION, 1402
    RESTRICTION ENDONUCLEASE, E.COLI, 1417*
  DNA, CIRCULAR
    POLYACRYLAMIDE GEL ELECTROPHORESIS, 1327
  DNA METABOLISM
    SUPERINFECTION, TRANSFORMED KIDNEY CELLS, 1358
  TRANSFORMATION
    DIBUTYRL CYCLIC AMP, 1328
    FIBRINOLYSIN, HAMSTER EMBRYO CELLS, 1342
    PROTEIN CHARACTERIZATION, REVIEW, 1208
    TEMPERATURE-SENSITIVE MUTANT, RAT CELL, 1390
  TRANSFORMED KIDNEY CELLS
    POLYSACCHARIDE CONTENT, MONKEY, 1382
    TUMORIGENICITY, 5-BROMODEOXYURIDINE, HAMSTER, 1394
  TUMORIGENESIS
    HAMSTER, 1414*
  VIRUS, HERPES
    CONCOMITANT INFECTION, HAMSTER, 1429*

VIRUS, SV40-LIKE
  NUCLEIC ACID
    ISOLATION, PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY PATIENT, 1406

VIRUS, VISNA
  NUCLEIC ACID SEQUENCES
    MOLECULAR HYBRIDIZATION, 1392
  THIOSEMICARBAZONE
    INHIBITION, 1424*

VITAMIN A
  1,2-DIMETHYLHYDRAZINE-INDUCED INTESTINAL CARCINOMA
    RAT, 1267

VITAMIN C
  CELL PROLIFERATION
    REVIEW, 1213

WARFARIN
  CHEMOTHERAPY
    RIBOSOMAL RNA CONTENT, HEPATOMA, MOUSE, 1622

WHEAT GERM AGGLUTININ
  BINDING
    LYMPHOCYTE TRANSFORMATION, HUMAN, 1668*
  BINDING SITE
    ISOLATION, CELL-SURFACE GLYCOPEPTIDES, RAT HEPATOMA, 1455

WOUND HEALING
  MOLECULAR REPAIR
    CARCINOGENESIS, REVIEW, 1206

XERODERMA PIGMENTOSUM
  CHROMOSOME ABERRATIONS
    N-METHYL-N'-NITRO-NITROSOGUANIDINE, HUMAN CELLS, 1260
    4-NITROQUINOLINE-1-OXIDE, HUMAN CELLS, 1260
  DNA REPAIR SYNTHESIS
    UV RADIATION, HUMAN, 1301

YEAST
  ANTITUMOR VACCINATION
    MOUSE, 1506*

ZINC
  MALIGNANT DISEASE
    HUMAN, 1283*

☆U.S. GOVERNMENT PRINTING OFFICE:1973-544-910/3

LIBRARY U. OF I. URBANA-CHAMPAIGN

LIBRARY U. OF I. URBANA-CHAMPAIGN

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list.

DHEW Publications No. (NIH) 72-301

APRIL 1973
Abstracts & Citations Nos. 1801-2400

Vol. 11
No. 4

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health

LIBRARY U. OF I. URBANA-CHAMPAIGN

ume XI, No. 4
April

Abstract and Citation Nos. 1801 - 2400

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

Editor

Robert Love, M.D.

Jefferson Medical College, Philadelphia

Associate Editor

George P. Studzinski, M.D.

Jefferson Medical College, Philadelphia

NCI Staff Consultants

Elizabeth Weisburger, Ph.D.

Sidney Siegel, Ph.D.

THE LIBRARY OF THE
FEB 4 1974
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

LIBRARY U. OF I. URBANA-CHAMPAIGN

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Etiology Area
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication
approved by the Director of the Bureau of
the Budget on July 25, 1967.

LIBRARY U. OF I. URBANA-CHAMPAIGN

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
µg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	U	unit(s)
kg	kilogram(s)	UV	ultraviolet
LD$_{50}$	median lethal dose(s)	WBC	white blood cells (leukocytes), white blood count
m	meter(s)		
M	molar	wk	week(s)
mEq	milliequivalent(s)	wt	weight(s)
mM	millimolar	yr	year(s)
µM	micromolar		
mC,µC	milli-,microcurie(s)		

LIBRARY U. OF I. URBANA-CHAMPAIGN

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	1801-1813	271
CHEMICAL CARCINOGENESIS	(Chem)	1814-1909	274
PHYSICAL CARCINOGENESIS	(Phys)	1910-1924	295
VIRAL CARCINOGENESIS	(Viral)	1925-2043	298
IMMUNOLOGY	(Immun)	2044-2129	320
PATHOGENESIS	(Path)	2130-2142	337
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	2143-2158	340
MISCELLANEOUS	(Misc)	2159-2400	344
AUTHOR INDEX			
SUBJECT INDEX			

LIBRARY U. OF I. URBANA-CHAMPAIGN

01 HOW POLYCYCLIC HYDROCARBONS CAUSE CANCER.

(E.) Grover, P. (Chester Beatty Res. Inst., ndon, England). *New Scientist* 58(850):685-687, 73.

lycyclic hydrocarbons, which occur as common conminants of the urban environment and as constients of tobacco smoke, probably represent a real vironmental hazard to humans. The initial, basic rk on hydrocarbon carcinogenesis established at they require activation to exert their biolocal effects, which are mediated through intertion with cellular DNA. Hydroxylated derivatives, re precisely the K-region epoxides, are formed rough the action of microsomal mixed function oxises, are the active product. The K-region epoxes are electrophilic and thus readily react with cleic acid *in vitro* and *in vivo*. Preliminary studs implicate alkylation of the nucleic acid guanine ieties. Biologically, polycyclic hydrocarbons e carcinogenic, presumably as a result of their ility to induce frameshift mutations. (No references)

02 THE PREDICTIVE VALUE OF MOUSE LIVER TUMOUR INDUCTION IN CARCINOGENICITY. A LITERATURE RVEY.

(E.) Tomatis, L. (Int. Agency Res. Cancer, ons, France), C. Partensky and R. Montesano. *Int J ncer* 12(1):1-20, 1973.

survey of the available literature was conducted in attempt to verify the possible correlation between e capacity of a number of chemicals to induce renchymal liver tumors in the mouse and their pacity to induce tumors in the liver and/or other gans in the rat and hamster. Data on 58 chemicals re collected. A positive correlation appears to ist between the capacity of a chemical to induce ver tumors in the mouse and its capacity to induce mors at any site in the rat or hamster. The strongt correlation exists with chemicals which induce th liver and other tumors in adult mice of both xes. Induction of liver tumors in mice by a chemil does not necessarily signify that the liver would the target organ in the rat or hamster. Of the chemicals considered, seven (3-4-benzo(a)pyrene, aminobiphenyl, benzidine, auramine, 2-naphthylamine, ilbestrol and aflatoxin) are established or suscted human carcinogens. All were hepatocarcinonic in mice and six were carcinogenic in the liver d/or other organs in the rat. Four were tested in msters and found to be carcinogenic. (369 references)

03 HUMAN LEUKAEMIA: RECENT TISSUE CULTURE STUDIES ON THE NATURE OF MYELOID LEUKAEA.

(E.) Metcalf, D. (Cancer Res. Unit, Walter d Eliza Hall Inst., Melbourne, Australia). *Br Cancer* 27(3):191-202, 1973.

cent information obtained from tissue culture udies on normal and leukemic granulocytic and nocytic cells is reviewed. Semi-solid agar culre studies of normal marrow, spleen or blood ll populations have shown that monocytes and crophages are closely related to granulocytes and have a common precursor cell--the "*in vitro* colony-forming cell" (*in vitro* CFC). Colony formation *in vitro* depends on the presence of colony stimulating factor (CSF), a naturally occurring neuraminic acid-containing glycoprotein. Lipoproteins are demonstrable in normal human and mouse serum and are termed "CSF inhibitors" because they exert a species-specific blocking action *in vitro* on CSF although the inhibitors do not appear to be cytotoxic for colony-forming cells. Both CSF and the inhibitor probably operate *in vivo* to control maturation of stem cells into the monocyte-macrophage pathway. *In vitro* studies of cells from acute myeloid leukemic (AML) patients have shown that the transition from relapse to remission is associated with reemergence of a population of normal colony-forming cells and the simultaneous loss or decline of the leukemic population. Similar, although less clear, results have been obtained for chronic myeloid leukemic (CML) cells. In a study of more than 70 AML and CML patients, in every case the growth of the leukemic cells in agar was dependent on, and responded to, stimulation by CSF either as assessed by colony growth rates or the total numbers of colonies or clusters developing. This finding supports earlier observations on the responsiveness of murine myelomonocytic and myeloid leukemic cells, and strongly suggests that myeloid leukemic cells in humans rarely ever reach the theoretical end-stage of showing autonomy with respect to the normal regulator. AML patients and CML patients in terminal blast crisis have elevated serum and urine CSF levels, and half of the AML patients show decreased or undetectable CSF-inhibitor. Results from limited studies of "preleukemic" patients (i.e., with diseases predisposing to AML or CML) show early disturbances in serum and urine CSF and CSF-inhibitor levels. (74 references)

1804 BIOCHEMICAL PROPERTIES OF NORMAL AND NEOPLASTIC CELL SURFACES; A REVIEW.

(E.) Emmelot, P. (The Netherlands Cancer Inst., Amsterdam). *Europ J Cancer* 9(5):319-333, 1973.

Changes in the surface of transformed and neoplastic cells are reviewed in relation to data obtained in the author's laboratory. The particular phenotypic pattern of transformed cell growth may stem from a disorder of social interactions between the cell as a result of chemical alteration of the cell periphery. This alteration hampers the establishment between cells of functional contact, the latter being defined as that form of intercell contact that normally triggers off a series of reactions leading to the inhibition of initiation of DNA synthesis. The resemblance with embryonic surface expressions and preponderance of the growth-movement over the resting-maturated state of tumor as in early embryonic cell activity is pointed out. Three types of changes in the transformed cell surface affecting the carbohydrate components of glycosphingolipids and glycoprotein, which seem to be of more than random character are discussed. The conclusion is reached that in contrast to the dynamic surface expression of the normal cell, which modulates during the cell cycle, (part of) the surface expression of transformed or neoplastic cells is frozen in a conformation adverse to functional contact. The crucial aberration is thought to be that the tumor-cell surface is incomplete (lack of protein "cover"), or unable to modulate, or that the proper surface layer allowing functional contact is constantly being formed but digested by proteolytic action of the tumor cells. The absence of a proper configuration is considered to be of greater importance than the actual surface expression. The role of cyclic-AMP formed by cell-surface adenylate cyclase in functional contact is discussed, and the tentative conclusion is reached that tumor cells may differ from normal cells in the net formation or assimilation of this signal. (references available from author)

1805 ARE TUMORS CAUSED BY DNA VIRUSES? *(Ger.)*

Hirt, B. (Swiss Inst. Exp. Cancer Res., Lausanne, Switzerland). *Pathol Microbiol* 39(1):24-31, 1973.

Papilloma, herpes, polyoma, and SV40 viruses and adenovirus are described. All these viruses affect the nucleus and cause cell death. Papilloma virus can be isolated from warts, but warts are very rarely transformed to malignant tumors. The herpes viruses are very complex; their DNA contains more than 200 genes. Several herpes viruses are described, and possible relations between Epstein-Barr virus and Burkitt's lymphoma and between herpes virus type 2 and cervical carcinoma are discussed. So far, no tumor is known to be caused by an adenovirus, but the adeno-antigen and adeno-nucleic acids have been isolated from tumor cells. Polyoma and SV40 viruses are the smallest known tumor viruses; their DNA is ring-shaped and contains less than 10 genes. Like adenoviruses, polyoma and SV40 viruses are not known to induce natural tumors. Immunological reactions, cell transformations, and the mechanisms of cell regulation in the presence of tumor viruses are discussed. (14 references)

1806 ONCOGENESIS OF MAREK'S DISEASE. *(E.)*

Nazerian, K. (Regional Poultry Res. Lab., U.S. Dept. Agriculture, East Lansing, Mich.). *Cancer Res* 33(6):1427-1430, 1973.

Marek's disease is a lymphoproliferative disease of chickens and is caused by a herpesvirus. Attempts to show the exclusive role of either thymus or bursa of Fabricius in pathogenesis of the disease have been unsuccessful. Infection of chickens with Marek's disease virus usually remains latent and does not cause a clinical disease. Early infection with nonpathogenic strains of the virus and viral specific maternal antibody in newly hatched chicks may play an important role in the latency of the virus. (39 references)

1807 A UNIFYING CONCEPT OF CARCINOGENESIS AND ITS THERAPEUTIC IMPLICATIONS. *(E.)*

Smith, A. E. (Physics Dept., Sir George Williams U., Montreal, Canada) and D. H. Kenyon. *Oncology* 27(5):459-479, 1973.

The Warburg theory of carcinogenesis is reexamined in the light of recent information; objections are discussed and found to be not valid in view of the present data. Two general features of the Warburg theory are emphasized: 1) in a cancer cell, the coupling between respiration and glycolysis has been damaged and glycolysis operates more autonomously than it did in the normal cell; 2) glycolysis is phylogenetically a more primitive mode of energy production than respiration. Intercellular communication in normal cells and its lack in tumors is discussed. A model of carcinogenesis in which cell oscillations are related to intercellular communication is shown to have support. Several different pathways to cancer are shown to result. Therapeutic implications, including activated oxygen by means of negative ions and electromagnetic excitation as therapy, are suggested. A possible role of antioxidant vitamins in retarding cancer is considered. A unified outlook of several levels of carcinogenesis is now possible and lines of further research are suggested. (59 references)

1808 THE LECTIN BINDING AND AGGLUTINATION APPROAC FOR DETECTING ALTERATIONS IN TRANSFORMED CEL SURFACES INDUCED BY SV40 AND POLYOMA. *(E.)*

Levine, A. J. (Dept. Biochem. Sci., Princeton U., N.J.). *PAAB REV* 2(2):289-297, 1973.

While it is not clear whether cell to cell contact is responsible for density-dependent inhibition of growth *in vitro*, the term "contact inhibition" has stimulated much research on the cell surfaces of transformed and normal cells. Among many approaches, the use of plant lectins has recently received wide attention. The basic observation underlying this approach is that low concentrations of lectin will agglutinate SV40- and polyoma-transformed cells but not the normal cells from which they are derived. One of the lectins, a 26,000 molecular wt glycoprotein from wheat germ lipase, is called wheat germ agglutinin (WGA); another, a 54,000 molecular wt dimer protein from Jack bean, Concanavalin A (ConA). Trypsin treatment of normal cells renders them agglutinable, however, indicating that the receptor is present in normal cells but ordinarily unavailable for the reaction. Binding studies with isotopically labeled ConA showed that the same amount of ConA is bound to normal cells as to transformed cells, on a cell basis. Isotope binding and agglutination involve similar membrane sites. Several models have been proposed to explain at the same time the equal binding, the differences in agglutinability, and the result of trypsin treatment of normal cells. One model assumes that normal cells have "cryptic sites" that are exposed by enzyme treatment. A modified model presupposes that the lectin sites in normal cells are able to bind lectin but that trypsin-susceptible surface components located near these sites sterically block the agglutination reaction. A further modification suggests that ionic repulsion prevents agglutination of normal cells that have bound lectins and that trypsin treatment reduces such charges. A second model suggests a redistribution of lectin sites in the normal cell to a different pattern in the transformed cell or even that the lectin itself induces allosteric conversion in distribution of lectin sites on the cell membrane. A third model suggests heterogeneity of lectin sites in the membrane, the sites having different binding constants. Since agglutination reactions, compared to binding tests, do not employ washing procedures, more lectin might be bound to weak sites in the agglutination reaction, thus causing the observed agglutination. Whichever model is correct, the lectin binding and agglutination approach to cell surface alterations provides a useful tool to probe problems associated with control mechanisms of eukaryotic cells. (101 references)

1809 A BRIEF REVIEW OF THE BIOCHEMISTRY OF HERPESVIRUS-HOST CELL INTERACTION. *(E.)* Kaplan, A. S. (Vanderbilt U. Sch. Med., Nashville, Tenn.). *Cancer Res* 33(6):1393-1398, 1973.

Some of the interactions of the herpesviruses and their host cells at the molecular level are reviewed. In general, productive infection of cells with most members of the herpes group is qualitatively the same; there is an inhibition of the synthesis of cell specific macromolecules and a concomitant synthesis of virus-specific components. Cellular DNA synthesis is gradually inhibited in cells infected in logarithmic phase, and by 7 to 8 hr postinfection rabbit kidney cells infected with pseudorabies virus no longer synthesize cell-specific DNA. This inhibitory process requires protein synthesis. Several possible mechanisms for this virus-induced inhibition of cellular DNA synthesis are discussed. Infection of cells with the herpesviruses leads to a decrease in the rate of RNA synthesis. There is a rapid inhibition of the synthesis and processing of cellular ribosomal RNA. Although the synthesis of functional cell-specific messenger RNA is completely inhibited, even at relatively early stages of the infectious process, a new species of polyadenylated cell-specific RNA accumulates in the cytoplasm of infected cells. The decrease in the rate of synthesis of cellular RNA as well as the inhibition of the synthesis of cellular messenger RNA is mediated by a virus-induced protein. The type of proteins (cellular or viral) synthesized by herpesvirus-infected cells can be identified by various criteria. There is in the infected cells a gradual inhibition of cell-specific protein synthesis and a concomitant increase in the rate of synthesis of virus-specific proteins. The inhibition of cell-specific protein synthesis is a function coded by the "immediate early" viral messenger RNA. (27 references)

1810 SMOKING AND CANCER. *(E.)* Burch, P. R. J. (Gen. Infirm., Leeds, England). *Lancet* (7815):1315-1316, 1973. (10 references)

1811 TESTICULAR CANCER IN FINLAND. *(E.)* Teppo, L. (Dept. Path., U. Helsinki, Finland). *Acta Pathol Microbiol Scand* [*A*] *Suppl* 238:1-80, 1973. (170 references)

1812 ANALOGICAL CHARACTER OF SCHIZOPHRENIA AND CANCER. *(E.)* Galzigna, L. (Dept. Med. Biochem., U. Nairobi, Kenya). *Agressologie* 14(1):1-4, 1973. (8 references)

1813 CARCINOEMBRYONIC ANTIGEN (CEA): A REVIEW AND CURRENT STATUS. *(E.)* Blundell, G. P. (Doctors Hosp., Washington, D.C.). *Med Ann DC* 42(5):222-224, 1973. (12 references)

1814 ENHANCED ACTIVITY OF BENZPYRENE HYDROXYLASE IN RAT LIVER AND LUNG AFTER ACUTE *CANNABIS* ADMINISTRATION. *(E.)* Witschi, H. P. (Dept. Pharmacol., U. Montreal, Canada) and B. Saint-Francois. *Toxicol Appl Pharmacol* 23(1):165-168, 1972.

Hepatic and pulmonary microsomal benzpyrene hydroxylase activities were assayed *in vitro* 24 and 48 hr after administration by stomach tube of a crude *Cannabis* resin (30-500 mg/kg) to male Wistar rats. Crude *Cannabis* resin produced a dose-dependent induction of both lung and liver hydroxylase. The effect was similar to the induction produced by 3-methylcholanthrene, and simultaneous administration of both substances produced a slight additive effect. Hydroxylase induction by *Cannabis* was more sensitive in lung than liver. Administration of pure Δ^8- or Δ^9-tetrahydrocannabinol produced induction only in lung. The lung may thus represent an important detoxifying organ for *Cannabis*.

1815 EFFECT OF MATERNAL CIGARETTE SMOKING ON 3,4-BENZPYRENE AND *N*-METHYLANILINE METABOLISM IN HUMAN FETAL LIVER AND PLACENTA. *(E.)* Pelkonen, O. (Dept. Pharmacol., U. Oulu, Finland), P. Jouppila and N. T. Kärki. *Toxicol Appl Pharmacol* 23(1):399-407, 1972.

The effect of maternal cigarette smoking on the metabolism of 3,4-benz(a)pyrene (BP) and N-methylaniline (MA) was studied *in vitro* in human fetal liver and placenta homogenates. Spectrofluorometric assays revealed BP hydroxylase and MA demethylase activities in all fetal livers studied. Maternal smoking habits had no influence on these hepatic liver enzyme activities. Although maternal smoking had no effect on placental MA demethylase activity, which was very low during pregnancy, placental BP hydroxylase activity was increased under these circumstances. A fetal wt of about 10 gm (about 10-11 wk) was apparently critical for the appearance of placental enzyme. BP hydroxylase activity was present in all term placentas from mothers smoking more than five cigarettes per day. There was no correlation between the levels of hepatic and placental BP hydroxylase from the same mother.

1816 DOSE-RESPONSE CORRELATION FOR THE INDUCTION OF RESPIRATORY-TRACT TUMOURS IN SYRIAN GOLDEN HAMSTERS BY INTRATRACHEAL INSTILLATIONS OF BENZO(A)PYRENE. *(E.)* Feron, V. J. (Central Inst. Nutrition Food Res. TNO, Zeist, The Netherlands), and P. Emmelot. *Eur J Cancer* 9(5):387-390, 1973.

Respiratory tract tumorigenesis was studied in adult male Syrian hamsters which received once weekly intratracheal instillations of 0.0625 to 1.0 mg benzo(a)pyrene (BP) in saline for 52 wk. All animals were sacrificed and autopsied when they became moribund or after 78 wk if still living. The results showed a positive dose-response relationship with respect to an increased number of respiratory tract tumors and a decreased latent period for their development. Respiratory tract carcinomas developed only in hamsters receiving the two highest BP doses (0.5 or 1.0 mg). The most frequent non-neoplastic lesions, which also showed a positive dose-response relationship, were hyperplasia and squamous metaplasia of the tracheobronchial lining epithelium, peribronchiolar adenomatoid lesions and several types of alveolar metaplasia.

1817 INDUCTION OF CHROMOSOME ABERRATIONS IN CULTURED HUMAN LYMPHOCYTES BY 4-NITROQUINOLINE 1-OXIDE. *(E.)* Hiragun, A. (Hiroshima U. Sch. Med., Japan) and Y. Nishimoto. *Gann* 64(2):183-187, 1973.

Chromosome-type aberrations, induced by 4-nitroquinoline 1-oxide (4-NQO), in cultured human peripheral lymphocytes were examined with emphasis on stable-type chromosome aberrations which could survive mitosis and persist to become marker chromosomes in malignant transformed cells. Chromosome-type aberrations (dicentrics) were found to increase in 2-day cultures treated with 10^{-5} M 4-NQO when almost all mitotic cells are thought to be in their first mitosis *in vitro*. This suggests the possibility that 4-NQO acts on cells in G_1-S stage. Contrary to data from previous studies, the frequency of chromatid-type and chromosome-type aberrations was similar (0.046/cell and 0.035/cell, resp., in 10^{-5} M treated cultures). It is not clear why chromosome-type aberrations could not be found in Yoshida sarcoma cells or mouse bone-marrow cells. This suggests a difference in susceptibility to 4-NQO among Yoshida sarcoma cells, bone-marrow cells, and human lymphocytes. From the data which have been obtained to date, it is still not possible to deny the possibilit that in *in vitro* carcinogenesis by 4-NQO the marker chromosomes found in malignant transformed cells can arise from structurally altered chromosomes induced by the direct effect of 4-NQO.

1818 INCREASED CARCINOGENIC ACTION OF DIMETHYLNITROSAMINE AFTER PRIOR ADMINISTRATION OF CARBON TETRACHLORIDE. *(E.)* Pound, A. W. (Dept. Path., U. Queensland, Brisbane, Australia), T. A. Lawson and L. Horn. *Br J Cancer* 27(6):451-459, 1973.

The occurrence of tumors of the liver and kidney induced in rats by a single dose of dimethylnitrosamine (DMN, 20 mg/kg body weight) given a short interval after a single necrosis-producing dose of carbon tetrachloride (CCl_4) is examined. Male, Sprague-Dawley rats were given DMN alone or 42 or 60 hr after a non-lethal hepatotoxic dose of CCl_4 and killed 12 months later. DMN alone produced no tumors in the kidney and a few in the liver, but when given 42 hr after CCl_4, tumors formed in the kidneys and the number in the liver was increased. When given after 60 hr, the incidence of kidney tumors was less but that of liver tumors was further increased. A larger dose of DMN (40 mg/kg) was tolerated 42 hr after CCl_4 and enhanced the number of kidney and liver tumors, the latter apparently due to an increased proportion of cholangiomata. Numerous small focal proliferations of a typical liver cells and of bile duct epithelium were observed after treatment with DMN. The incidence of

ıe lesions in the different experimental treat-
ts varied in a similar manner to the liver tumors.
ıeems possible that tumors may arise as a result
continued growth of these focal proliferations or
ernatively that they are susceptible to a further
ıge leading to a neoplastic type of growth. It
ıot known if these focal proliferations may be
ı soon after a single dose of DMN, nor is it
m if they continue to grow, remain static in
e after a time or finally regress.

9 INHIBITION OF *IN VITRO* PROTEIN SYNTHESIS BY AFLATOXIN B_1 DERIVATIVES.

(E.) Sarasin,
(Inst. Sci. Res. Cancer, Villejuif, France) and
Moule. *FEBS Letters* 32(2):347-350, 1973.

effects of aflatoxin B_1 and several of its meta-
ites were studied on protein synthesis in a cell-
e rat liver polysome system. Crystalline afla-
in B_1 showed no effect on the *in vitro* incorpora-
ı of ^{14}C-leucine when compared with untreated con-
l preparations. However, aflatoxin B_1 "activated"
incubation *in vitro* in a rat liver microsome sys-
containing NADPH was able to inhibit incorpora-
ı of amino acid by 27-77%. Although thin layer
omatographic analysis of the "activated" prepara-
ı showed the presence of aflatoxin M_1 and five
er substances, the active compound was not iden-
ied. These results support the hypothesis that
atoxin B_1 derivatives have a direct effect on
ysome function which is independent of an effect
transcription.

0 THE FORMATION OF CARCINOGENIC HYDROCARBONS DURING HEATING OF FOODS. 5. CONTAMINATION
GRILLS OVER CHARCOAL.

(Ger.) Fritz, W. (Central
t. Nutrition, German Acad. Sci., Berlin, Germany).
ch Lebensmittel-Rundschau 69(3):119-122, 1973.

st beef, pork chops and sausages were grilled
h on a conventional and a new type of camping
ll over commercial charcoal, charcoal from
apped railway crossbars, paper or pine cones.
benzo[a]pyrene content was the same in pro-
ts grilled with the two types of charcoal. The
tent of benzo[a]pyrene in 1 kg of sausage was
2 µg, in fat pork loin 6-8 µg, in lean pork
n 4.2-4.5 µg and in roast beef 0.6-0.8 µg. Paper
pine cones, however, caused considerably more
tamination with carcinogenic hydrocarbons:
zo[a]pyrene contents were between 18 and 140
kg. The content of other hydrocarbon compounds,
. anthracene, pyrene, phenanthrene, benz[a]
hracene or fluoranthene was also higher in the
ducts grilled over paper and pine cones.

1 ABSENCE OF CORRELATION BETWEEN HAEMOLYSING SPLENIC CELL NUMBERS AND RESISTANCE TO
CER INDUCTION BY METHYLCHOLANTHRENE.

(E.) Oth,
(Exp. Cancer Radiobiol. Res. Unit, Vandoeuvre-les-
cy, France), D. Sabolovic and F. Robert. *Beitr*
hol 149(1):60-69, 1973.

carcinogenic effect of a given dose of MCA was
studied as a function of the immune status of an untreated category of mice, of the degree of immunodepression elicited by this dose in animals of the same normal status, and of the intensity of the adjuvant effect in counteracting the immunodepression. Accordingly, groups of mice presenting different levels of immune reactivity (according to age, strain and treatment with *Brucella abortus*), were injected with individual doses of 0.1 or 0.01 mg of methylcholanthrene (MCA). The number of splenic cells capable of forming plaques of hemolysin (PFC), and the number of cancers developed, were considered in each group. No correlation was found between PFC number, alterations of PFC number due to MCA immunodepression or to *Brucella* treatment, and susceptibility to MCA carcinogensis.

1822 THE PATTERN OF METHYLATED PURINES FORMED IN DNA OF INTACT AND REGENERATING LIVER OF RATS TREATED WITH THE CARCINOGEN DIMETHYLNITROSAMINE.

(E.) Craddock, V. M. (Med. Res. Council Lab., Carshalton, Surrey, England). *Biochim Biophys Acta* 312(2):202-210, 1973.

The methylated bases formed in DNA after treatment of intact rats with dimethylnitrosamine, and after treatment of partially hepatectomized rats under conditions known to induce liver cancer, were studied. Di[^{14}C]methylnitrosamine was given to normal female, Wistar albino rats and also 24 hr after partial hepatectomy, and liver DNA was isolated 5-7 hr and 24 hr after treatment. O^6-Methylguanine had been formed. This base had not been detected previously in DNA of animals treated with dimethylnitrosamine owing to its acid lability. No O^6-methylguanine was detected after treatment with methyl methanesulphonate, a methylating agent found not to be a liver carcinogen even when given after partial hepatectomy. The amounts of 7-methylguanine, O^6-methylguanine, 3-methyladenine, 1-methyladenine and 3-methylguanine were similar in DNA isolated from intact and from regenerating liver. The results suggest that, for carcinogenesis to ensue, O^6-methylguanine must be present in DNA at the time of DNA replication. The methylated base is then likely to cause miscoding during transcription, so that the transient abnormality of methylation would be converted into a permanent inherited change in the genetic material.

1823 COMPARISON OF IMMUNOLOGICAL AND MORPHOLOGICAL CHANGES OCCURRING IN LIVER TISSUE AFTER β-NAPHTHYLAMINE ADMINISTRATION.

(Rus.) Korosteleva, T. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR), A. F. Kondrat'yeva and A. P. Skachkov. *Vopr Onkol* 19(3):66-72, 1973.

β-Naphthylamine was administered daily in food to dogs (50 mg/kg) and to mice (2 mg/kg). The mice were killed after 4, 15, 30, and 60 days of this diet and tissue extracts were obtained. The dogs were killed after 30 days by bleeding from the carotid artery and soluble antigens from various tissues were obtained. Tissue extracts from intact animals served as controls. β-Naphthylamine-haptene was assayed with rabbit antisera against heterologous β-naphthylamine-azoproteins. Morphological examinations were made on the livers of mice and dogs which had received β-naphthylamine. β-Naphthylamine-haptene was detected in dog serum and kidneys, and in mouse serum and liver. Livers of dogs were microscopically within normal limits 30 days after feeding β-naphthylamine. In the same period, mice showed dystrophic changes in the liver. Analogous changes in the kidneys were seen in dogs only after 200 days, while after 200 days, the mice had developed diffuse hyperplasia of the liver.

1824 HISTOPATHOLOGY AND CYTOGENETICS OF TUMORS INDUCED BY THE APPLICATION OF 7,12-DIMETHYLBENZ[a]ANTHRACENE (DMBA) IN MOUSE CERVIX.

(E.) Joneja, M. G. (Dept. Anat., Queen's U., Kingston, Ontario, Canada) and D. B. Coulson. *Eur J Cancer* 9(5):367-374, 1973.

DMBA (7,12-dimethylbenz[a]anthracene)-coated threads were placed in the vaginal fornix of 4-5 wk-old ICR mice. Malignant tumors developed in 56% of the 126 animals after an average latent period of 22.8 ± 12.4 wk. The tumors included squamous cell carcinomas (37%), fibrosarcomas (34%), leiomyosarcomas (5%), lymphosarcomas (4%), mixed tumors (16%) and double tumors (4%). No tumors appeared in 54 sham operated control mice. Cytogenetic analysis of three fibrosarcomas, two squamous carcinomas and one lymphosarcoma revealed a wide range of karyotypic composition. The two fibrosarcomas were predominantly diploid, while the third had a bimodal distribution in the tetraploid and octaploid range. One squamous carcinoma was tetraploid and the other aneuploid with 41 chromosomes in modal cell line. The lymphosarcoma had a heterogeneous cell population with a majority of the metaphase plates containing four metacentric chromosomes. The variation in the cytogenetic pattern of these tumors was in contrast with that of thymic lymphosarcomas induced in mice by the same carcinogen. The occurrence of a wide variety of neoplasms indicates that DMBA has the ability to become dispersed throughout the body and affect a large spectrum of tissues and organs.

1825 TRANSPORT OF ORALLY ADMINISTERED 9,10-DIMETHYL-1,2-BENZANTHRACENE IN THE SPRAGUE-DAWLEY RAT.

(E.) Grubbs, C. J. (U. Tennessee Med. Units, Memphis) and R. C. Moon. *Cancer Res* 33(7): 1785-1789, 1973.

The extent to which 9,10-dimethyl-1,2-benzanthracene (DMBA) is carried in the lipid fractions of blood and deposited in target tissues was studied in adult female Sprague-Dawley rats. Rats which had undergone previous thoracic duct cannulation received a mixture of 20 mg DMBA and ^{14}C-DMBA via gastric fistula. The chylomicrons were collected over a 24 hr period, disrupted by freeze-thawing, and analyzed by autoradiography following separation by thin-layer chromatography. The results indicated that DMBA remained as an intact molecule in the lymphatic system and that it did not associate with any particular lipid fraction. In other experiments, animals received hourl injections of either NaCl, protamine sulfate, or heparin starting 1 hr before and lasting 5 hr afte DMBA intubation. Analysis of radioactivity in plasma and various organs showed that both protami and heparin decreased DMBA uptake by mammary gland, gastrointestinal tract, liver, and fat parenchymal cells. Whereas protamine increased the relative level of circulating plasma DMBA, heparin had the opposite effect. These results indicate that chylomicrons play an integral part in the transport of DMBA.

1826 EFFECTS OF NEONATALLY ADMINISTERED SEX STEROIDS ON 7,12-DIMETHYLBENZ(A)-ANTHRACENE-INDUCED MAMMARY NEOPLASIA IN RATS.

(E. Shellabarger, C. J. (Brookhaven Natl. Lab., Upton, N.Y.) and V. A. Soo. *Cancer Res* 33(7):1567-1569, 1973.

The effect of sex steroids on 7,12-dimethylbenz(a)-anthracene (DMBA)-induced mammary carcinogenesis was studied in female Sprague-Dawley rats. Animals which received an s.c. injection of estradiol (1.0 mg) or testosterone (1.25 mg) five days after birth and DMBA (20 mg) by gastric intubation 50 days later had an increased incidence of mammary fibroadenomas and a decreased incidence of mammary adenocarcinomas when killed at 190 days of age compared with animals receiving only DMBA. Both steroid groups had fewer developing ovarian follicles than controls and no corpora lutea. Only the testosterone-treated animals lactated. Treatment with progesterone (1.25 mg) on day 5 followed by DMBA treatment resulted in an increased mammary adenocarcinoma, but not fibroadenoma, response. Progesterone had no effect on ovarian structure.

1827 LIFELONG EXPOSURE OF SPF RATS TO AUTOMOTI EXHAUST GAS. DILUTION CONTAINING 20 PPM OF NITROGEN OXIDES.

(E.) Stupfel, M. (Air Polluti Res. Ctr., Paris, France), M. Magnier, F. Romary, M.-H. Tran and J.-P. Moutet. *Arch Environ Health* 26(5):264-269, 1973.

Pathogen-free Sprague-Dawley rats (SPF) were exposed to automotive exhaust gas from 2½ months to lifetime (two years) for six hours per day, five days each week. The exhaust gas contained 50 ppm of carbon monoxide and two different concentrations of nitrogen oxides (NO_x) (0.2 and 23.0 ppm), carbon dioxide (0.065% and 0.370%), and aldehydes (0.1 and 2.0 ppm). Only the dilution with the highest content of NO_x (23 ppm) produced biological effects: decrease of body weight, diminution of the sound avoiding reflexes, and increase in the number of spontaneous tumors. However, this dilution does not influence heart rate, ventricular complex (QRS) II electrocardiographic deflection, the rats' survival, or the renal and aortic lesions resulting from aging processes, although the emphysematous process may be accelerated. Incidental findings among exposed rats included a lymphosarcoma of the testis, a hemangioma of the neck, a sarcoma of the gut, and adenoma of the prostate. In the two control series

ch were not exposed to automotive exhausts, a
static adenoma, a sarcoma of the gut, an abscess
the parotid, and subcutaneous abscesses were dis-
ered. No tumor or abscess of the lung was ob-
ved in any animal.

3 ISOLATION OF CARCINOGENIC AMINOAZO DYE-BINDING PROTEIN AND ITS IDENTIFICATION
LCOHOL DEHYDROGENASE. *(E.)* Tokuma, Y.
logical Inst., Fac. Sci., U. Tokyo, Japan) and
erayama. *Biochem Biophys Res Commun* 54(1):341-
1973.

jor carcinogenic aminoazo dye-binding protein
ng Ip of 9.7 (isolectric focusing) was isolated
the liver cystosol of male, Wistar rats given
g 3'-methyl-4-dimethylaminoazobenzene. The
ein has the molecular wt of 6.8 x 10^4 (gel-filtra-
) and two subunits of about 3.9 x 10^4 molecular
SDS-polyacrylamide gel electrophoresis). The
o acid composition was similar to that reported
liver alcohol dehydrogenase of animals. The enzy-
c activity was shown to be associated consistently
the dye-binding protein fractions throughout the
fication steps suggesting identity of the dye-
ling protein as liver alcohol dehydrogenase.

) VARIATIONS IN EXTENT OF ARYL HYDROCARBON HYDROXYLASE INDUCTION IN CULTURED HUMAN
HOCYTES. *(E.)* Kellermann, G. (M. D. Anderson
. Tumor Inst., Houston, Texas), E. Cantrell
C. R. Shaw. *Cancer Res* 33(7):1654-1656, 1973.

pheral lymphocytes cultured from 103 healthy
e and female adults were tested for *in vitro*
ction of aryl hydrocarbon hydroxylase (AHH)
ivity by 3-methylcholanthrene (3MC). Exposure
.75 μM 3MC for 24 hr produced a two- to five-
increase in AHH specific activity over the
rol level of 0.03-0.04 u/10^6 cells. Although
degree of induction varied from individual
ndividual, that for a given person was highly
oducible. There was no significant sex difference
he extent of induction. Similar results were
ined with other inducers such as benzanthra-
e and PPO (undefined). No difference in the ex-
of induction was observed between smokers and
mokers. The high degree of reproducibility of
induction for individuals suggests that it is
tically controlled.

) CARCINOGENESIS BY URETHAN VIA MOTHER'S MILK AND ITS ENHANCEMENT OF TRANSPLACENTAL CAR-
GENESIS IN MICE. *(E.)* Nomura, T. (Osaka U. Med.
, Japan). *Cancer Res* 33(7):1677-1683, 1973.

onary tumors were induced in mice that were
led by mothers that received urethan during lac-
on. The earlier the treatment while nursing,
greater the number of tumors induced in sucklings.
pregnant mice were treated with urethan during
ation and lactation their offspring had a signi-
ntly increased incidence of pulmonary tumors,
the number of tumors per mouse was compatible
the sum of tumors in mice who received either
prenatal or postnatal treatment. However, when prenatal urethan treatment was coupled with postnatal exposure via mother's milk, offspring of mothers treated on Day 9 or 15 of gestation bore more tumors than the sum. These two days correspond to the most sensitive stages to urethan for lung malformation and carcinogenesis. Induction of ovarian cystadenomas might also suggest that subtumorigenic effects of urethan on embryonic cells of the developing female gonad on Day 11 and 13 were made manifest by the postnatal exposure. The same was observed in induction of cystic kidneys on Day 11 of gestation. However, manifestation was not observed so clearly in induction of hepatomas. It is assumed that transplacental exposure to urethan from Days 11 to 15 is a major cause of hepatocarcinogenesis in mice. Prenatal and postnatal exposure to urethan may suppress the induction of lymphosarcomas (node type). Mothers developed endometrial hyperplasias and uterine hemangiomas when treated during pregnancy. Additional treatment after delivery increased the incidence of uterine hemangiomas.

1831 CARCINOGEN-BINDING PROTEIN DISTRIBUTION IN PRIMARY LIVER TUMORS INDUCED IN RATS BY 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE. *(Rus.)* Bannikov, G. A. (Inst. Exp. Clin. Oncol., Moscow, USSR), V. I. Gel'shteyn and T. A. Chipysheva. *Biull Eksp Biol Med* 75 (3):90-92, 1973.

Primary liver tumors were induced in Wistar rats by 3'-methyl-4-dimethylaminoazobenzene (0.6 g/kg food). Multiple liver tumors, which developed in rats fed this diet for 4.5 months, included 4 highly differentiated trabecular hepatomas; 13 mixed tumors consisting of adenocarcinomas, poorly differentiated hepatomas and anaplastic carcinomas; and 2 anaplastic carcinomas. Sections from these 19 primary liver tumors were examined by an immunofluorescent method, using monospecific antibodies to the carcinogen-binding protein. The level of this protein was directly related to the degree of tumor cell differentiation. The carcinogen-binding protein was absent in poorly differentiated hepatomas, anaplastic carcinomas, and adenocarcinomas but was present in highly differentiated hepatomas, in which it was evenly distributed in all cell populations.

1832 IMMUNOGENIC STRENGTH OF SARCOMAS INDUCED BY METHYLCHOLANTHRENE IN MILLIPORE FILTER DIFFUSION CHAMBERS. *(E.)* Parmiani, G. (Natl. Inst. Study, Cure Tumors, Milan, Italy), G. Carbone and R. Lembo. *Cancer Res* 33(4):750-754, 1973.

Normal BALB/c fibroblasts enclosed in two-compartment diffusion chambers with a methylcholanthrene (MCA) disc were left in the peritoneal cavity of BALB/c mice or were incubated *in vitro* for 4 to 24 weeks while protected from the immunoselective process. Protection was demonstrated by the fact that cells of a BALB/c sarcoma induced by MCA, which were immunogenic when injected s.c., were unable to sensitize when grown for 2 weeks within 0.22-μm filter diffusion chambers placed in the peritoneal cavity of syngeneic mice. The chambers with the fibroblasts exposed to MCA were recovered and opened at 4-week intervals; their contents were implanted s.c. into immunodepressed syngeneic or semisyngeneic mice. In 41 to 63% of the cases, the *in vivo* and *in vitro* diffusion chamber cultures gave rise to sarcomas. When the diffusion chambers were made with 1.2-μm, lymphocyte-permeable filters and were kept *in vivo*, only 10% of the cultures produced tumors. Fifty tumors were tested for immunogenicity by transplantation methods and 45 were found to be immunogenic. The immunogenic strength, however, showed a large variability without any correlation with the latent period, length of exposure to the carcinogen, or growth rate. It is concluded that an immunological control operates during MCA carcinogenesis but that other factors influence the immunogenic strength of these sarcomas.

1833 PROTEINS FROM DIFFERENT CLASSES OF LIVER NUCLEI IN NORMAL AND THIOACETAMIDE-TREATED RATS. *(E.)* Gonzalez-Mujica, F. (Dept. Biochem., U. Coll. London, England) and A. P. Mathias. *Biochem J* 133(3):441-455, 1973.

The synthesis and absolute amounts of the four different types of nuclear proteins of liver were studied in male Wistar rats which had received a diet containing thioacetamide for 2 wk. One hr before sacrifice, control and treated animals were injected i.p. with ^{14}C-amino acids. After sacrifice, the livers were removed, the different classes of liver nuclei were separated by zonal centrifugation, and the proteins were isolated and analyzed by SDS-polyacrylamide gel electrophoresis and electrofocusing electrophoresis. The proteins isolated from the various classes of normal liver nuclei showed a great deal of heterogeneity which was most evident between the nonhistone chromosomal proteins of stromal and hepatic parenchymal cells. Migration patterns also differed for nonhistone proteins between diploid and tetraploid parenchymal nuclei, and these, in turn, differed from those of nuclei undergoing DNA replication. The protein class with the highest specific activity was residual protein in all types of nuclei except those replicating DNA where it was soluble protein. Complex and nonuniform changes, which depended on the age of the animal and the type of nucleus, occurred in the liver of treated rats when compared with the migration patterns from normal controls. In general, hepatic nuclei of treated animals showed increased labeling of the soluble and nonhistone chromosomal proteins and decreased labeling of residual proteins.

1834 COMPETITIVE RNA-DNA HYBRIDIZATION OF NUCLEAR AND MICROSOMAL RNA DURING DIETHYLNITROSAMINE HEPATOCARCINOGENESIS. *(E.)* Garrett, C. T. (U. Wisconsin Med. Sch., Madison), C. Katz, R. E. Moore and H. C. Pitot. *Cancer Res* 33(7):1662-1669, 1973.

Competitive RNA-DNA hybridization studies were conducted to determine the patterns of RNA synthesis in the livers of male Charles River white rats given weekly i.p. injections of diethylnitrosamine (DEN) for 2, 4, 6, 8, or 10 wk. Animals were sacrificed 2 wk after the final DEN injections and the hepatic nuclear RNA (nRNA) and microsomal RNA fractions wer isolated. Competition of nRNA from treated and con trol rats against normal *in vivo* ^{3}H-orotate-labeled nRNA for hybridization to rat liver DNA showed no significant differences in nucleotide sequences between normal RNA and that in animals treated from 2 through 8 wk with DEN. However, after 10 wk the liver of DEN-treated rats showed a qualitative loss of nRNA species which correlated with the first appearance of histologically identifiable primary hepatomas. These nRNA species were apparently simi lar or identical to those missing in the 5123C minimal-deviation Morris hepatoma. After 2, 4, and 10 wk of DEN injections, microsomal RNA from livers of treated rats contained sequences not found in normal liver microsomal RNA. Cytoplasmic RNA sequence changes were evident as early as 2 wk after the start of DEN injections. This fact plus the absence of any changes in RNA sequences detected in the nucleus after 10 wk of DEN injections supports the hypothesis that an alteration in the nuclear to cytoplasmic transport of mRNA or cytoplasmic stabilization of mRNA, rather than changes in transcription rates, is the primary event in the initiation of the neoplastic transformation in liver.

1835 TRANSFORMATION OF RAT AND MOUSE EMBRYO CE BY A NEW CLASS OF CARCINOGENIC COMPOUNDS LATED FROM PARTICLES IN CITY AIR. *(E.)* Gordon, R. (U. Southern California Sch. Med., Los Angeles), R. Bryan, J. S. Rhim, C. Demoise, R. G. Wolford, A. E. Freeman and R. J. Huebner. *Int J Cancer* 12(1):223-1973.

The residue from the benzene extraction of airborne particulate matter collected in Los Angeles contain substances soluble in methanol, some of which are organic. This methanol extract and a number of its component fractions were tested in rodent cell cult systems developed as assay methods for screening ca cinogens. In one system a high-passage Fischer rat embryo cell line was used; in the other a low-passa cell line derived from NIH Swiss albino mouse embry and infected with AKR leukemia virus. In both syst the methanol extract showed cell transformation act vity approaching that of 3-methylcholanthrene. In the mouse cell system the activities of several fra tions were also examined. The major inorganic component, ammonium nitrate, showed no activity. Neit the neutrals alone nor the recombined non-neutral fractions separately showed any activity, although they were active together. The basic materials sep rated by ion exchange were also active. The methan extract combined with the conventional benzene extr showed much lower activity, 50 μg/ml, than either extract alone, 1 μg/ml.

1836 VASCULAR TUMORS IN FEMALE SWISS MICE AFTE INTRAPERITONEAL INJECTION OF DIMETHYLNITR SAMINE. *(E.)* Cardesa, A. (U. Nebraska Med. Ctr., Omaha), P. Pour, J. Althoff, and U. Mohr. *J Natl Cancer Inst* 51(1):201-208, 1973.

Intraperitoneal injection of 6 mg/kg body weight di methylnitrosamine (DMN) once weekly for 10 wk to male and female Swiss mice resulted in a statistica

significant increase in the incidence of vascular
nors, mainly in the retroperitoneum, in females.
ere was a low incidence of hepatic vascular tumors
both sexes. Histologic patterns of vascular
nors are described. The mechanism by which DMN
nances blood vessel tumorigenesis in female Swiss
ce is not known. However, since estrogens are
activated in the liver, the high incidence of
dominal extrahepatic tumors in contrast to the
v rate of hepatic tumors in treated females could
attributed to an estrogenic influence on DMN
tabolism.

37 RESPIRATORY TRACT TUMORS IN HAMSTERS
INDUCED BY BENZO(a)PYRENE. *(E.)* Henry,
C. (Life Sci. Res., IIT Res. Inst., Chicago, Ill.),
D. Port, R. R. Bates, and D. G. Kaufman. *Cancer*
s 33(7):1585-1592, 1973.

nzo(a)pyrene (BP), prepared as a colloid suspension
fine particles in 0.5% gelatin-0.9% NaCl solution,
s administered by intratracheal instillation to
male and 50 female Syrian golden hamsters. The
ntrol group, which consisted of 25 males and 25
nales, received 0.5% gelatin-0.9% NaCl solution.
e hamsters were treated weekly for 8 wk and re-
ived a total dose of 111 mg of BP. BP induced
spiratory tumors in 26 of the 65 hamsters examined.
ese tumors included squamous cell and anaplastic
rcinomas, adenocarcinomas, fibrosarcomas, adenomas,
lyps, and papillomas. Each segment of the respira-
ry tract, with the exception of the bronchi, had
similar incidence of tumors. Only two tumors were
agnosed in the bronchial region. Of 47 control
msters, one animal carried a tracheal polyp. Rate
clearance of BP from lung after a single admini-
ration was dependent on the dose of carcinogen.
v doses of BP were cleared rapidly while the
ekly dose instilled in this study was retained for
long time. Thus respiratory tract tumors can be
duced in hamsters by the intratracheal admini-
ration of BP without the simultaneous administra-
on of a carrier dust or surface-active agent. It
also apparent that the physical characteristics
suspensions can influence the incidence and dis-
ibution of respiratory tract tumors. The amount
BP acting at a particular site rather than the
tal dose administered defines the threshold dose
r lung carcinogenesis.

38 TRANSPLACENTAL EFFECT OF SOME CARCINOGENIC
SUBSTANCES ON EMBRYONIC LUNG ORGAN CULTURES
GOLDEN HAMSTERS. *(Rus.)* Kolesnichenko, T. S.
nst. Exp. Clin. Oncol., Moscow, USSR). *Vopr Onkol*
(10):48-52, 1972.

study was made of transplacental effects of nitro-
methylurea (NMU), diethylnitrosamine (DENA), and
12-dimethylbenz[a]anthracene (DMBA) on lung tissues
golden hamsters. Pregnant hamsters received: (1)
mg DMBA s.c.; (2) 0.02 mg DENA s.c.; and (3) 3 mg
U s.c. Embryonic lung tissues were isolated from
ese hamsters for culturing. Most of the experimen-
l cultures (about 1000) died soon after culturing due
to toxic effects of DMBA and DENA. Only 88 explants were successfully cultured in 8-25 days. Of 42 explants from hamsters receiving NMU, 18 cultures (42.9%) cultures displayed dystrophic changes in the bronchial epithelium; six cultures (14.3%) showed diffuse and localized hyperplasia of the epithelium. Of 23 explants exposed to DENA, 14 cultures (60.9%) showed dystrophic changes; five (21.7%) showed local hyperplasia of the bronchial epithelium. Of 23 explants exposed to DMBA, 10 (43.5%) showed dystrophic changes; 8 (34.8%) exhibited local hyperplasia of the epithelium. From these findings, it was concluded that NMU, DENA, and DMBA have toxic effects on embryonic lung tissues of hamster embryos. In surviving cultures, there was local proliferation of the bronchial epithelium, indicating precancerous changes.

1839 SPECTROPHOTOMETRIC STUDIES OF THE INTERACTION OF BENZO[a]PYRENE AND ITS NONCARCINOGENIC ISOMER WITH DNA. *(Rus.)* Mayevskiy, A. A. (Inst. Biol. Phys., Pushchino, USSR), M. M. Vilenchik, I. M. Mirson and B. I. Sukhorukov. *Biofizika* 18(2):371-374, 1973.

Molar extinction coefficients were determined for benzo[a]pyrene and its noncarcinogenic isomer, benzo[e]pyrene, in complexes with DNA. There were considerable differences in the absorption band intensities of these complexes and absorption band intensities of the hydrocarbons themselves dissolved in ethanol. Changes in the absorption spectra of the two complexes, studied under conditions of photoradiation, were due to the covalent binding of hydrocarbons to DNA. Exposure of these complexes in the spectral region corresponding to the first electron transfer of the hydrocarbon revealed that the maximum amount of benzo[a]pyrene and benzo[e]-pyrene bound to DNA was 50 and 14%, resp. of the amount of the complexed hydrocarbons. A new band with a ν max of 21 x 10^3cm^{-1} was identified after photoradiation of the benzo[a]pyrene-DNA complex. Irradiation of the hydrocarbon-DNA complex with a wave length corresponding to the first DNA singlet electron transfer revealed that DNA bound only benzo[a]pyrene. The nature of the spectral changes of the complex differed from those occurring upon irradiation in absorption bands of benzo[a]pyrene. These findings show the different mechanisms involved in the reactions between benzo[a]pyrene and DNA induced by light and the roles of these reactions in carcinogenesis.

1840 ELECTRON MICROSCOPIC STUDY OF LIVER LESIONS, ESPECIALLY HEPATIC MEGALOCYTOSIS INDUCED BY 1-(4-DIMETHYLAMINO-BENZYLIDENE)-INDENE (DABI) IN MICE. *(E.)* Polyzonis, M. (Lab. Path., U. Thessaloniki, Greece) and C. G. Gabrielides. *Pathol Eur* 7(3-4):229-235, 1972.

Livers of male and female inbred AKR/Sy mice fed a diet containing 0.02% DABI for six months were examined at autopsy for evidence of ultrastructural or light microscopic changes. The most striking light microscopic change was an increased size of hepatocytes ("megalocytes") and the presence within them of huge nuclei which often contained inclusions. These inclusions proved, when seen under the electron microscope, to be cytoplasmic invaginations bounded by double membranes and containing lysosomes, distended vesicles and glycogen, but no mitochondria or rough endoplastic reticulum. The nucleoli were multiple and enlarged, and their appearance was reminiscent of segregation. Nuclei also contained dense, electron opaque finely granular material surrounded by rows of granules and associated with the nucleoli. In contrast, hepatoma cells were more regular looking than the megalocytes. They never exhibited nuclear invaginations or the intranuclear electron opaque bodies. These results do not support suggestions that hepatic megalocytosis plays a role in liver carcinogenesis.

1841 INHIBITION BY URETHAN OF THE SYNTHESIS OF FREE AND MEMBRANE-BOUND RIBOSOMAL RNA IN REGENERATING LIVER. *(E.)* Glazer, R. I. (Dept. Pharmacol. Emory U., Atlanta, Ga.). *Cancer Res* 33(7):1759-1765, 1973.

The response of normal and regenerating liver to the hepatocarcinogen urethan was evaluated by means of an examination of the synthesis of free and membrane-bound ribosomal RNA. Partial hepatectomy of male Sprague-Dawley rats weighing 125 to 150 g results in a five- to ninefold elevation of the incorporation of orotic acid-5-^{3}H into free and membrane-bound ribosomal RNA 18 hr after operation; no noticeable changes occurred in the specific activity of uridine triphosphate during this regenerative period. Treatment of animals with 1 g of urethan per kg, 6 or 12 hr after operation, resulted in 50% inhibition of the incorporation of orotic acid into free and membrane-bound ribosomal RNA in partially hepatectomized rats but not in sham-operated animals. Administration of an equivalent dose of the noncarcinogenic analog methyl carbamate did not affect RNA synthesis in either group of animals. The specific activity of uridine triphosphate in 18-hr regenerating liver was not significantly altered by treatment of animals with urethan 6 hr after operation. Fractionation of ribosomal RNA into 18S and 28S components revealed that inhibition of incorporation of orotic acid-5-^{3}H by urethan was equally pronounced on both constituents. Analysis of nucleolar RNA by double labeling with methionine-methyl-^{3}H and orotic acid-6-^{14}C showed preferential inhibition by urethan of both methylation and synthesis of nucleolar RNA but that not of extranucleolar RNA. These results are consistent with the differential inhibition by urethan of the synthesis of ribosomal precursor and ribosomal RNA of regenerating liver but not of normal liver.

1842 INHALATION OF TOBACCO SMOKE BY PIPE AND CIGAR SMOKERS. *(E.)* Castleden, C. M. (St. Bartholomew's Hosp., London, England) and P. V. Cole. *Lancet* (7819):21-22, 1973.

Venous blood carboxyhemoglobin (COHb) and cholesterol levels were studied in 11 male pipe and cigar smokers who had never previously smoked cigarettes ("primary" smokers) and in 39 male pipe and cigar smokers who had switched from cigarette smoking ("secondary" smokers). Spectrophotometric assay showed that the mean COHb level of primary smokers (2.3%) was similar to that or urban nonsmokers, whereas that of secondary smokers (5.2%) was significantly ($P < 0.0005$) higher and comparable to that of cigarette smokers. Standard chemical analysis of blood cholesterol showed no correlation with venous COHb levels in any group. Venous COHb levels of secondary smokers tended to be highest in those who smoked more and who inhaled. It was thus concluded that little is gained by cigarette smokers who switch to pipes or cigars.

1843 THERMAL ENHANCEMENT OF DNA DAMAGE BY AN ALKYLATING AGENT IN HUMAN CELLS. *(E.)* Bronk, B. V. (Biol. Div., Oak Ridge Natl. Lab., Tenn.), R. J. Wilkins and J. D. Regan. *Biochem Biophys Res Commun* 52(3):1064-1070, 1973.

The effect of temperature on methyl methanesulfonate (MMS)-induced DNA strand breakage in cultured human diploid foreskin fibroblasts was studied. Cells prelabeled with either ^{3}H-thymidine or $^{32}PO_4$ were incubated at various temperatures during and after MMS treatment and the number of single-strand breaks was estimated by alkaline sucrose sedimentation. MMS-induced DNA damage was enhanced by temperatures above 37 C when compared with controls. The inability of treated cells to repair MMS-induced DNA strand breaks above 41.5 C suggested that a critical repair enzyme was being inactivated. The enhancement of MMS-induced DNA damage was temperature dependent and particularly evident above 42 C. Similar results were obtained with cultured Chinese hamster cells. Temperatures which enhanced MMS-induced DNA single strand breakage also increased that death rate of treated cells. These results indicate that chemotherapeutic treatment of tumors may be rendered more effective if the area of the tumor is heated mildly.

1844 THE ULTRASTRUCTURE OF RENAL NEOPLASMS INDUCED BY AFLATOXIN B$_1$. *(E.)* Merkow, L. P. (Allegheny Gen. Hosp., Pittsburgh, Pa.), S. M. Epstein, M. Slifkin and M. Pardo. *Cancer Res* 33(7):1608-1614, 1973.

Electron and light microscopic studies were conducted on renal carcinomas induced in male Wistar rats fed a diet containing 1-3 ppm purified aflatoxin B$_1$ for 391 days. The gross and light microscopic appearances of the tumors were as previously reported. Survey of the electron micrographs indicated that the tumor cells were well-differentiated and essentially retained the characteristics of normal renal cells. However, the renal carcinoma cells showed a lack of orientation of their brush borders, with no predilection for a normal apical orientation. Mitochondria were generally distributed throughout the tumor cell cytoplasm and were not concentrated in any particular area. Many mitochondria had a vesicular appearance resulting from loss of the granular matrix. These characteristics were consistent with a tubular origin for these tumor cells.

45 NONHISTONE CHROMATIN PROTEIN PHOSPHORYLA-
TION DURING AZO-DYE CARCINOGENESIS. *(E.)*
u, J.-F. (M.D. Anderson Hosp. Tumor Inst., Houston,
as), C. Craddock, S. Getz and L. S. Hnilica.
S Letters 33(2):247-250, 1973.

activity of protein phosphokinases in the cyto-
sm and chromatin fractions of liver homogenates
m male Fisher rats fed a diet containing 0.06%
-dimethyl-*p*-(*m*-tolylazo)alanine (3'MDAB) was
died. An observed gradual increase in the capa-
y of purified liver chromatin to serve as tem-
te for *in vitro* RNA synthesis which occurred over
6-day period in 3'MDAB-fed rats was accompanied
a similar increase in chromatin-bound and cyto-
smic phosphokinase activity. Polyacrylamide gel
ctrophoretic analysis of the phosphorylated pro-
ns from the livers of treated rats showed that
substrates for the increased enzyme activity
e primarily the nonhistone chromatin proteins.
DAB had no effect on the absolute and relative
ntities of the different protein moieties.

6 CHROMOSOME DAMAGE INDUCED BY CHEMICALS.
(E.) Bishun, N. P. (Res. Dept., Marie
ie Mem. Fdn., Surrey, England), D. C. Williams,
Mills, N. Lloyd, R. W. Raven and D. V. Parke.
m Biol Interact 6(6):375-392, 1973.

role of drugs, food additives, pesticides and
er environmental chemicals in causing chromosomal
age is reviewed. Mutation-induced susceptibility
disease, once largely self-eradicating, is now
ng conserved and even propagated. In addition,
erous synthetic chemicals have been added to our
ernal and external environments, the cytogenetic
perties of some of which have been reviewed, and
which man's metabolism has not necessarily been
ditioned and adapted. These chemicals may have
potential to augment the number of mutations of
h succeeding generation. Chromosomal studies and
luation of mutagenic potential of all new and
ablished drugs, food additives and environmental
micals are advocated as integral aspects of the
rent practices of safety evaluation of these
erials. The same stringent principles should be
lied to the numerous chemicals met with as sol-
ts and intermediates in industrial processes.
sons exposed to high risk, such as those working
chemical industries, or patients on continuous
motherapy, should be offered routine chromosomal
itoring, and the appearance of adverse effects
abnormalities noted and correlated.

7 THE MECHANISM OF ACTION OF AFLATOXIN B_1 ON
PROTEIN SYNTHESIS; OBSERVATIONS ON MALIG-
T, VIRAL TRANSFORMED AND UNTRANSFORMED CELLS IN
TURES. *(E.)* Garvican, L. (U. Coll. Hosp. Med.
, London, England), F. Cajone and K. R. Rees.
n Biol Interact 7(1):39-50, 1973.

mechanism of action of aflatoxin B_1 in inhibiting
tein synthesis was investigated in detail in
ican green monkey kidney CV-1 cell cultures. Stud-
on incorporation of ^{3}H-uridine and ^{14}C-protein
hydrolysate in RNA and protein, resp., of several different malignant, virus-transformed and untransformed lines of aflatoxin-treated cells indicated that inhibition of RNA synthesis was a prerequisite for inhibition of protein synthesis. Analysis of polysome profiles of CV-1 cells by sucrose density centrifugation showed that aflatoxin B_1 treatment induced polysome disaggregation, the extent of which correlated with the degree of inhibition of protein synthesis. The monosomes released by aflatoxin were, however, still functional in a cell-free protein synthesizing system using a poly U template, indicating that the decreased protein synthesis was not due to ribonuclease activity. Comparison of the temporal effect of aflatoxin and actinomycin D on inhibition of RNA and protein synthesis in CV-1 cells led to the conclusion that polysome disaggregation was not secondary to inhibition of mRNA synthesis. Electron micrographs of aflatoxin-treated cells showed no effect on rough endoplasmic reticulum or free polysomes, which were abundant. The presence of adequate polysomes *in vivo*, coupled with their loss after isolation, suggested that aflatoxin was functioning by producing polysome instability, possibly by direct action.

1848 ALTERATIONS IN EPIDERMAL GROWTH KINETICS INDUCED BY COAL TAR OINTMENT AND METHOTREXATE. *(E.)* Elgjo, K. (Inst. Path., U. Oslo, Norway) and T. E. Larsen. *J Invest Dermatol* 61(1): 22-24, 1973.

The cell cycle kinetics of hyperplasic epidermis induced in hairless mice by daily application for 4 wk of Goeckermann ointment, a coal tar-containing product, were studied. At the end of the treatment period half the animals received an i.p. injection of Colcemid 4 hr before sacrifice. Mitotic rate and duration of epidermal cells were then determined. The results showed that the coal tar-induced hyperplastic epidermoid cells had a low mitotic rate and a prolonged mitotic duration compared with those of normal mouse epidermal cells. Once weekly i.p. injections of methotrexate had no effect on these parameters, including the extent of hyperplasia, in treated animals. Epidermal cells from animals treated with vaseline containing 2% sulfur had increased mitotic rates and normal mitotic durations compared with untreated cells. These results indicate that ointments containing coal tar products and sulfur should be avoided.

1849 AGE- AND SEX-ASSOCIATED DIETHYLNITROSAMINE DEALKYLATION ACTIVITY OF THE MOUSE LIVER AND HEPATOCARCINOGENESIS. *(E.)* Rao, K. V. N. (Pritzker Sch. Med., U. Chicago, Ill.) and S. D. Vesselinovitch. *Cancer Res* 33(7):1625-1627, 1973.

Liver homogenates from 1-, 7-, 15-, 28-, 42-, and 70-day-old male and female (C57BL x C3H)F_1 mice were assayed by a chemical method for their ability to dealkylate diethylnitrosamine (DENA, 100 or 200 nmoles). Enzyme activity, which was present in newborns, increased in both sexes equally to a peak level between the 7th and 15th day of age. Thereafter, activity declined faster in females than in males, resulting in a significant ($p < 0.01$) difference after day 28 and reaching a plateau by day 42. A carcinogenicity bioassay showed a significantly higher incidence of liver tumors in mice treated i.p. at day 15 with 10 μg/g body wt DENA than in those treated at 6 wk of age. The sex difference in tumor incidence observed at wk 56 disappeared by wk 66 in animals treated with DENA as infants, indicating the same degree of carcinogenesis for both sexes and a slower rate of neoplastic expression for females.

1850 INCREASE OF SOLUBLE RNA METHYLASE ACTIVITIES BY CHEMICAL CARCINOGENS.

(E.) Hancock, R. L. (Fac. Med. U. Calgary, Canada) and P. I. Forroster. *Cancer Res.* 33(7):1747-1753, 1973.

High levels of the hepatocarcinogen ethionine (1% of the DL isomer mixture) in the rat diet increased soluble RNA methylase activity of both rat and hamster liver cells. Enzyme increases were detected after only 4 days of ethionine feeding. Thus far, ethionine has not been shown to be a hepatocarcinogen in hamsters. This increase in RNA methylase activity in hamsters, therefore shows that: 1) the increase in activity is not directly related to carcinogenesis; 2) the hepatomas have not been induced because of incorrect ethionine feeding regime; and 3) ethionine is not required for induction *per se* but is a "core-requirement" of the carcinogenic mechanism. Dimethylaminoazobenzene, another hepatocarcinogen, also increased soluble RNA methylase activity. Noncarcinogenic analogs of ethionine, norleucine and L-methionine, did not cause this increase in activity. Liver enzyme preparations form ethionine-treated rats methylated yeast phenylalanine transfer RNA more extensively than did enzymes from normal liver. The only nucleotides methylated were uridylic acid and cytidylic acid with the latter being six times more methylated. Ethionine treatment did not alter the amounts of soluble RNA methylase inhibitors. The same ethionine enhancement of soluble RNA methylase activity could be demonstrated in metabolizing yeast cells.

1851 HYPERPLASTIC CHANGES OF THE EPITHELIUM OF KIDNEY ORGAN CULTURES AFFECTED BY THE TRANSPLACENTAL EFFECT OF DIMETHYLNITROSAMINE.

(Rus.) Sorokina, Yu. D. (Inst. Exp. Clin. Oncol., Moscow, USSR) and S. P. Bogovskii. *Vopr Oncol* 18(10):52-56, 1972.

Three groups of pregnant BALB/c mice each received dimethylnitrosamine (0.1 mg x 3, 0.1 mg x 2 + 0.2 mg x 2, or 0.3 mg x 2). The kidneys of 19-21-day-old embryos of these mice were used as organ cultures. Viability of embryonic kidney explants depended upon the dose of the carcinogen and the schedule of its administration. A small dose of 0.3 mg dimethylnitrosamine increased the survival rate of organ cultures (80.4%), in comparison to organ culutres exposed to 0.3 mg x 2 (34.1%) and 0.1 mg x 2 + 0.2 mg x 2 (70.7%). Diffuse and focal hyperplasia was seen in the epithelium of the tubules and urinary passages, indicating precancerous changes had occurred. Cystoadenoma was observed in four explants (7%) exposed to 0.6 mg dimethylnitrosamine.

1852 DEPRESSION OF ENZYMATIC ESTERIFICATION OF N-HYDROXY-AAF IN THE LIVER DURING AMINOAZO DYE CARCINOGENESIS IN THE RAT.

(E.) Blunck, J. M. (Dept. Path. U. Melbourne, Australia) and B. J. Lees. *Res Comm Chem Pathol Pharmacol* 5(2): 421-429, 1973.

An *in vitro* radioassay measuring the esterification of N-hydroxy-2-acetylaminofluorene (N-hydroxy-AAF) by 102,000 x *g* liver homogenates was used to monitor the ability of rat liver to form sulfate esters 3, 10 and 20 days after initiation of a diet containing the hepatocarcinogen 3'methyl-4-dimethylaminoazobenzene (3'MeDAB). Although not apparent in rats fed carcinogen for three days, administration of 0.06% 3'MeDAB or 0.06% 3'MeDAB plus 2% chloramphenicol (CAP) in the diet for 10 or 20 days produced a 50-70% decrease in total hepatic enzymic esterification of N-hydroxy-AAF compared to controls. These results, taken together with previously shown similarities in the *in vivo* metabolites of azo dyes and AAF derivatives and the overlapping specificities of some sulfotransferases, suggest that esterification may be operative in the metabolic activation of these carcinogens *in vivo*.

1853 EFFECT OF THE URINE AND CALCULUS FORMATION ON THE INCIDENCE OF BLADDER TUMORS IN RATS IMPLANTED WITH PARAFFIN WAX PELLETS.

(E.) Chapman, W. H. (Sch. Med., U. Washington, Seattle), D. Kirchheim and J. W. McRoberts. *Cancer Res* 33(6):1225-1229, 1973.

Experiments have been performed to determine whether urine has a role in bladder carcinogenesis beyond that of being simply a carrier for known carcinogens. A double pouch of bladder was formed by division of the bladder of inbred weanling, male Fischer 344 rats and implantation of paraffin pellets into each pouch. The upper pouch could be in contact with or isolated from the urinary stream, while the lower pouch was always in contact with the urine. No tumors formed in 99 isolated upper pouches while 49 formed in 119 communicating upper pouches in contact with the urine. The presence of 20% 3-hydroxyanthranilic acid in the pellets made no change in the incidence of tumors. When the urine was in contact with the pouch, stone formation was evident in one-half of the pouches and, in those pouches with stone formation, the incidence of tumors approximately doubled. The technique allows comparison of the effect of the pellet in the presence or absence of urine. The data suggest that there are at least two factors necessary for the formation of bladder tumors in this experimental design: 1) an unidentified substance and/or factor related to the urine, which could be a carcinogen or even a physical factor such as the repetitive distension and contraction of the bladder; 2) the presence of a foreign body.

INHIBITION OF AFLATOXIN BIOSYNTHESIS OF
DICHLORVOS. *(E.)* Hsieh, D. P. H. (Dept.
ental Toxicology, U. California, Davis).
od Chem 21(3):468-470, 1973.

os (dimethyl 2,2-dichlorovinyl phosphate)
d to possess a strong inhibitory effect on
n biosynthesis by *Aspergillus parasiticus*
17. At 10 ppm it inhibited 90% of aflatoxin
on in three types of liquid media, mini-
ral media (MM), MM supplemented with 0.1%
extract, and a yeast extract-sucrose
without affecting the fungal growth. When
vely synthesizing cultures were supplemented
ppm of dichlorvos and [1-^{14}C]acetate, a re-
ount of aflatoxin B_1 was produced which con-
lmost no label from the acetate, suggesting
hlorvos inhibits an early step in the secon-
abolic pathway for aflatoxin biosynthesis.
nts with other organophosphorus insecticides
hat dichlorvos was particularly inhibitive
iosynthesis of aflatoxins.

EVIDENCE FOR A GLUCURONIC ACID CONJUGATE
OF *N*-HYDROXY-4-AMINOBIPHENYL IN THE URINE
GIVEN 4-AMINOBIPHENYL. *(E.)* Radomski, J. L.
i Sch. Med., Fla.), A. A. Rey and E. Brill.
es 33(6):1284-1289, 1973.

of the presence of substantial quantities
curonic acid conjugate of *N*-hydroxy-4-
henyl in the urine of dogs given 4-amino-
was obtained. The conjugate is very acid
readily liberating free *N*-hydroxy-4-amino-
at lowered pH and in the presence of β-
idase. It was responsible for approximately
he tritium-labeled excretory products of
iphenyl in the urine. It is apparently
ce of the *N*-hydroxy compound previously
in dog and monkey urine and is believed
volved in the induction of bladder cancer
minobiphenyl. That the liberated *N*-hydroxy
is spontaneously converted to the nitroso
ve in aqueous solutions suggests that the
ubstance may be more important to the
enic process than the *N*-hydroxy compound.
not necessarily true, however, because it
hat the liberation of the *N*-hydroxy com-
om the conjugate by β-glucuronidase in
oximity to the bladder mucosa is the signi-
vent.

BINDING PATTERNS OF 3-METHYLCHOLANTHRENE
TO REPLICATING AND NONREPLICATING DNA OF
ASTIC NODULE OF MOUSE MAMMARY GLAND. *(E.)*
, M. R. (Inst. Cellular Res., U. Nebraska,
, D. L. Kinder and J. E. Wagner. *Cancer*
):862-866, 1973.

ding of 3-methylcholanthrene (MC) to DNA and
tion to DNA synthesis in the mammary tumor
ee hyperplastic alveolar nodule outgrowth
/c mouse mammary gland were determined. As
by tritium radioactivity, the carcinogen
nd to bind to nodule DNA at 12 and 24 hr after
intragastric feeding of 500 μg MC-^{3}H to
mice carrying the outgrowth. The binding of MC to DNA *in vivo* was slightly lower when DNA synthesis in the nodule cells was more active at 4 wk compared to the period of reduced DNA synthesis at 8 weeks after fat-pad transplantation. In a separate experiment both 4- and 8-wk-old outgrowths were incubated *in vitro* and DNA was double labeled with MC-^{3}H and thymidine-2-^{14}C. During the 6-hr incubation in a shaking water bath at 37°, both MC-^{3}H and thymidine-2-^{14}C radioactivity was present in nodule DNA, but no direct relation to MC-^{3}H and thymidine-2-^{14}C radioactivity was present between the 4- and 8-wk-old nodule outgrowth cells *in vitro*. When the nodule DNA, double labeled with 5-bromodeoxyuridine and MC-^{3}H, was centrifuged to equilibrium in CsCl, the radioactive carcinogen was almost equally distributed between the preexisting and the newly synthesized, 5-bromodeoxyuridine-labeled heavier fractions of the nodule DNA. The results indicate that binding of MC to DNA of the preneoplastic nodule outgrowth of mouse mammary gland is not influenced by or dependent upon the rate of DNA synthesis in the tissue. Hence increased susceptibility of the DNA-replicating cell to tumorigenic action of MC may not be related to augmented binding alone.

1857 A SCREEN FOR PESTICIDE TOXICITY TO PROTEIN AND RNA SYNTHESIS IN HeLa CELLS. *(E.)*
Myhr, B. C. (Battelle, Columbus Lab., Ohio). *J Agr Food Chem* 21(3):362-367, 1973.

Suspension cultures of HeLa cells were used in a screen for toxicity based upon an initial biochemical response rather than the usual measurements of growth inhibition. Cells were exposed for 30 min to 350 μg/ml dosages of 30 different pesticides, and the effect on [^{3}H]uridine and ^{14}C-labeled amino acids incorporation into RNA and protein were determined. Dose-response curves for DDT, aldrin, carbaryl, and parathion yielded ID_{50} values that showed this method was as sensitive as 48-hr growth measurements. One-half of the compounds studied had no effect on one or both incorporation activities. Eight pesticides selectively inhibited amino acid incorporation, while only one, chlorpropham, was a selective inhibitor or uridine incorporation. Propham, however, inhibited both processes. The actions of the organophosphorus compounds were as varied as the side chains, but all of the chlorinated hydrocarbons except dieldrin strongly inhibited amino acid incorporation. The carbamates and thiocarbamates tested appeared to be generally more inhibitory than the organophosphorus group, with carbaryl, EPTC, and pebulate being especially inhibitory to amino acid incorporation. Uncouplers of oxidative phosphorylation were highly inhibitory.

1858 CHANGES IN THE PASSAGE OF 9,10-DIMETHYLBENZ(a)ANTHRACENE (DMBA) THROUGH THE PLACENTA IN MICE. *(Rus.)* Shendrikova, I. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR), M. N. Ivanov-Golitsyn, V. N. Anisimov and A. Ia. Likhachev. *Vopr Onkol* 19(5):75-79, 1973.

An emulsion of 9,10-dimethylbenz(a)anthracene (DMBA; 40 mg/kg i.v.) was injected into noninbred albino mice on day 17 to 19 of gestation. The mothers were killed 5 min to 6 hr after injection, and fetuses and placentas were removed. At 5 min after DMBA injection the concentration of DMBA in maternal liver was about 100 times and that in the placenta was 10 times higher than that in fetal tissue. During the next hour the DMBA concentration in fetal tissue remained almost unchanged, while that in the maternal liver and placenta decreased to 10% and 12%, resp. of the initial concentrations in these organs. DMBA concentrations were the same in maternal and fetal tissues 4 hr after injection. DMBA concentrations in the maternal liver and placenta were 0.4 and 0.8%, resp. of the initial values, while those in fetal tissue had only decreased to 40% of initial values. At 1 hr after injection of a smaller dose of DMBA (20 mg/kg i.v.), the DMBA concentration in fetal tissue was twice that found after 5 min; a significant decrease in DMBA occurred in fetal tissue during the second hour. The DMBA concentration in fetuses 6 hr after injection was only 0.9% of the concentration in fetuses 1 hr after injection. In fetuses removed 1 hr after injection and kept alive in an incubator for 5 more hr, the DMBA concentration was 34% of that at 1 hr. DMBA excreted by these fetuses was a very small percentage of that found in fetal tissue. Thus, fetuses are capable of metabolizing DMBA, but the rate at which the carcinogen is metabolized is significantly lower than in adult mice. The very low DMBA concentrations found in fetal tissue 6 hr after injection could be due either to the passage of enzymes which metabolize DMBA through the placental barrier to the fetus or to metabolism of the carcinogen by the mother after it had been transported through the placental barrier from the fetus. These findings support the hypothesis that tumors which develop in the offspring of mice given DMBA during pregnancy are caused by passage of the carcinogen through the placenta.

1859 K-REGION EPOXIDES OF POLYCYCLIC HYDROCARBONS: FORMATION AND FURTHER METABOLISM OF BENZ[a]ANTHRACENE 5,6-OXIDE BY HUMAN LUNG PREPARATIONS.

(E.) Grover, P. L. (Royal Cancer Hosp., London, England), A. Hewer and P. Sims. *FEBS Lett* 34(1):63-68, 1973.

Human lung preparations were used to demonstrate that: a) an epoxide, which had been identified as the K-region derivative, benz[a]anthracene 5,6-oxide, is formed as a microsomal metabolite of benz[a]anthracene and b) this epoxide can be further metabolized to the corresponding dihydrodiol, *trans*-5,6-dihydro-5,6-dihydroxybenz[a]anthracene, and to the glutathione conjugate, S-(5,6-dihydro-6-hydroxybenz[a]anthracene-5-yl)glutathione by enzymes present in these preparations. Collectively, the results indicate that all three enzymes known to be involved in the formation and further metabolism of polycyclic hydrocarbon epoxides are present in human lung. The mixed function oxidase, which catalyzes the initial oxidation of the aromatic double bonds to epoxide intermediates, may well be involved in the metabolic activation of carcinogenic polycyclic hydrocarbons.

1860 STILBESTROL THERAPY IN PREGNANCY. RELATI SHIP TO VAGINAL NEOPLASIA IN OFFSPRING.

(E.) Nissen, E. D. (Dept. Gynecol. Obstet., U. Cal fornia, Irvine) and A. I. Goldstein. *J Gynecol Obs* 11(4):138-142, 1973.

A case of vaginal mesonephroma with adenosis is reported in a 16-year-old girl whose mother had taken high doses (e.g., 75.0 mg) of diethylstilbestrol (DES) during the first trimester of her pregnancy. This represents the 13th reported case of vaginal carcinoma in the offspring of a mother treated with synthetic estrogens during pregnancy. Biopsy of a lesion in the vaginal wall of the patient's 18-year old sister provided a diagnosis of benign papilloma tosis. In the case of this patient, the mother had taken DES starting at the 8th wk of pregnancy where as in the case of her 16-year-old sister, the mothe had taken DES starting at the 6th wk. It is suggested that this difference of 2 wk may account for the absence of frank malignancy in the older siblin

1861 AFLATOXIN HEPATIC CARCINOGENESIS: AN EXPERIMENTAL COMPARATIVE STUDY. *(E.)*

Sen, S. (Armed Forces Med. Coll., Poona, India). *Indian J Med Res* 60(9):1261-1266, 1972.

Hepatocarcinogenesis was studied in male albino Nor wegian rats fed a diet containing toxic groundnut meal with 5-10 ppm aflatoxin B_1. Of 17 toxin-fed mice which survived for over 12 months, all were au topsied because of gross abdominal masses. Twelve of these had massive liver tumors, and almost all died between 16-24 months after the start of the experiment. No control rats developed liver tumors during this time period. Grossly, the tumor-bearin livers were enlarged and contained multiple, large nodules with central necrosis. Five of the tumors had metastasized to distant organs including lung, kidney, testes and mesenteric lymph nodes. None of the animals had gross cirrhotic liver changes and all tumors arose in noncirrhotic areas. Histologically, the tumor cell resembled hepatic cord cells although the smaller tumors were trabecular in appearance. The cell types varied from well-differentiated to anaplastic. In addition to the ability to metastasize, these tumors also grew invasively into surrounding structures.

1862 ANTIOXIDANTS AND CARCINOGENESIS: BUTYLAT HYDROXYTOLUENE, BUT NOT DIPHENYL-*P*-PHENYL ENEDIAMINE, INHIBITS CANCER INDUCTION BY *N*-2-FLUORE ACETAMIDE AND BY *N*-HYDROXY-*N*-2-FLUORENYLACETAMIDE I RATS.

(E.) Ulland, B. M. (Bionetics Res. Lab., Inc., Bethesda, Md.), J. H. Weisburger, R. S. Yamamoto and E. K. Weisburger. *Food Cosmet Toxicol* 11:199-207, 1973.

The effect of butylated hydroxytoluene (BHT) and diphenyl-*p*-phenylenediamine (DPPD) on cancer induction by *N*-2-fluorenylacetamide (FAA) and its *N*-hydroxy derivative (N-OH-FAA) has been studied in two strains of rat. Groups of Charles River rats were fed carcinogen and antioxidant in a molar rati of 1:30 (223 ppm FAA or 239 ppm N-OH-FAA (1mM) and 7850 ppm DPPD or 6600 ppm BHT) in Wayne Chow for

wk (males) or 32 wk (females). Male and female
s then continued on control diet for another
wk. With FAA alone 70% of male rats had hepatoma
20% of females had mammary adenocarcinoma;
n N-OH-FAA, 60% of males had hepatoma and 70% of
ales had mammary adenocarcinoma. The simultaneous
inistration of DPPD failed to alter cancer in-
tion by these agents in male or female rats.
ever, BHT reduced the incidence of hepatoma in
es to 20% when the carcinogen was FAA. With N-
FAA, BHT lowered the incidence of liver tumors
15% and of mammary cancer in females to 35%.
ilar data on inhibition by BHT were obtained
ng different levels of carcinogens in rats of
Fischer strain. Equimolar levels of sulphate
led to increase liver-tumor incidence in animals
en N-OH-FAA and BHT. Also, BHT failed to depress
excretion of free urinary inorganic sulphate.
er- and esophageal-tumor induction with 51 ppm
thylnitrosamine in drinking water for 24 wk was
affected by BHT or DPPD, nor was tumor induction
propane sultone. It is improbable that the anti-
dant properties of BHT played a role in its in-
ition of tumor induction. If these properties
e involved, the equally effective antioxidant,
D, should have acted likewise, but it had no
ect on cancer incidence.

3 EFFECT OF AMINOACETONITRILE ON THE FINE
STRUCTURE AND CYTOCHEMISTRY OF HEPATIC
LS IN RATS FED TWO CARCINOGENIC *N*-NITROSODIALY-
INES. *(E.)* Hadjiolov, D. (German Cancer Res.
., Heidelberg, W. Germany) and D. Markov. *J*
Cancer Inst 50(4):979-988, 1973.

ultrastructural and histochemical changes in-
ed in male Wistar rat liver cells by oral admini-
ation of dimethylnitrosamine (DMNA) and diethyl-
rosamine (DENA) have been studied. Both di-
ylnitrosamines produced progressive disorgani-
ion of the granular endoplasmic reticulum and
ertrophy of the agranular reticulum, accompanied
the disappearance of glycogen. DMNA and DENA
o caused a loss of centrolobular glucose-6-
sphatase activity and markedly decreased DPNH-
TPNH-tetrazolium reductase activities. The
idence of these morphologic alterations was
uced by the subsequent s.c. administration of
noacetonitrile. Persistent cytoplasmic ab-
malities were a slight dilation of the membran-
profiles of the granular endoplasmic reticulum
a moderate hypertrophy of the agranular reti-
um. The glycogen content was almost normal.
trolobular glucose-6-phosphatase activity also
ghtly decreased. These findings suggested that
noacetonitrile has a direct stabilizing effect
cytoplasmic membrane systems and ribosomes
the early stage of nonspecific liver damage
sed by 2 dialkylnitrosamines.

4 EFFECTS OF ONE-YEAR ADMINISTRATION OF
ETHYLENETHIOUREA UPON THE THYROID OF THE
. *(E.)* Graham, S. L. (Dept. Hlth. Education,
fare, Washington, D.C.), W. H. Hansen, K. J.
is and C. H. Perry. *J Agr Food Chem* 21(3):324-
, 1973.

Five groups of 68 male and 68 female Charles River rats, approximately 5-wk-old, were fed ethylenethiourea (ETU) for 2, 6, or 12 months at levels of 5, 25, 125, 250, or 500 ppm in the diet. Control groups were fed the basic laboratory chow. Body weight, thyroid and other organ weights, thyroidal ^{131}I uptake, hematology, and histology were the criteria studied. Significant decreases in body weight and increases in thyroid weight were seen at the 125-, 250-, and 500-ppm levels. Uptake of ^{131}I was significantly decreased in male rats after 12 months at 500 ppm but was increased in females. After 12 months, microscopic examination of the thyroids revealed the development of nodular hyperplasia at dose levels of 125 ppm and higher; carcinomas were found at dose levels of 250 and 500 ppm. At the 500-ppm dose level, the pituitary was moderately hyperplastic. Sharp elevations in the weight of the thyroid appeared to be more closely associated with the development of malignant tumors which tended to be large rather than with tumors of a benign histological pattern. The development of thyroid carcinomas indicates that, under the conditions of this study, ETU is a carcinogen.

1865 EFFECT OF THE ANTIOXIDANT BUTYLATED HYDROXYTOLUENE (BHT) ON THE METABOLISM OF THE CARCINOGENS *N*-2-FLUORENYLACETAMIDE AND *N*-HYDROXY-*N*-2-FLUORENYLACETAMIDE. *(E.)* Grantham, P. H. (Natl. Cancer Inst., Bethesda, Md.), J. H. Weisburger and E. K. Weisburger. *Food Cosmet Toxicol* 11:209-217, 1973.

The effect of the antioxidant butylated hydroxytoluene (BHT) on the metabolism of *N*-2-fluorenylacetamide (FAA) and *N*-hydroxy-*N*-2-fluorenylacetamide (N-OH-FAA) was studied in male and female rats. Twelve male and 12 female 6-wk-old Charles River CD rats were fed 6600 ppm BHT, mixed in the diet, for 4 wk. Twelve male and 12 female rats were maintained as controls on Wayne laboratory meal. After the 4-wk period, six male and six female rats pre-fed BHT, as well as six of each sex from the controls, were given a single i.p. injection of labelled FAA in a dose of 22.3 mg/kg (specific activity 0.48 mc/m-mole). Another group of six male and six female rats pre-fed BHT, and also six controls of each sex were given a single i.p. dose (23.9 mg/kg) of labelled N-OH-FAA (specific activity 0.50 mc/m-mole). Compared with control animals, both male and female rats pre-fed BHT exhibited considerable liver enlargement. More total radioactivity was present in the livers of male than of female rats. It was also higher in rats given N-OH-FAA than in those given FAA. In all cases however, pre-feeding with BHT reduced the total amount of radioactivity found in the livers. Likewise, radioactivity in the blood and blood plasma collected from the rats exhibited the same relative effects except that the levels were almost identical in males and in females. Significantly, BHT prefeeding decreased circulating carcinogens. DNA isolated and purified from male rats had a higher level of radioactivity in animals injected with N-OH-FAA than in those injected with FAA. In both cases, BHT reduced the amount of carcinogen bound firmly to DNA. Both male and female rats excreted similar amounts of radioactivity, representing metabolites of the injected FAA or N-OH-FAA. In rats of both sexes, BHT increased very considerably the amount of radioactivity excreted from both carcinogens. With respect to urinary excretion of free metabolites, male rats had similar levels of glucuronides and sulphates whether injected with FAA or with N-OH-FAA. However pretreatment with BHT almost doubled the percentage of a dose of FAA excreted as a glucuronide, and almost tripled the proportion with N-OH-FAA. This higher level of excretion was accounted for by glucuronic acid conjugates which tie up the N-OH-FAA reaching the liver. Sulphuric acid esters were decreased. It is concluded that BHT increases the detoxification metabolites of FAA and N-OH-FAA, and thus lowers the amount of a given dose available for activation reactions.

1866 FORMATION OF NITROSAMINES FROM LECITHIN AND NITRITE.

(Ger.) Möhler K. (German Inst. Food Chem., Munich, Germany) and E. Hallermayer. *Z Lebensm Unters Forsch* 151(1):52-53, 1973.

Volatile carcinogenic N-nitrosamines from lecithin and nitrite are formed by boiling these substances in buffer solutions in the range of pH 3.5 - 7.0. At pH 3.5 1 kg of lecithin and 0.5 kg nitrite after 2 hr boiling release 2 - 4 mg of dimethylnitrosamine, 0.3 - 1.0 mg diethylnitrosamine and 0.1 - 1 mg dipropylnitrosamine. Smaller amounts of nitrosamines are obtained at higher pH. On the basis of these results the authors assume that lecithin and nitrite present in food are the substrates for the formation of carcinogenic nitrosamines.

1867 EFFECTS OF CARCINOGENIC HYDROCARBONS ON LIPIDS OF PROTOPLASMIC MEMBRANES AND MITOCHONDRIA OF *SACCHAROMYCES CEREVISIAE*.

(Fr.) Baraud, J. (Biochem Lab., Univ. Bordeaux, Talence, France), A. Maurice, C. Napias and J. Velours. *Biochim Biphys Acta* 296(3):481-492, 1973.

Incubation of *Saccharomyces cerevisiae* with benzo(a)pyrene, dibenz(a,h)anthracene or dibenz(a,c) anthracene (0.15-12.5 mg/liter culture) caused changes in the nature and content of lipids, especially phospholipids, in whole cells, protoplasmic membranes and mitochondria of the yeast. A large reduction in total lipids was observed; phospholipids were decreased by 90% while neutral lipids were only slightly affected. Titration of phosphorus showed that all fractions of phospholipids (phosphatidylglycerol, phosphatidylethanolamine, phosphatidylcholine, phosphatidylinositol) were greatly reduced. Changes induced by strongly carcinogenic benzo(a)pyrene and dibenz(a,h)anthracene were more pronounced than those caused by the weakly carcinogenic dibenz(a,c)anthracene. Experiments with ^{3}H-benzo(a)pyrene showed that the carcinogen penetrated into the cell where it was partially metabolized; some of the carcinogen and its degradation products were found in the membrane and mitochondria. The level of phospholipids increased to about half the normal values when the culture, which had previously been incubated with benzo(a)pyrene, was subsequently grown in a carcinogen-free medium. Comparative experiments with *Escherichia coli* showed that benzo(a)pyrene had no effect on the phospholipid content of this organism.

1868 THE INDUCTION OF MULTIPLE LARGE BOWEL NEOPLASMS IN MICE.

(E.) Evans, J. T. (Roswell Park Mem. Inst., Buffalo, N.Y.), G. Lutman and A. Mittelman. *J Med (Basel)* 3(4-5):212-215, 1972.

Multiple polypoid neoplasms of the large bowel were induced in inbred Ha/ICR Swiss mice following repeated weekly s.c. injections of 1,2-dimethylhydrazine (15 mg/kg). This strain does not develop spontaneous intestinal neoplasms. No small bowel lesions, lymphomas, leukemias or sarcomas were found at sacrifice in any of the animals. The larg bowel lesions, which began to appear grossly after 19 wk, were sessile or pedunculated and originated from the mucosal epithelium. Neither metastatic disease nor blood vessel invasion was observed. Attempts to induce similar neoplasms in C57Bl mice were unsuccessful. The ability to induce large bowel neoplasms in Ha/ICR Swiss mice may provide a useful model for the study of human colonic carcinoma.

1869 DIFFERENT CYTOTOXIC EFFECTS OF METHYL- AN ETHYLNITROSOUREA ON DEVELOPING RAT TISSUE AND THE INFLUENCE OF CYCLOHEXIMIDE ON CELL DEATH.

(E.) Bosch, D. A. (Dept. Path., U. Groningen, Holland), A. Ipema and E. J. Ebels. *Z Krebsforsch* 79(4):255-266, 1973.

The effect of methyl- and ethylnitrosourea on the duration of mitotic inhibition, the fate of the population of lethally damaged cells, and the mechanism of cell death was examined. Eight-day-old rats were exposed to 100 mg/kg body weight of eithe methylnitrosourea (MNU) or ethylnitrosourea (ENU). In the *extraneural* tissues mitotic inhibition lasts longer after MNU than after ENU. In the case of ENU, the number of mitotic cells rapidly increases once they reappear, whereas in the MNU-treated material reappearing mitotic figures are present in small numbers only at all intervals studied. Lethally damaged cells disappear slowly, but at the sam rate for both MNU and ENU. The *neural* tissues show a much longer duration of mitotic inhibition. But as in the extraneural tissues after their reappearance in ENU-treated animals mitotic figures rapidly increase in number, whereas after MNU only a very limited number of cells in mitosis is found at all intervals. Lethally damaged cells in the neural tissues disappear much later than in the extraneural ones, but again at the same rate for both MNU and ENU. Among the mitotic figures in all tissues examined abnormal ones are frequently noticed. Delay of cellular death was observed in all tissues examined when, after exposure to MNU or ENU, protei synthesis was blocked by cycloheximide, indicating that cell death is an active process depending on *de novo* synthesis of protein(s). In comparison wit

milar results for well-known alkylating agents in
e literature, the findings add further evidence
at the cytotoxic action of both MNU and ENU is
used by alkylation of cellular macromolecules.
e marked differences for mitotic inhibition be-
een MNU and ENU may be significant with respect to
e well-known difference in LD-50 of these compounds.

70 ULTRASTRUCTURAL EFFECTS OF N-METHYL-N-
NITROSOUREA ON THE TRACHEOBRONCHIAL
ITHELIUM OF THE SYRIAN GOLDEN HAMSTER. *(E.)*
rris, C. C. (Natl. Cancer Inst., Bethesda, Md.),
G. Kaufman, M. B. Sporn, J. M. Smith, F. Jackson
d U. Saffiotti. *Int J Cancer* 12(1):259-269, 1973.

e genesis of squamous-cell carcinoma of the lung
s investigated in morphological and biochemical
rms. Male Syrian golden hamsters initially
ighing 100-120 g were used in the experiments.
n intratracheal instillations of N-methyl-N-
trosourea (NMU) caused squamous metaplastic and
oplastic changes in the tracheobronchial epithe-
um. Abnormal squamous metaplastic cells contained
larged nuclei deeply indented by cytoplasmic
vaginations, pleomorphic nucleoli, filamentous
anules and many cytoplasmic fibrils. Prior to
e appearance of tumors, autoradiograms revealed
lls preparing for division, first in basal and
en in all layers of the abnormal squamous meta-
astic epithelium. Defects in the basement lamina
re found in squamous metaplastic lesions. The
trastructural changes in the tracheobronchial
ithelium were similar to those described in
msters exposed to benzo[a]pyrene-ferric oxide as
ll as to those described in smoking dogs and in
man bronchogenic carcinoma. The squamous meta-
astic changes induced by NMU were clearly distin-
ishable from squamous metaplasia found in vitamin-
deficiency.

71 WATER SOLUBLE PROTEINS FROM RAT BRAIN AND
FROM EXPERIMENTAL TUMORS OF RAT NERVOUS
STEM. *(E.)* Stavrou, D. (Fac. Vet. Med., U.
nich, Germany) and K. G. Haglid. *Z Krebsforsch*
(4):249-254, 1973.

order to determine whether the model of experi-
ntal brain tumors in animals displays any similar-
ies to human brain and brain tumors, soluble pro-
ins of rat brain and experimental methylnitrosourea-
duced tumors of the rat brain were subjected to
ectrophoretic and immunochemical analyses. Disc
ectrophoretic separation of soluble proteins of
t brain and experimental tumors of the rat nervous
stem were performed in 10% polyacrylamide gels.
judged on the basis of relative mobilities, dif-
rences in the prealbumin protein band pattern in
ls of rat brain and different tumors of the rat
rvous system induced by methylnitrosourea (MNU)
re more of a quantitative nature. The front bands
ter electrophoresis in 7.5% polyacrylamide gels
ing a continuous buffer system of soluble proteins
rat brain, mixed gliomas, oligodendrogliomas,
trocytomas and neurinomas all contained antigen-
ally identical material to beef S_{100} protein in
reement with an earlier immunodiffusion study.
It was also shown in an earlier study, that the soluble proteins of normal human brain and different brain tumors of man expose a disc electrophoretic protein band pattern in the prealbumin area that differs from tissue to tissue more in a quantitative way than in the complete absence or presence of new protein components. This may be an indication that spontaneous human brain tumors and experimental rat brain tumors as induced in this study are parallel systems that could be compared on a biochemical basis.

1872 A MODEL OF CARCINOGENESIS. *(Ger.)* Popp, F. A. (Radiation Clin., U. Marburg, Germany). *Strahlentherapie* 144(2):208-215, 1972.

A resonance hypothesis of carcinogenesis is evolved from the difference in the electronic structure beteeen the carcinogen benzo(a)pyrene and the non-carcinogen benzo(e)pyrene, which explains the different behavior of both substances. The false resonance phenomenon caused by benzo(a)pyrene is the first step in carcinogenesis through destroying the H-bridges between DNA- and RNA-bases. This effect can lead through mutation to cancer. The hypothesis also renders possible an interpretation of X-ray cancer and the theory of a latent virus.

1873 CIGARETTE SMOKE AS A CARCINOGEN. *(E.)* Weiss, W. (Philadelphia Gen. Hosp., Pa.). *Am Rev Respir Dis* 108(2):364-366, 1973.

A total of 486 men who smoked 0.5 pack of cigarettes per day or more, were born between 1890 and 1899, and developed lung cancer between 1956 and 1965, were studied with regard to the relationship between age at diagnosis and smoking habits in a preliminary attempt at developing a mathematical model of the relationship between cigarette smoking and carcinogenesis in humans. Age distribution shifted downward as the number of cigarettes smoked per day increased and as the age at which smoking began decreased. These observations suggest that cigarette smoke acts as a carcinogen in humans.

1874 EXPERIMENTAL BLADDER CARCINOGENESIS - USING CIGARETTE SMOKE CONDENSATE IN THE RAT BLADDER. *(E.)* Wagle, D. G. (Roswell Park Mem. Inst., Buffalo, N.Y.) and J. Lee. *J Surg Oncol* 5(3):197-203, 1973.

The incidence of transitional cell tumors following implantation of cigarette smoke condensate into "Buffalo rat" bladders was examined. On hundred and ten male and female rats were used in this study with an additional 40 serving as controls. The implantation technique consisted of creating an isolated free bladder graft filled with the test material. The test pellets were made from cigarette smoke condensate obtained under standard conditions on an automatic machine. The rats were sacrificed 20 months after implantation. Nine rats were unsuitable for further study, leaving 101 isolated bladder pouches for histopathologic evaluation. Of these

101 rats, 7% had concretions, 23% had squamous metaplasia, and 2% had malignant tumors. There was no relationship between the presence of a concretion and that of a malignant tumor. The malignant tumors were of transitional cell type; no distant metastases were observed. This treatment did not develop a significant number of bladder tumors in isolated rat bladder pouch through impregnation of cigarette smoke condensate. It therefore suggests that certain additional tumor promoting factors may be necessary for the induction of such bladder malignancy. Such factors may be under the influence of certain urinary enzymes.

1875 INTRATRACHEAL INSTILLATION METHOD FOR MOUSE LUNGS. *(E.)* Ho, W. (Inst. Chem. Biol., U. San Francisco, Calif.) and A. Furst. *Oncology* 27(5):385-393, 1973.

An intratracheal instillation technique was developed which permits the repeated administrations of precise volumes of particulate suspension into mouse lungs. Benzo(a)pyrene (BP) adsorbed to ferric oxide was used to test the feasibility of the method on Swiss albino, random-bred, female mice of 21 ± 1 gm body weight. When the animal, anesthetized with an i.o. injection of pentobarbital (60 mg/kg), became flaccid it was placed in a specially designed holder in such a way that its head, neck and thorax hung down vertically. A dissecting microscope equipped with fiber optics was used to focus and illuminate the buccal cavity. A 0.02 ml aliquot containing 0.3 mg each of BP and ferric oxide was drawn into a microsyringe fitted with a cannula which permits the delivery of the suspension near the region of the bronchial bifurcation. After practice, the method required 1-2 min to treat each mouse. The majority of the animals survived four weekly treatments. Histological and fresh tissue sections showed extensive penetration of both chemicals into the lung parenchyma; however, while BP was eliminated from the organ rapidly, the ferric oxide was retained for over 100 days. These dust particles were observed inside macrophages collected around terminal bronchioles and lymphoid tissues of the lungs. The advantage of this procedure is that the instilled material bypasses most of the filtering action of the respiratory tract, thus permitting a greater penetration of the material than otherwise possible.

1876 REPAIR SYNTHESIS AND SEDIMENTATION ANALYSIS OF DNA OF HUMAN CELLS EXPOSED TO DIMETHYLNITROSAMINE AND ACTIVATED DIMETHYLNITROSAMINE. *(E.)* Laishes, B. A. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada) and H. F. Stich. *Biochem Biophys Res Commun* 52(3):827-833, 1973.

The capacity of dimethylnitrosamine(DMN), and of DMN activated by a NADPH-fortified mouse liver microsomal preparation, to elicit DNA alterations in cultured human fibroblasts was examined. A maximum induction of DNA repair synthesis, estimated by unscheduled incorporation of tritiated thymidine, occurred following 60-minute incubation of the human cells with DMN activated by a NADPH-fortified mouse liver microsomal preparation. A low level of DNA repair activity followed exposure to DMN alone, or to DMN mixed with the microsomal preparation without NADPH or without O_2. The extent of DNA damage, estimated by velocity sedimentation of DNA through alkaline sucrose gradients, was maximum following treatment with DMN mixed with the NADPH-fortified microsomal preparation. These DNA changes seem to lead to single-strand breaks. The combined application of *in vitro* activation systems and estimation of DNA repair synthesis in cultured cells may be exploited in the detection of precarcinogens.

1877 CARCINOGEN-INDUCED CHROMOSOMAL BREAKAGE DECREASED BY ANTIOXIDANTS. *(E.)* Shamberger, R. J. (Cleveland Clin. Fdn., Ohio), F. F. Baughman, S. L. Kalchert, C. E. Willis and G. C. Hoffman. *Proc Natl Acad Sci USA* 70(5):1461-1463, 1973.

Blood leukocyte cultures were incubated with antioxidants and the carcinogens sodium cyclamate and 7,12-dimethylbenz(α)anthracene in different combinations. There were 17.4% more chromosomal break in the group of cells treated with dimethylbenzanthracene only than in the untreated controls. The reductions in chromosomal breaks by the antioxidants were as follows: ascorbic acid, 31.7%; butylated hydroxytoluene, 63.8%; Na_2SeO_3, 42.0%; and *dl*-α-tocopherol, 63.2%. Multiple chromosomal breaks were distributed equally throughout the experimental groups. Sodium cyclamate had only slightly more chromosomal breaks than the controls (11.6 compared to 10.9%). In the cyclamate groups treated with Na_2SeO_3, 11.2% of chromosomes were broken. More acrocentric-type chromosomal breaks (21.7%) were seen in the untreated cells than the cells treated with cyclamate (3.4%) or dimethylbenzanthracene alone (4.8%). The carcinogen-treate groups had a higher percentage of meta breaks than the untreated controls. The protection against chromosomal breakage by antioxidants appears to be consistent with epidemiological evidence that antioxidants prevent cancer. The anti-carcinogenic effect in tissue culture, however, cannot be differentiated from mutagenic, teratogenic, or cytotoxic effects.

1878 DEMONSTRATION OF NITROSAMINE-INDUCED TUMOURS IN THE RESPIRATORY TRACT OF THE EUROPEAN HAMSTER (*CRICETUS CRICETUS* L.) BY X-RAY EXAMINATION. *(E.)* Eckel, H. (Hannover Med. Sch., Germany), H. Reznik-Schüller, G. Reznik, J. Hilfrich and U. Mohr. *Strahlentherapie* 145(5):600-603, 1973.

Captured European hamsters (*Cricetus cricetus* L.) were subcutaneously treated once weekly for life wit 1/5, 1/10, or 1/20 LD_{50} of N-diethylnitrosamine (DEN or N-dibutylnitrosamine (DBN). Eight weeks after th beginning of treatment, the thorax and skulls were X-rayed every 14 days. Tumors of the lung and of the nasal cavities with a minimum diameter of 2 mm were observed as early as 12 wk after beginning treatment in two animals in the highest DEN group. Similar tumors were found 2 wk later in the

imals of the low dosage groups. The first
aor of the nasal cavity was detected by X-
7 22 wk after the beginning of treatment. Compari-
a of sequential X-rays showed the diameter to have
creased to 12 x 16 mm 32 wk after beginning treat-
nt. The results demonstrate that experimentally
duced tumors of the respiratory tract in European
msters can be detected by X-ray examination when
ey are 2 mm in diameter and the growth rate ob-
rved and the status of the tumors described before
ath. Autopsies verified the tumors detected by
rays.

79 TRANSPLANTABLE MYELOID RAT LEUKAEMIA INDUCED BY 7,12-DIMETHYLBENZ(A)ANTHRACENE.

.) Gal, F. (Res. Inst. Oncopathol., Budapest,
ngary), S. Somfai and Z. Szentirmay. *Acta*
ematol (Basel) 49(5):281-290, 1973.

e histological, hematological and biological
atures of a transplantable myeloid rat leukemia
duced by repeated i.v. administrations of 7,12-di-
thylbenz(a)anthracene (DMBA) were examined. For
e leukemogenesis, 50 two-month-old rats, weighing
0-150 g were used, of which 25 were WOP/H-Onc
bred females and 25 Wistar/H-Onc outbred (12
males and 13 males) animals. With four i.v. ad-
nistrations of DMBA, leukemia was induced in only
% of the animals; this leukemia proved to be of the
eloid type. In the course of transplantations two
rms developed: 1) the subacute form maintained
th liver and spleen cell suspensions, yielding a
ue leukemic blood picture; and 2) the acute as-
tic form without a leukemic blood picture. The
bacute form of leukemia following serial trans-
antation kept its original myeloid character.

80 EFFECTS OF DIETHYLSTILBESTROL ON HYBRIDIZABILITY OF MOUSE TESTICULAR RNA. *(E.)*

toh, K. (U. Utah Coll. Med., Salt Lake City),
A. Huseby, A. Baldi and L. T. Samuels. *Cancer*
s 33(6):1247-1252, 1973.

toplasmic RNA's (cyt RNA's) were prepared from
yptorchid testes of BALB/c mice after treatment
th diethylstilbestrol (DES) for three days and
, 22, 26, and 28 weeks. Testes of this strain
ow a "spurt" in DNA synthesis within three days
ter beginning DES treatment and develop a
gh proportion of malignant interstitial cell
mors when treated with the estrogen for periods
seven months or longer. Comparable preparations
re made from untreated animals of similar ages.
e cyt RNA's were then tested for their ability
compete against RNA-^{3}H synthesized on mouse DNA
vitro, using both "presaturation" and simultane-
s hybridization techniques. The preparations
om the DES-treated animals always competed better
an the equivalent controls. The difference
s greater after long periods of treatment.
t RNA's from cryptorchid testes of DES-treated
H_{B1} X A_{B1} F_1 mice treated for three days with DES
d not show any increase in competitive ability
er untreated controls. This strain develops rela-
vely few Leydig cell tumors after a much longer
riod of estrogen treatment and does not exhibit an early "spurt" in DNA synthesis in the interstitium. Preparations of cyt RNA from the livers of BALB/c mice treated with DES also did not compete better than similar preparations from livers of untreated controls. There appears to be a correlation between the DNA "spurt" in the interstitial cells, the tendency to develop Leydig cell tumors, and the increased ability of components of testicular cyt RNA's to compete for sites on mouse DNA. The evidence supports an early and continuing effect of DES on the nucleic acid metabolism of the testes of strains of mice which ultimately develop interstitial cell tumors while no such effect could be identified in a strain with low tumor incidence.

1881 HETEROGENEITY OF FERRITIN FROM 3'METHYL-4-(DIMETHYLAMINO)AZOBENZENE-INDUCED HEPATOMAS.

(E.) Urushizaki, I. (Sapporo Med. Coll., Japan), K. Ishitani, H. Natori, M. Yokota, M. Kitago and Y. Niitsu. *Gann* 64(3):237-246, 1973.

Ferritins from hepatomas induced by 3'-methyl-4-(dimethylamino)azobenzene(3'-Me-DAB) were compared with ferritins from liver of Wistar strain rats in which the original hepatomas developed. Three of five ferritin samples isolated from 3'-Me-DAB hepatomas migrated faster than rat liver ferritin; the remaining two samples migrated normally in disc electrophoresis on polyacrylamide gels. Electrofocusing column fractionation by carrier ampholytes with a narrow pH range was employed for the qualitative analysis of ferritins from 3'-Me-DAB-induced hepatomas and normal liver. The ferritins obtained from normal rat liver were fractionated into three heterogeneous peaks with pH values of 5.16, 5.24, and 5.29. Ferritins from 3'-Me-DAB-induced hepatomas, which had fast mobility on electrophoresis, showed four heterogeneous peaks with pH values of 5.05, 5.16, 5.23, and 5.29. The appearance of acidic peaks of ferritin with a pH below 5.08 was characteristic as tumor ferritin in 3'-Me-DAB-induced hepatomas. Immunological tests by immunodiffusion with rabbit antisera specific for rat liver ferritin showed no antigenic differences between normal and tumor ferritin.

1882 CORRELATION OF CARCINOGENIC ACTIVITY OF 4-NITROQUINOLINE 1-OXIDE AND RELATED COMPOUNDS WITH NEAR-ULTRAVIOLET ABSORPTION CHARACTERISTICS AND PARTITION COEFFICIENT.

(E.) Okano, T. (Pharmaceutical Inst., Tohoku U., Japan), J. Maenosono, T. Kano and I. Onoda. *Gann* 64(3):227-236, 1973.

Absorption spectra and partition coefficients between octanol and a buffer of pH 7.0 were measured on 32 kinds of compounds related to 4-nitroquinoline 1-oxide, including 17 strongly or weakly carcinogenic compounds and 15 noncarcinogenic compounds. The measured values were compared with the known carcinogenic activity. It was found that the carcinogenicity of 4-nitroquinoline 1-oxide and related compounds depended on the partition coefficient (P) and on the molar extinction coefficient at 260 nm (ε_{260}) and the absolute value ($|\Delta\lambda|$) of the wavelength difference between the absorption maximum of the band nearest 260 nm and 260 nm. The value of Γ defined by the equation $\Gamma = \log \varepsilon_{260} - \log(|\Delta\lambda|+1) + \log P$ corresponded well with the carcinogenicity of 4-nitroquinoline 1-oxide and related compounds. When plotted on a graph with axes of $[\log \varepsilon_{260} - \log(|\Delta\lambda|+1)]$ and log P, 31 of these compounds, excluding 4-hydroxyaminoquinoline 1-oxide, which was presumed to show the activity by metabolic conversion to an ultimate carcinogenic form, were distinctly divided into two groups of carcinogenic and noncarcinogenic compounds. The reason for the dependence of carcinogenicity on the absorption characteristic of a compound in the region of 260 nm and on its partition coefficient is discussed from the electronic perturbation of DNA by the quinoline compound which causes changes in the genetic information, and from the permeability of the compound through a biomembrane.

1883 INDUCTION OF MITOTIC CROSSING OVER IN *SACCHAROMYCES CEREVISIAE* BY BREAKDOWN PRODUCTS OF DIMETHYLNITROSAMINE, DIETHYLNITROSAMINE, 1-NAPHTHYLAMINE AND 2-NAPHTHYLAMINE FORMED BY AN *IN VITRO* HYDROXYLATION SYSTEM.

(E.) V. W. Mayer (Food and Drug Administration, Washington, D.C.). *Genetics* 74(3):433-442, 1973.

The induction of mitotic crossing over in *Saccharomyces cerevisiae* by breakdown products of dimethylnitrosamine, diethylnitrosamine, 1-naphthylamine and 2-naphthylamine formed in the UDENFRIEND hydroxylation medium is reported. Mitotic crossing over was detected as red sectored colonies resulting from induced homozygosity of the *ade*2 marker. Dimethylamine and diethylamine, which lack the nitroso group of the nitrosamines, did not induce mitotic crossing over under any of the test conditions. To further confirm that the induced sectored colonies were the result of mitotic crossing over, they were tested for the presence of reciprocal products. The expected reciprocal products were found in over 67% of the isolates tested. It is concluded that mitotic recombination is a significant and practical indicator of genetic damage potential of chemicals.

1884 EFFECT OF VARIOUS FACTORS ON INDUCTION OF URINARY BLADDER TUMORS IN ANIMALS BY N-BUTYL-N-(4-HYDROXYBUTYL)NITROSOAMINE.

(E.) Ito, N. (Nara Med. U., Japan), K. Matayoshi, M. Arai, Y. Yoshioka, Y. Kammamoto, S. Makiura and S. Sugihara. *Gann* 64(2):151-159, 1973.

The effect of various factors on the development of urinary bladder tumor in rats, guinea pigs, and hamsters given N-butyl-N-(4-hydroxybutyl)nitrosoamine was examined histologically. The effect of sex and age, and of the dose and period of administration of N-butyl-N-(4-hydroxybutyl)nitrosoamine on the incidence of urinary bladder tumors in rats was also investigated. No sex difference was found in the incidence of urinary bladder cancer in rats. However, the incidence of cancer and of squamous metaplasia in areas of cancerous tissues was greatly influenced by the age of animals, being much higher in older rats than in younger ones. For development of urinary bladder cancer in rats, the minimum carcinogenic dose of N-butyl-N-(4-hydroxybutyl)nitrosoamine given in the drinking water is 0.005% while 0.001% is the maximum non-carcinogenic dose. On administration of 0.1% carcinogen solution to rats, the minimum period for the induction of urinary bladder cancer is 4 wk. Guinea pigs and hamsters were less susceptible than Wistar strain rats to the induction of urinary bladder cancer by N-butyl-N-(4-hydroxybutyl)nitrosoamine The findings suggest that the urinary concentration of the metabolite, butyl(3-carboxypropyl)-nitrosoamine, may be important in inducing bladder cancer in rats. A difference in the sensitivity of different species of animals to the carcinogen may be due to the difference in the concentration of butyl(3-carboxypropyl)-nitrosoamine in the urine.

1885 EFFECT OF HYDROGEN PEROXIDE, FENTON'S REAGENT, AND IRON IONS ON THE CARCINOGENICIT OF 3,4-BENZOPYRENE.

(E.) Nagata, C. (Natl. Cancer Ctr. Res. Inst., Tokyo, Japan), Y. Tagashira, M. Kodama, Y. Ioki and S. Oboshi. *Gann* 64(3):277-285, 1973.

Effects of hydrogen peroxide (H_2O_2), Fenton's reagent (H_2O_2 + $FeCl_2$), ferrous chloride ($FeCl_2$), and ferric chloride ($FeCl_3$) on the carcinogenicity of benzo(a)pyrene in female mice of ddN strain, about 30 days old and weighing 18-25 g, were studied. The animals were given s.c. injections of freshly prepared sample on their backs. Immediately after the injection of the first sample, the second one was injected at the same site. $FeCl_2$, $FeCl_3$, or H_2O_2 alone showed no effect. Some accelerating effect in terms of Iball index was observed after a single application of Fenton's reagent. Two possibilities are suggested for this effect: 1) that the OH radical acts as an activator of benzo(a)pyrene, and 2) that OH or O_2H radical makes the tissue or organ sensitive to chemical carcinogenesis through direct attack on the cells. Repeated applications of H_2O_2 markedly inhibited the carcinogenicity of benzo(a)-pyrene. This may be due to the toxic effect of H_2O_2 on the cell.

1886 CARCINOGENIC EFFECT OF N-METHYL-N'-NITRO-N-NITROSOGUANIDINE WITH VARIOUS KIND OF SURFACTANT IN THE GLANDULAR STOMACH OF RATS.

(E. Takahashi, M. (Nagoya City U. Med. Sch., Japan), S. Fukushima and H. Sato. *Gann* 64(3):211-218, 1973.

The effect of various surfactants on the production of adenocarcinomas by N-methyl-N'-nitro-N-nitrosoguanidine in the glandular stomach of rats was studied. Sixty-eight male Wistar rats, weighing from 140 to 155 g at the beginning of the experiment were divided into four experimental groups and a control group. The four experimental groups were administered various concentrations of the nitrosoguanidine in combination with a surfactant. The control group received the nitrosoguanidine only. Surfactants used were polyoxyethylene sorbitan monostearate, polyoxyethylene nonyl phenyl ether, and two types of alkylbenzenesulfonate. Many cases of undifferentiated adenocarcinoma consisting of anaplastic glandular cells were induced in the experimental groups. Lymphatic invasion of cancer cells was found in six cases, and one metastasized to intestinal lymph nodes. On the contrary, although the control group revealed many cases of well-differentiated adenocarcinomas, neither lymphatic invasion nor metastasis was found. There were no differences in the rate and time of tumor induction among the four experimental groups and the control. The four experimental groups, however, did exhibit more prominent carcinogenic reactions than the control group. This is explained by the strong penetrating properties of the surfactants which enables the nitrosoguanidine to invade the protective mucous barrier of the stomach and contact the gastric mucosa directly when administered with one of the surfactants as a vehicle. Surfactants might also facilitate the penetration of N-methyl-N'-nitro-N-nitrosoguanidine into target cells of the gastric mucosa.

887 GLUTATHIONE REDUCTASE AND GLUTATHIONE PEROXIDASE ACTIVITIES IN HEPATOMOUS LIVERS OF RATS TREATED WITH DIETHYLNITROSAMINE.

(E.) Pinto, . E. (Dept. Biochem., U. Sheffield, England) and W. Bartley. *FEBS Letters* 32(2):307-309, 1973.

Glutathione reductase and glutathione peroxidase activities were compared in homogenates of normal rat liver and diethylnitrosamine-induced hepatoma. Tumor induction was carried out by adding 50 mg/l diethylnitrosamine to the drinking water of Wistar rats for 4 months. Reductase activity of hepatomatous liver was about 2.2 times that of normal liver and was independent of sex. Peroxidase activity, which was twice as great in females as males, was decreased in hepatomatous liver to 60% of the normal liver value, with maintenance of the normal sex difference. Peroxidase/reductase ratios of hepatomas, which were .7 ± 1.0 and 6.8 ± 1.8 in males and females, resp., were one-third to one-half the ratios of normal liver. Study of enzyme activities in liver from starved male animals indicated that the decrease of peroxidase activity, but not the increase of reductase activity, in hepatomas could be accounted for by simple starvation and weight loss.

1888 THE ACTIVITY OF SOME NITROSO COMPOUNDS IN THE MOUSE DOMINANT-LETHAL MUTATION ASSAY. I. ACTIVITY OF *N*-NITROSO-*N*-METHYLUREA, *N*-METHYL-*N*-NITROSO-*N*'-NITROGUANIDINE AND *N*-NITROSOMORPHOLINE.

(E.) Parkin, R. (Middlesex Hosp. Med. Sch., London, England), H. B. Waynforth and P. N. Magee. *Mutat Res* 21(3):155-161, 1973.

N-Nitroso-*N*-methylurea (NMU) at a dose of 50 mg/kg body wt significantly increased the incidence of early fetal death (dominant lethal mutations) when compared with the solvent controls in the mouse dominant lethal test. The mutagenic activity of NMU was considerably less than that of methylmethanesulphonate (MMS) which was used as a positive control mutagen but, in comparison, it affected a greater part of the spermatogenic cycle of the treated male mice. *N*-Methyl-*N*-nitroso-*N*'-nitroguanidine (MNNG) produced a small increase in dominant lethal mutations with a low level of significance ($P < 0.05$) at doses of 50 and 70 mg/kg body wt during the first week only of the 8-wk assay period. A highly significant, though still moderately increased response was found for the near-LD_{50} dose of 100 mg/kg. At this dose level the mutagenic activity was confined to the first 4 days of the first week. *N*-Nitrosomorpholine could not be tested during the first 3 to 4 wk of the assay at dose levels of 50 and 100 mg/kg due to an adverse effect on mating activity but no increase in the incidence of dominant lethal mutations was observed between the 5th and 8th wk. At a dose of 35 mg/kg mating activity was normal during the initial 3-wk period studied but there was no evidence of increased mutations. These results indicate that any program for the comprehensive screening of compounds for mutagenic activity should include more than one assay procedure. Moreover, negative results can only be meaningful if the highest dose levels compatible with adequate mating activity have been used.

1889 TRIAZENES, A NEW GROUP OF INDIRECT MUTAGENS. COMPARATIVE INVESTIGATIONS OF THE GENETIC EFFECTS OF DIFFERENT ARYLDIALKYLTRIAZENES USING *SACCHAROMYCES CEREVISIAE*, THE HOST-MEDIATED ASSAY, *DROSOPHILA MELANOGASTER*, AND HUMAN CHROMOSOMES *IN VITRO*.

(E.) Vogel, E. (Cent. Lab. Mutation Res., Freiburg, Germany), R. Fahrig and G. Obe. *Mutat Res* 21(3):123-136, 1973.

Four genetic test systems were compared based on the activation of "indirect" mutagens in each. Three aryldialkyltriazenes were tested: I-phenyl-3,3-dimethyltriazene (PDT), I-(pyridyl-3)-3,3-dimethyltriazene (PyDT) and I-(pyridyl-3-N-oxide)-3,3-dimethyltriazene (PyNDT). In the first system mitotic gene conversion was observed in *Saccharomyces cerevisiae*. Secondly, the host-mediated assay (HMA) was set up using the mouse and Saccharomyces. In *Drosophila melanogaster* various genetic abnormalities including X-linked recessive lethals, 2-3 translocations, complete loss of the X-or Y-chromosome, and partial loss of the Y were analysed after treatment. Finally, chromosome aberrations were determined in human leukocytes cultured *in vitro*. Some evidence that only PyDT and PyNDT can be designated as "indirect" mutagens was obtained. PDT, on the other hand, also seems to act as a "direct" mutagen. The relative genetic activity of the triazenes in the different test systems can be summarized as follows: (a) Saccharomyces PDT > PyDT (PyNDT no effect); (b) HMA PyDT ≃ PyNDT >PDT; (c) Drosophila PDT > PyNDT > PyDT; (d) Human chromosomes PDT ≃ PyDT ≃ PyNDT (questionable effects). PDT and PyNDT induced recessive lethals and chromosome aberrations in the male germ cells of Drosophila. These genetic changes were observed in comparable frequencies for both chemicals. PyNDT, however, was quite ineffective in causing dominant lethals, whereas PDT showed strong activity in this test. This suggests that the dominant lethals produced by PDT hardly can be due to chromosome breakage but rather are caused by the highly cytotoxic properties of PDT. In the *in vitro* test with human chromosomes the triazenes showed very weak dose-independent effects, suggesting that potent "indirect" mutagens of the triazene type cannot be recognized in this test system.

1890 TIME RELATED CHANGES OF SOME EFFECTS OF AMARANTH AND BUTTER YELLOW IN RATS. *(Fr.)*

Albrecht, R. (Conservatoire Natl. Arts Metiers, Paris, France), Ph. Manchon, C. Keko-Pinto and R. Lowy. *Food Cosmet Toxicol* 11:175-184, 1973.

Weanling (30-day old) male Wister CF rats were fed a diet containing either amaranth (0.3 or 1%) or butter yellow (0.12%) for 1-8 months. Groups of animals were sacrificed after having ingested an average 1.4, 3, 16 or 43 g, resp. of amaranth or 0.6, 1.2, 1.9 or 5.1 g, resp. of butter yellow. The activities of several hepatic enzymes were compared to those of controls. Amaranth caused only slight and transient changes and was considered nontoxic in the doses used. Butter yellow had a pronounced effect on enzyme activity. It greatly reduced the activity of glucose-6-phosphatase at 2, 4 and 9 months but not at 3 months. It induced biosynthesis of glucose-6-phosphate dehydrogenase and of phosphogluconate dehydrogenase at 2 and 9 months but not at 3 and 4 months. Changes in activity related to duration of administration confirmed histological studies which indicated that hepatocarcinogenesis involves successive processes of cell destruction and regeneration. Azoreductase activity was increased when a water-soluble substrate was used (in contrast to reported reduction in activity when a fat-soluble substrate was employed). Different effects of the carcinogen on azoreductase, depending upon the type of substrate, suggest that several azoreductases are present in the liver.

1891 COMPARATIVE CARCINOGENICITY IN SWISS MICE OF *N*-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]-ACETAMIDE AND STRUCTURALLY RELATED 5-NITROFURANS AND 4-NITROBENZENES. *(E.)*

Cohen, S. M. (U. Wisconsin Med. Sch., Madison), G. M. Lower, Jr., E. Ertürk and G. T. Bryan. *Cancer Res* 33(7):1593-1597, 1973.

Eleven different 5-nitrofurans, 4-nitrobenzenes, and 4-aminobenzenes were fed to female Swiss mice to determine structure-carcinogenicity relationships with respect to organ specificity. *N*-[4-(5-Nitro-2-furyl)-2-thiazolyl]acetamide (NFTA, 0.1 and 0.05% of diet) induced a high incidence of lymphocytic leukemia (44/52 animals) and a low incidence of forestomach squamous cell papillomas after 14 wk of feeding. Four other chemicals in which the acetyl group was replaced by a formyl group induced few leukemias or forestomach tumors, although one of these (*N*-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide) induced urinary bladder tumors in 20 of 21 mice. These results suggest that the acetyl group of NFTA is necessary for its leukemogenic activity. Six other free aminothiazole derivatives induced a high incidence of forestomach tumors with few leukemias.

1892 CARCINOGENIC ACTION OF DIISOPROPYLSULFATE (DIPS) AND ITS ABSENCE WITH MONOMETHYLSULFATE IN BD RATS. *(Ger.)*

Druckrey, H. (Res. Group Preventative Med., Freiburg, Germany), J. Gimmy and C. Landschütz. *Z Krebsforsch* 79(2):135-140, 1973.

Starting 190 days after the first injection, 14 of 15 adult BD rats died of sarcomas which developed at the site where diisopropylsulfate (DIPS; 100 mg/kg/week s.c. for 15 weeks) had been injected. Polymorphocellular sarcomas were found in 10 and spindle cell sarcomas in 4. To determine whether the tumors were induced by carcinogenic activity or local nonspecific irritation, 18 rats were given a single dos of 1000 mg/kg of DIPS. After a mean induction peri of 325 days, 5 of the 18 developed polymorphocellular sarcomas at the site of injection; 2 of the 5 had multiple pulmonary metastases. Another rat had a squamous cell carcinoma in the forestomach at autopsy. After a mean survival time of 476 days, 11 of 15 rats, injected with a single dose of 300 mg/kg DIPS, developed malignant tumors. The tumors included polymorphocellular sarcomas (3), spindle cell sarcomas (2), fibrosarcoma (1), myosarcoma (1), and myxosarcoma (1) at the site of injection. Other tumors were 1 fibrosarcoma of the left orbit, 1 cecal fibrosarcoma, and 1 pleomorphic glioma in the brain. A squamous cell carcinoma was found in the forestomach of 1 of 3 rats autopsied 365 days after a single p.o. dose of 1500 mg/kg DIPS. No tumors were found in 20 BD rats given 100 mg/kg/week s.c. of monomethylsulfate (total dose of 6300 mg/kg) in a 10% solution; the mean survival time was 746 days and the maximum, 978 days. These were the same as survival times in untreated controls.

1893 THE PASSAGE OF 9,10-DIMETHYLBENZ(a)ANTHRACENE THROUGH THE PLACENTA AND THE DYNAMICS OF ITS ENTRANCE INTO THE FETUS IN RATS. *(Rus.)*

Aleksandrov, V. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR) and I. A. Shendrikova. *Vop Onkol* 18(10):56-61, 1972.

9,10-Dimethylbenz(a)anthracene (DMBA; 25 mg/kg i.v.) was injected in a water-lipid emulsion into noninbre albino rats on day 21 of gestation. Rats were killed 5 min to 5 hr after injection and fetuses, placentas and maternal livers were removed. DMBA was detected in fetal tissue as soon as 5 min after it was injected into the mothers, reached a maximum at 60 min, decreased to 1/3 of this level at 2-3 hr after injection, and was present only in trace quantities after 5 hr. Because of the rich blood supply in the maternal liver and placenta, maximum DMBA contents in these organs were reached 5 min after injection. After 1-2 hr DMBA contents decreased rapidly in these organs. It should be emphasized that the DMBA content of the maternal liver was significantly higher than that in fetal tissue during the entire period studied, while the DMBA content of the placenta was higher than that in fetal tissue during the first 45 min and was the same or lower later. These findings suggest that it takes DMBA 40-60 min to cross the placental barrier, probably by diffusion.

1894 *IN VIVO* AND *IN VITRO* BINDING OF CARBON TETRACHLORIDE WITH NUCLEIC ACIDS AND PROTEINS IN RAT AND MOUSE LIVER. *(E.)*

Rocchi, P. (Inst Gen. Path., U. Bologna, Italy), G. Prodi, S. Grilli and A. M. Ferreri. *Int J Cancer* 11(2):419-425, 1973.

The binding of carbon tetrachloride (CCl_4) with nucleic acids in mice and rats *in vivo*, and with DNA

d polynucleotides *in vitro* was examined. Adult
le Wistar rats weighing 280 g and male Swiss mice
ighing 30 g were injected i.p. with 3-methylchol-
threne (MCA) (5 mg in 0.5 ml corn oil) 24 hr before
l_4 treatment. A control group received the i.p.
l_4 injection but was not pretreated with MCA. The
imals were killed 12 hr after carcinogen administra-
on. Four fractions, DNA, rRNA, nuclear proteins
d cytoplasmic proteins, were extracted from the
oled livers. It was found that ^{14}C-labelled CCl_4
nds *in vivo* to DNA of mouse liver and to rRNA of
t liver if the animals have been pretreated with
A. A noticeable amount of radioactivity was also
served in liver proteins. *In vitro* CCl_4 is acti-
ted by microsomes and pH 5 enzymes of MCA-treated
imals to a metabolite which can react with DNA and
lynucleotides; this effect is more evident if sub-
llular fractions from mouse liver are used. The
fferences found in binding to DNA and RNA in mice
d rats may be due to a different pattern of enzymes
microsomal fractions.

95 STUDIES OF ALKYLATION OF NUCLEIC ACIDS IN RATS BY CYCLIC NITROSAMINES. *(E.)* Lijinsky,
(U. Nebraska Med. Ctr., Omaha), L. Keefer, J. Loo
d A. E. Ross. *Cancer Res* 33(7):1634-1641, 1973.

e metabolic fate of five deuterium-labeled cyclic
trosamines, nitrosoazetidine, nitrosopyrrolidine,
trosopiperidine, nitrosomorpholone, and dinitro-
piperazine, was studied in liver and esophagus
llowing administration to adult rats by gavage.
cleic acids from the organs of the treated animals
re purified and hydrolyzed and the presence of
beled aklylated bases was assayed by mass spectro-
try. No deuterium-labeled alkylated bases were
tected in the nucleic acid hydrolysates of any of
e cyclic nitrosamine-treated rats. Rats treated
th a sixth compound, nitrosomethylcyclohexylamine,
re the only ones which contained a deuterium-
beled 7-methyl-guanine which was present in liver,
t not esophageal, nucleic acid hydrolysates,
though esophagus rather than liver is a target
gan for nitrosamine. These results indicate no
rrelation between carcinogenic activity of the
trosamines and *in vivo* alkylation of nucleic acids.
sults of experiments using ^{3}H-labeled nitrosamines
ggested that the alkylation of nucleic acids by
beled nitrosamines previously reported by others
s probably due to the presence of trace contaminants
^{3}H-labeled methylalkylnitrosamines.

96 CHARACTERIZATION OF CYTOCHROME P-450 SPECIES IN RAT LIVER MICROSOMES, I.
FFERENCES IN THE *O*-DEALKYLATION OF 7-ETHOXYCOUMARIN
TER PRETREATMENT WITH PHENOBARBITAL AND 3-METHYL
OLANTHRENE. *(E.)* Ullrich, V. (Physiological-
em. Inst., U. Saarland, Homburg, Germany. *Hoppe
ylers Z Physiol Chem* 354(5):514-520, 1973.

97 RELATIONSHIP BETWEEN VAGOTOMY, PEPTIC ULCER AND GASTRIC ADENOCARCINOMA IN RATS
D 2,7-DIACETYLAMINOFLUORENE. *(E.)* Kowalewski, K.
ept. Surg., U. Alberta, Edmonton, Canada). *Can
Surg* 16(3):210-217, 1973.

1898 LEUKEMIA AFTER CHEMOTHERAPY OF HODGKIN'S DISEASE. *(E.)* Castro, G. A. M. (U. Massachusetts Med. Sch., Worcester), A. Church, L. Pechet and L. M. Snyder. *N Engl J Med* 289(2): 103-104, 1973.

1899 SMOKING AND CANCER. *(E.)* Cole, T. J. (Llandough Hosp., Penarth, Glamorgan, Wales). *Lancet* (7819):49, 1973.

1900 CORRELATION BETWEEN LETHALITY AND DNA SINGLE-STRAND BREAKS IN *BACILLUS SUBTILIS* CELLS TREATED WITH *N*-METHYL-*N'*-NITRO-*N*-NITROSOGUANIDINE. *(E.)* Dugle, D. L. (Med. Biophys Branch, Whiteshell Nuclear Res. Establishment, Pinawa, Manitoba, Canada), C. E. Campbell, B. E. Meeker and C. J. Gillespie. *Mutat Res* 18(3):237-245, 1973.

1901 POLY(ADENOSINE DIPHOSPHATE RIBOSE) SYNTHESIS DURING THE CELL CYCLE OF TRANSFORMED HAMSTER LUNG CELLS. *(E.)* Miwa, M. (Inst. Med. Sci., Tokyo U., Japan), T. Sugimura, N. Inui and S. Takayama. *Cancer Res* 33(6):1306-1309, 1973.

1902 SPECIAL SUSCEPTIBILITY OF CLARA CELLS OF THE MURINE TERMINAL BRONCHIOLE TO 4-NITROQUINOLINE 1-OXIDE: AN ELECTRON MICROSCOPIC OBSERVATION. *(E.)* Terao, K. (Sch. Med., Chiba U., Japan) and H. Otsu. *Gann* 64(2):179-181, 1973.

1903 DISSOCIATION OF RIBOSOMES PRODUCED BY DIMETHYLNITROSAMINE AND LASIOCARPINE IN MOUSE LIVER. *(E.)* Anonymous. *Biochem Pharmacol* 22(13):1681-1682, 1973.

1904 MULTIPLE HALO NEVI DEVELOPING AFTER OXYMETHOLONE TREATMENT OF "APLASTIC ANEMIA". *(E.)* Jhung, J. W. (St. Vincent Hosp., Worcester, Mass.), L. M. Edelstein, A. Church, L. M. Snyder, R. W. Bain, S. Castro and N. Cariglia. *Cutis* 12(1):56-61, 1973.

1905 MODIFICATION OF THREE ENZYMES OF THE β-KETOADIPATE PATHWAY BY *N*-METHYL-*N'*-NITRO-*N*-NITROSOGUANIDINE. *(E.)* Johnson, B. F. (Palo Alto Med. Res. Fdn., Calif.) and J. Greenberg. *Chem Biol Interact* 7(1):17-28, 1973.

1906 ELECTRON MICROSCOPIC STUDIES OF MONONUCLEAR CELLS FROM DMBA-INDUCED TUMORS IN THE RAT. *(E.)* Gogate, S. A. (Dept. Path., Ohio St. U., Columbus) and E. von Haam. *Acta Cytol* 17(4):372-375, 1973.

1907 ENZYME HISTOCHEMISTRY OF EXPERIMENTAL OVARIAN TUMORS IN MICE. *(E.)* Ueda, G. (Osaka U., Med. Sch., Japan), K. Hayakawa, N. Hamanaka, S. Yoshinare, Y. Sato and Y. Okudaira. *Acta Obstet Gynaecol Jap* 19(1):16-24, 1972.

1908 SENSITIVITY OF EMBRYONAL LUNG TISSUE OF MICE OF A AND C57Bl LINES TO TRANSPLACENTAL ACTION OF URETHANE IN ORGANIC CULTURES. *(Rus.)* Kolesnichenko, T. S. (Inst. Exp., Clin. Oncology, U.S.S.R. Acad. Med. Sci., Moscow). *Biull Eksp Biol Med* (5):71-73, 1973.

1909 LATENT EFFECT OF DMBA ON ADRENAL GLANDS OF SPRAGUE-DAWLEY RATS: AN ULTRASTRUCTURAL STUDY. *(E.)* Murad, T. M. (U. Alabama Med. Ctr., Birmingham), J. Liebach and E. von Haam. *Exp Mol Pathol* 18(3):305-315, 1973.

See also:

* (Rev): 1801, 1802, 1810
* (Viral): 1981
* (Immun): 2067, 2078, 2088, 2105
* (Path): 2131, 2141
* (Epid-Biom): 2147

ı A MOLECULAR THEORY OF CELL SURVIVAL. *(E.)*
Chadwick, K. H. (Inst. Atomic Aci. Agri-
ure, Wageningen, Netherlands) and H. P. Leenhouts.
Med Biol 18(1):78-87, 1973.

eory is presented to explain the effect of ra-
ion on cell survival. The theory is based on
assumption that a double strand break in the DNA
x is the critical damage. The theory is derived
the radiation induced molecular bond breaks in
DNA strands and parameters are included to take
unt of various repair processes which may occur
een the radiation event and the biological
lt. Implications of the theory with respect to
tive biological effect, the oxygen effect and
ological protection are mentioned and a fit of
theoretically derived expression to experimental
for 250 kV_p X-rays and 15 MeV neutrons is pre-
ed. An appendix contains data which show that
enzymatic repair of single strand breaks in
is in accordance with the theoretical analysis
rotracted irradiations and that a coherent
ysis of the variation of radiation sensitivity
he cell cycle is in strong support of the
ary assumption that a double strand break in the
helix is the critical damage leading to cell
oductive death.

RESPONSE OF γ-IRRADIATED MOUSE SALIVARY
GLAND CELLS TO A PROLIFERATIVE STIMULUS.
Sasaki, T. (Tokyo Med. Dental U., Sch. Dentis-
Japan) and T. Nogami. *Cancer Res* 33(7):1701-
, 1973.

effect of γ-irradiation during G_0 phase on the
ntial of a cell to be stimulated into DNA syn-
sis was examined. Male SWR mice, weighing about
, were used in the experiments. It was found
the stimulation of DNA synthesis in mouse
ivary gland cells caused by a single administra-
of isoproterenol was inhibited by prior γ-irrad-
ion to the upper one-third of the body in a dose-
ndent manner. This effect of irradiation was due
decrease in the fraction of acinar cells stimu-
ed to enter the S phase, rather than an abscopal
ect of irradiation. Isoproterenol-stimulated DNA
thesis in irradiated salivary glands gradually
reased several days following irradiation; it
ame identical to that in unirradiated mice after
days. These observations indicate that the capac-
of salivary gland cells to synthesize DNA was
porarily depressed by irradiation and recovered
er irradiation. However, the newly synthesized
in irradiated cells was found to be quickly de-
ded, while that in unirradiated controls was rela-
ely stable. An autoradiographic analysis of the
otids indicated that the degradation of the DNA
due, at least in part, to degeneration of acinar
ls in the irradiated glands.

2 POPULATION HETEROGENEITY HYPOTHESIS ON
RADIATION INDUCED CANCER. *(E.)* Baum,
W. (Health Phys. Safety Div., Brookhaven Nat. Lab.,
on, N.Y.). *Health Phys* 25:97-104, 1973.

lyses of dose-effect relations for radiation induced tumors and cancers in drosophila, mice, rats and man reveal that increase in incidence can often be represented by a simple power function of dose. The exponent depends on genetic factors, type of cancer, radiation quality, dose-rate, and so on. An important observation is that the exponent is frequently less than 1.0 for carcinogenesis in man. The proposed explanation for this is based on the fact that "man" is a heterogeneous population with mixed predisposition to cancer due to genetic differences, and sensitivity which varies with such factors as age, sex, environmental factors, cell type, and stage of cell cycle. At low doses, those fractions of the population which respond according to one-target kinetics tend to dominate the dose-effect relationship. In a mixed population, the most sensitive sub-group responds at the lowest dose and causes a steep slope in this portion of the dose-effect curve. At higher doses the less sensitive sub-groups become dominant with consequent reduction in slope of the dose-effect curve. The resultant curve can be convex upward. Hence, greater effects might be observed at low doses than would be predicted by linear extrapolation from higher doses. Thus, the assumption of a linear non-threshold relationship used by standards setting groups is not necessarily conservative in estimating cancer risk at low doses, particularly in a heterogeneous population such as man.

1913 PROTECTION AGAINST RADIATION LEUKEMOGENESIS BY REPEATED INJECTIONS OF IMMUNE AND NONIMMUNE FOREIGN SERA. *(E.)* Ferrer, J. F. (Stanford U. Sch. Med., Calif.), M. Lieberman and H. S. Kaplan. *Cancer Res* 33(6):1339-1343, 1973.

Significant inhibition of lymphoid tumor development was observed in X-irradiated strain C57BL mice treated with multiple injections of rat antiserum prepared by immunization of Wistar-Furth or (Wistar-Furth·BN) F_1 rats with histocompatible lymphomas induced by the radiation leukemia virus (RadLV). However, there was also significant inhibition, relative to phosphate-buffered saline controls, in C57BL mice that received injections of normal rat serum. This prompted additional experiments in which fetal calf serum or Freund's complete adjuvant were injected as nonspecific immunostimulants. Strong protection was observed with fetal calf serum, and a slight inhibitory effect (not significant) was also seen with Freund's complete adjuvant. The results are discussed in terms of the following alternative interpretations, which are not mutually incompatible: (*a*) the inhibitory effect of the rat anti-RadLV serum, as well as that of "normal" serum, which may have contained natural antibody to RadLV, is due to passive immunization by specific antiviral antibody, thus further implicating RadLV in the etiology of radiogenic lymphomas; (*b*) the protective effect is due to hyperimmunization of the mice against foreign (rat and bovine) sera, leading to nonspecific stimulation of the immune surveillance mechanisms of the host animals; and/or (*c*) prevention by the foreign sera is due to the radiation leukemia protection factor described by Berenblum *et al.*

1914 REPAIR REPLICATION IN X-IRRADIATED LYMPHOMA CELLS *IN VITRO*. *(E.)* Fox, M. (Christie Hosp., Manchester, England) and B. W. Fox. *Int J Radiat Biol* 23(4):333-358, 1973.

Repair replication using isopycnic CsCl density-gradient techniques has been studied in P388 cells and in two lines of L5178Y lymphoblasts showing a large difference in radiosensitivity. The results indicate that in all three cell-lines only small numbers of bases < 10 are inserted/damaged site. The radiosensitive L5178Y cell line consistently showed greater base insertion/damaged site than the radioresistant line and P388 cells. DNA of the two L5178Y cell-lines was equally sensitive to breakage by x-irradiation, as shown by alkaline sucrose gradient sedimentation techniques. A component of DNA associated with a lipid containing material has been identified which contaminates native DNA after all extraction procedures used. Characterization of this material suggests that it represents DNA attached to a membrane fraction. Such material when present may be mistaken for 'repair' incorporation. The present data fit the generalization that only small numbers of bases are inserted/break and are in close agreement with those published by Painter and Young (1972) for P388, B14FAF and W138 cells. The quantitative comparison of the amount of repair replication cannot be expected to give meaningful information as to the capacity of a cell to repair damage unless the nature of the initial reaction of the damaging agent with DNA is known. Although single-strand breaks are not lethal in mammalian cells, the significance of the way in which they are repaired is not known. It is further not known whether replication over a repaired DNA represents a damage fixation process in mammalian cells and whether the insertion of excess bases into the DNA, as appears to be the case in L5178Y cells, results in a greater number of mutations, a large number of which may be lethals, thus resulting in greater sensitivity.

1915 DNA LESIONS AND MAMMALIAN CELL KILLING: CAUSE AND EFFECT? *(E.)* Elkind, M. M. (Hammersmith Hosp., London, England) and E. Ben-Hur. *Isr J Chem* 10(6):1255-1272, 1972.

Some of the published biological evidence bearing on the relation between the lethal effects of cell irradiation and DNA damage and repair is reviewed. The available evidence indicates that repeated sublethal doses of X- of γ-irradiation have a cumulative effect in killing cells *in vitro* and that this effect reflects the ability of the cell to repair the radiation-induced damage over a finite time. Cells can be sensitized to X-ray killing by pretreatment with actinomycin D, suggesting that the site of damage and repair is nuclear DNA. Alkaline density gradient sedimentation studies have revealed two repair processes: repair of single-strand breaks, and repair of a DNA-lipid-protein complex. Although single-strand repair is rapid, the rate of repair of the complex varies inversely with the radiation dose used to induce the damage. Experimental results suggest that in Chinese hamster cells grown in the presence of the thymidine analog BUdR and subjected to killing by fluorescent light, the lethal event is the single-strand break which, in this case, cannot be repaired. However, results of similar experiment using X- instead of fluorescent-irradiation imply that damage to a DNA-membrane (i.e., complex) structure is a more likely target than single-strand breaks for X-ray cell killing.

1916 RADIATION CHEMISTRY OF NUCLEIC ACIDS. ISOLATION AND CHARACTERIZATION OF GLYCOLS OF 1-CARBAMYLIMIDAZOLIDONE AS PRODUCTS OF CYTOSINE. *(E.)* Hahn, B. S. (Dept. Biochem., Johns Hopkins U., Baltimore, Md.), S. Y. Wang, J. L. Flippen and I. L. Karle. *J Am Chem Soc* 95(8):2711-2712, 1973.

Two major isomeric products, *cis*- and *trans*-1-carbamylimidazolidone-4,5-diols, were isolated from the products of radiolysis of cytosine in oxygenated aqueous solution. The structures of the two compounds were confirmed from ultraviolet, infrared, nuclear magnetic resonance mass spectroscopy and X-ray diffraction studies. Although the mechanism of formation of the *cis* and *trans* products is obscure, it undoubtedly involves a facile rearrangement of an initial intermediate.

1917 AUTOPSY STUDY OF LEUKEMIA IN ATOMIC BOMB SURVIVORS, HIROSHIMA-NAGASAKI, 1949-1969. *(E.)* Liu, P. I. (Atomic Bomb Casualty Commission, Hiroshima, Nagasaki, Japan), T. Ishimaru, D. H. McGregor, T. Yamamoto and A. Steer. *Cancer* 31(6):1315-1327, 1973.

1918 INHIBITION OF REPAIR OF UV-DAMAGED DNA BY CAFFEINE AND MUTATION INDUCTION IN CHINESE HAMSTER CELLS. *(E.)* Trosko, J. E. (Dept. Human Development, Michigan St. U., East Lansing) and E. H. Y. Chu. *Chem Biol Interact* 6(5):317-332, 1973.

1919 MECHANISMS OF RADIATION-INDUCED STRAND BREA FORMATION IN DNA. *(E.)* Ward, J. F. (Lab. Nuclear Med., Radiation Biol., U. California, Los Angeles). *Isr J Chem* 10(6):1123-1138, 1972.

1920 CHROMOSOME ABERATION DOSIMETRY USING HUMAN LYMPHOCYTES IN SIMULATED PARTIAL BODY IRRADIATION. *(E.)* Lloyd, D. C. (Natl. Radiological Protection Board, Harwell, Dideot, Berkshire, England R. J. Purrott and G. W. Dolphin. *Phys Med Biol* 18(3):421-431, 1973.

1921 LATE EFFECTS OF IRRADIATION ON THE THYROID GLAND IN MICE. I. IRRADIATION OF ADULT MICE. *(E.)* Walinder, G. (Radiation Biol. Division, Res. Inst. Natl. Defence, Sundbyberg, Sweden). *Acta Radiol* [*Ther*] *(Stockh)* 11(5):433-451, 1972.

1922 LATE EFFECTS OF IRRADIATION ON THE THYROID GLAND IN MICE. II. IRRADIATION OF MOUSE FOETUSES. *(E.)* Walinder, G. (Radiation Biol. Division, Res. Inst. Natl. Defence, Sundbyberg, Sweden) and A.-M. Sjödén. *Acta Radiol* [*Ther*] *(Stockh)* 11(6): 577-589, 1972.

1923 THE SITES OF RADIATION INDUCED-BREAKAGE IN HUMAN LYMPHOCYTE CHROMOSOMES, DETERMINED BY QUINACRINE FLUORESCENCE. *(E.)* Roman, C. S. (Med. Res. Council, Population Genetics Unit, Headington, Oxford, England) and M. Bobrow. *Mutat Res* 18(3): 325-331, 1973.

1924 PROLIFERATIVE ACTIVITY OF GASTRIC MUCOSA IN RATS AFTER ORAL ADMINISTRATION OF RADON WATER AT VARIOUS POSTRESECTION PERIODS. *(E.)* Zaporozhchenko, I. G. (Inst. Human Morphology, U.S.S.R. Acad. Med. Sci., Moscow). *Biull Eksp Biol Med* (5):89-93, 1973.

See also:

* (Viral): 1981
* (Path): 2141

1925 INFECTIVITY AND RNA PATTERNS AS FUNCTIONS OF HIGH- AND LOW-DILUTION PASSAGE OF MURINE SARCOMA-LEUKEMIA VIRUS: EVIDENCE FOR AUTO-INTERFERENCE WITHIN AN ONCORNAVIRUS POPULATION. *(E.)* Bondurant, M. C. (Sch. Publ. Hlth., U. California, Berkeley), A. J. Hackett and F. L. Schaffer. *J Virol* 11(5):641-647, 1973.

Evidence is presented for autointerference in the Moloney murine sarcoma-leukemia virus [MSV(MLV)] complex. Heterogeneity of buoyant density and RNA content of virions of MSV(MLV) was the result of passage at low dilution. Heterogeneous stocks revealed two major RNA components in the population, with the smaller component, apparent mol wt 4 X 10^6 to 5 X 10^6, becoming predominant upon serial passage at low dilution. Concomitantly, infectivity titers of both MLV and MSV decreased upon serial passage at low dilution. MSV(MLV) passaged at high dilution retained high titers and a rather homogeneous high-molecular-weight RNA population characteristic of high-buoyant-density virions. Interference of both MLV and MSV replication was demonstrated by employing mixed inocula containing both low- and high-dilution passage stocks of MSV(MLV). In contrast to results with MSV(MLV), MLV freed of MSV by limit dilution did not show heterogeneity of buoyant density or of RNA when propagated at low dilution.

1926 QUANTITATIVE DETERMINATION AND LOCATION OF NEWLY SYNTHESIZED VIRUS-SPECIFIC RIBONUCLEIC ACID IN CHICKEN CELLS INFECTED WITH ROUS SARCOMA VIRUS. *(E.)* Parsons, J. T. (Molecular Biol. Inst., Zurich, Switzerland), J. M. Coffin, R. K. Haroz, P. A. Bromley and C. Weissmann. *J Virol* 11(5):761-774, 1973.

A sensitive and quantitative nucleic acid hybridization assay for the detection of radioactively labeled avian tumor virus-specific RNA in infected chicken cells has been developed. The fact that DNA synthesized by virions of avian myeloblastosis virus in the presence of actinomycin D (AMV DNA) is complementary to at least 35% of the sequences of 70*S* RNA from the Schmidt-Ruppin strain (SRV) of Rous sarcoma virus was used in these studies. Annealing of radioactive RNA (either SRV RNA or RNA extensively purified from SRV-infected chicken cells) with AMV DNA followed by ribonuclease digestion and Sephadex chromatography yielded products which were characterized as avian tumor virus-specific RNA-DNA hybrids by hybridization competition with unlabeled 70*S* AMV RNA, equilibrium density-gradient centrifugation in Cs_2SO_4 gradients, and by analysis of their ribonucleotide composition. The amount of viral RNA synthesized during pulse labeling with ^{3}H-uridine could be quantitated by the addition of an internal standard consisting of ^{32}P-labeled SRV RNA prior to purification and hybridization. This quantitative assay was used to determine that, in SRV-infected chicken cells labeled for increasing lengths of time with ^{3}H-uridine, labeled viral RNA appeared first in a nuclear fraction, then in a cytoplasmic fraction, and still later in mature virions. This observation is consistent with the hypothesis that RNA tumor virus RNA is synthesized in the nucleus of infected cells.

1927 SATELLITE DNA REPLICATION IN BABY MOUSE KIDNEY CELLS INFECTED WITH POLYOMA VIRUS. *(E.)* Hatfield, J. M. R. (Dept. Zool., U. Edinburgh, Scotland) and P. M. B. Walker. *Nature* [*New Biol*] 242(118):141-142, 1973.

Incorporation of ^{3}H-thymidine into DNA of polyoma virus-infected baby mouse kidney (BMK) cells was studied. There was no difference between confluent infected and mock-infected cultures with respect to ^{3}H-thymidine incorporation for the first 12 hr. Between 12-16 hr after infection, infected cultures showed increased incorporation. Pulse labels between 16-28 hr and 28-36 hr after infection revealed greatly increased incorporation compared with controls. Autoradiography also showed a dramatic increase in the percentage of labeled cells from 16 hr after infection. Repeated experiments, however, failed to reveal preferential replication of light-satellite DNA between 12-16 hr after infection as had been previously reported by others.

1928 INFLUENCE OF ADENO-ASSOCIATED SATELLITE VIRUS ON ADENOVIRUS-INDUCED TUMOURS IN HAMSTERS. *(E.)* Mayor, H. D. (Baylor Coll. Med., Houston, Texas), G. S. Houlditch and D. M. Mumford. *Nature* [*New Biol*] 241(106):44-46, 1973.

The effect of preinfection of newborn hamsters by adeno-associated satellite virus (ASV) on tumorigenesis induced by inoculation of the same animals one day later with human adenovirus type 31 was studied. Animals inoculated with ASV developed no tumors compared with a 50% tumor incidence in hamsters inoculated only with adenovirus type 31. ASV protection appeared to be specific as animals inoculated with ASV followed by SV40 developed the same number of tumors as those inoculated only with SV40. Inoculation of pregnant females with ASV 1 wk before littering afforded no protection to male offspring subsequently infected with type 31 virus as neonates and markedly enhanced tumorigenesis in the female offsprin

1929 ISOLATION OF HUMAN AMNION CELLS TRANSFORMED BY RESCUABLE MURINE SARCOMA VIRUS. *(E.)* Peebles, P. T. (Natl. Cancer Inst., Bethesda, Md.), P. J. Fischinger, R. H. Bassin and A. G. Papageorge. *Nature* [*New Biol*] 242(117):98-101, 1973.

Human F-49-1 amnion cells were transformed by a feline leukemia virus (FeLV) pseudotype of the Moloney murine sarcoma virus (MSV (FeLV)) rescued from sarcoma virus-positive leukemia virus-negative (S+L-) mouse cells. Unlike F-49-1 cells, the transformed cells (F-147) were pleomorphic and spindle-shaped, lacked contact inhibition and grew to high cell densities. MSV could only be rescued from F-147 cells following superinfection with FeLV or RD-114 virus. Such rescued virus possessed focus-forming activity on feline embryo fibroblasts and human cells but could not transform mouse 3T3FL or rat NRK cells. The host range of the MSV pseudotypes rescued from S+L- human amnion cells was apparently determined by the superinfecting leukemogenic helper virus. The rescued MSC pseudotypes acquired their envelope-

ociated host range and ability to interfere with
114 superinfection from the superinfecting helper
us. Immunodiffusion and complement fixation tests
ealed the presence of murine virus group-specific
igens and were consistent with the presence of the
ine S+L- type genome in human F-147 cells in the
ence of any detectable replicating FeLV.

0 C-TYPE VIRUS PARTICLES IN PLACENTAS AND FETAL TISSUES OF RHESUS MONKEYS. *(E.)*

idlovsky, G. (Pfizer Inc., Maywood, N.J.) and
Ahmed. *J Natl Cancer Inst* 51(1):225-233, 1973.

racellular C-type virus particles were detected
electron microscopy on undifferentiated mesenchy-
cells and syncytial trophoblasts of chorionic
li in each of four normal rhesus monkey placentas
aca mulatta) examined. Viral buds were also seen
erythroblasts and erythrocytes within fetal spleen
kidney tissues as well as on smooth muscle cells
hin the umbilical cord of associated embryos
-73 days). The virus particles appeared more
erous in placental than in embryonic tissue.

1 RIBONUCLEASES OF ROUS SARCOMA VIRUS. *(E.)*

Hung, P. P. (Dept. Virology, Abbott Lab.,
th Chicago, Ill.). *Virology* 51(2):287-296, 1973.

an attempt to study the origins of ribonucleases,
ribonuclease activities found in uninfected chick
ryo fibroblasts, in Rous sarcoma virus-infected
ls, and in the purified virus were compared.
ified Rous sarcoma virus and avian leukosis
us preparations contain ribonucleases which hy-
lyze polyuridylic acid (Poly U), polyadenylic
d (Poly A) and the double-stranded hybrid of
y U and Poly A (Poly A:Poly U). The ribonucleases
ome soluble when the virus is dissociated with a
tral detergent (Brij 35), mercaptoethanol, and
a. Carboxymethyl cellulose chromatography par-
lly separates the ribonucleases into two activity
ks; one for Poly U and the other for Poly A
Poly A:Poly U. The chromatographed fractions are
ther purified by gel filtration yielding approxi-
e molecular weights of 13,000 for the enzymes.
partially purified enzyme preparations also digest
viral RNA in disrupted virions, but not the viral
in the disrupted virions synthesized by reverse
nscriptase of the virus. The intact virus in sol-
on shows a very high ribonuclease activity to-
d Poly U, but not toward Poly A or Poly A:Poly U.
viral RNA in the intact virus is stable during
rolonged incubation in spite of the fact that the
us contains the ribonucleases capable of digesting
RNA. Moreover, the viral RNA in the disrupted
ions is also stable when incubated alone, although
becomes digested when the viral enzyme preparation
added. These observations suggest that the ri-
uclease which prefers Poly U is located on the sur-
e, and that the other ribonucleases which prefer
y A and Poly A:Poly U reside between inner and
er membranes of the virion. The origin of the
onucleases in Rous virus is not clear. From chick embryo fibroblasts, ribonucleases for Poly U, Poly A and Poly A:Poly U can be isolated with carboxymethyl cellulose chromatography. The ribonuclease activity for the double-stranded RNA is low in these preparations. After transformation by Rous virus, the chick cells demonstrate all these ribonuclease activities with a considerable increase in the activity for Poly A:Poly U. The fact that the enzyme activities for Poly A and for Poly A:Poly U in the transformed cells and purified Rous virus were similar and markedly higher than those from the uninfected cells suggests a possible encapsulation of ribonucleases of host origin into mature virions. The question of whether these ribonucleases are coded by the virus or induced by the virus from the genome of host cells remains unanswered.

1932 SIMIAN VIRUS 40 RAPIDLY LOWERS cAMP LEVELS IN MOUSE CELLS. *(E.)*

Rein, A. (Litton Bionetics, Inc., Bethesda, Md.), R. A. Carchman, G. S. Johnson and I. Pastan. *Biochem Biophys Res Comm* 52(3):899-904, 1973.

Cyclic AMP (cAMP) levels were assayed in contact-inhibited Balb/3T3 cultures following SV40 infection. The cAMP levels dropped by approximately two-fold within a few hr after infection, reaching a minimum at three hr. cAMP levels returned to normal by 8-12 hr after infection. No significant change in cAMP levels was observed in cells which were mock-infected or infected with heat-inactivated SV40. These results are consistent with a mechanism by which SV40-induced nuclear DNA synthesis occurs via lowered cellular cAMP levels.

1933 FURTHER INVESTIGATIONS ON THE BEHAVIOUR OF CULTIVATED SALIVARY GLAND ADENOCARCINOMA OF THE MOUSE. *(E.)*

Castelli, L. (Regina Elena Inst. Cancer Res., Rome, Italy), M. L. Marcante and A. Caputo. *Z Krebsforsch* 79(4):224-233, 1973.

The oncogenicity of the cultivated cells of C3H/He mouse salivary gland adenocarcinoma is strictly correlated with the presence of the viral particles (Bittner) without any associated action of hormones. From both a quantitative and a qualitative standpoint the growth of the implanted tumor is similar to that observed *in vivo* and completely different from the mammary gland tumor which is also produced by Bittner virus (BV). After 180 days the viral particles disappear from the cell and thereafter the cells are no longer oncogenic when implanted in isogeneic hosts. The early phases of culture are characterized by the epithelial feature of the tumor with a pseudotubular pattern. This feature is not modified with aging of the cultures, however a pseudo-fibroblastic transformation has occasionally been observed in very old samples. This step of evolution is characterized by "eosinophilic degeneration". In the evolution of *in vitro*-tumor a relationship appears to exist between the disappearance of virus particles, the loss of oncogenicity and the presence of eosinophilic bodies. From the data reported in this paper, the salivary gland adenocarcinoma appears to provide a suitable model for studying BV replication since the oncogenic capacity of the virus is not hormone dependent.

1934 ANEMIA VIRUS AS A DISTINCT COMPONENT OF THE MURINE LEUKEMIA-SARCOMA COMPLEX OF VIRUSES. *(E.)* Siegler, R. (Children's Cancer Res. Fdn., Boston, Mass.), I. Lane, S. Moran and P. Leavitt. *Cancer Res* 33(8):1858-1861, 1973.

The severe infectious anemia in rats previously associated with stocks of Abelson virus, which induces stem-cell leukemia, is demonstrated to be due to an infectious, replicating particle the size and density of a virus. This anemia virus is separable from Abelson virus by passage in rats and is probably a member of the leukemia-sarcoma complex of murine viruses.

1935 MUTANTS OF NONPRODUCER CELL LINES TRANSFORMED BY MURINE SARCOMA VIRUSES. *(E.)* Hatanaka, M. (Flow Lab., Inc., Rockville, Md.), R. Klein, R. Toni, J. Walker and R. Gilden. *J Exp Med* 138(2):356-363, 1973.

A variety of cell mutants were obtained by a single 5'-bromodeoxyuridine treatment of a nonproducer cell line transformed by the Kirsten strain of murine sarcoma virus (Ki-MSV). Isolation procedures of these cell mutants are described. The cell mutants obtained were classified by tumorigenic potential and shedding of Type C virus particles into four groups: 1) tumorigenic, without particles; 2) tumorigenic, with Type C particles; 3) nontumorigenic, without particles; and 4) nontumorigenic, with Type C particles. The tumorigenic cell lines showed variability in morphology with both flat and typical transformed appearing cell lines showing equal transplantability. The oncogenic properties remained constant through multiple passages over a six month period.

1936 PRODUCTION AND PURIFICATION OF LARGE AMOUNTS OF ROUS SARCOMA VIRUS. *(E.)* Smith, R. E. (Duke U. Med. Ctr., Durham, N.C.) and E. H. Bernstein. *Appl Microbiol* 25(3):346-353, 1973.

Procedures are described for production and purification of large amounts of Rous sarcoma virus. The virus was produced by Rous sarcoma virus-transformed chicken embryo fibroblasts in roller culture which produced up to 6 mg of virus per day per liter of supernatant fluid. Various methods of concentrating virus were evaluated, including pelleting, ammonium sulfate precipitation, and polyethylene glycol precipitation. Pelleting yielded the best results in terms of recovery of infectious virus. Purification was achieved by means of successive velocity and equilibrium density centrifugation by using sucrose solutions made in low-salt buffer. The purified virus could be stored at -85 C in TE buffer [0.005 M tris(hydroxymethyl)aminomethane (Tris)-hydrochloride, 0.001 M ethylenediaminetetraacetic acid] with the addition of calf serum to a final concentration of 10%. A rapid method for the optical density measurement of virus concentration was also developed.

1937 ISOLATION OF AN RNA VIRUS FROM TRANSITIONAL CELL TUMORS OF THE HUMAN URINARY BLADDER. *(E.)* Elliott, A. Y. (Dept. Surg., U. Minnesota, Minneapolis), E. E. Fraley, A. E. Castro, P. Cleveland, T. Hakala and N. Stein. *Surgery* 74(1):46-50, 1973.

An RNA virus (temporary designated EFMU) was isolated from tumors of the human urinary bladder and its properties examined. This virus was linked to the original tumors by morphological, virological, and immunological techniques. The EFMU closely resembles known RNA tumor viruses with respect to size, buoyant density, morphology, and also because it has an RNA-directed DNA polymerase. The role of EFMU in the pathogenesis of human transitional cell tumors is not clear.

1938 HORIZONTAL TRANSMISSION OF FELINE LEUKAEMI VIRUS. *(E.)* Hardy, W. D., Jr. (Memorial Sloan-Kettering Cancer Center, New York, N.Y.), L. J Old, P. W. Hess, M. Essex and S. Cotter. *Nature* 244(5414):266-269, 1973.

Horizontal transmission (viral spread by contact or contagion) of feline leukaemia virus (FeLV) is discussed. Cats infected in this manner run a high ris of developing lymphosarcoma. Serological studies show that FeLV is present in 90% of cats with feline lymphosarcoma. Peripheral blood smears from 543 apparently normal cats were tested for FeLV by indirect immunofluorescence using absorbed rabbit antiserum to FeLV group specific antigens. Of these animals, 33% were infected with FeLV. Normal cats in households with two or more cats with lymphosarcoma were twice as likely to be infected with FeLV than those from households with only one cat with lymphosarcoma or cats with other FeLV-associated diseases. Of 148 FeLV$^+$ normal cats, 16.2% developed lymphosarcoma in an average time of 5.3 months. A total of 23.7% of all FeLV$^+$ normal cats developed an FeLV-related disease. FeLV transmission, by saliva, urine, and blood is considered. The importance of breaking the cycle of horizontal transmission by vaccination or by selective segregation of FeLV$^+$ cats is emphasized.

1939 SIALIDASE ACTIVITY IN TRANSFORMED CELLS. *(E.)* Schengrund, C. L. (Milton S. Hershey Med. Ctr., Hershey, Pa.), R. N. Lausch and A. Rosenberg. *J Biol Chem* 248(12):4424-4428, 1973.

Sialidase activity, ganglioside patterns, and total cellular sialic acid content were determined for several normal and virus- and spontaneously transformed hamster cell lines and for lines derived from virus-induced hamster tumors. The total cellular sialic acid content showed no significant change with transformation, varying from 2.3 to 3.8 μg/mg protein Chromatographic analysis indicated that the predominant ganglioside was Gm$_3$ in normal and transformed

es except for one tumor-derived line (Ad12). Gm_1
the major ganglioside in Ad12. Sialidase acti-
y toward added extracellular disialo- and trisialo-
glioside substrate was present in all transformed
l lines studied. No measurable activity toward
ed substrate was found in cell-free preparations
untransformed hamster embryo fibroblasts, baby
ster kidney fibroblasts, or adult hamster kidney
roblasts. These results indicated that tumori-
ic transformation is accompanied by the appearance
sialidase activity against extracellular lipid-
nd substrate.

0 STRUCTURAL PROTEINS OF ONCOGENIC RIBONU-
CLEIC ACID VIRUSES. *INTERSPEC* II, A NEW
ERSPECIES ANTIGEN. *(E.)* Strand, M. (Albert
stein Coll. Med., Bronx, N.Y.) and J. T. August.
iol Chem 248(16):5627-5633, 1973.

Rauscher murine leukemia virus polypeptides with
arent molecular weights of 69,000 and 71,000 have
n purified and characterized. These polypeptides
tained a previously undescribed interspecies
igenic determinant of mammalian oncogenic C type
viruses (*interspec* II), as was demonstrated by
reaction of the murine antigen with anti-feline
kemia virus serum. Radioimmunoassay analysis
wed that both polypeptides were precipitated by
anti-feline virus serum, indicating that they
h contained the interspecies determinant or that
y were closely associated and coprecipitated.
interspec II antigen was distinguished from the
wn gs-3 interspecies antigen (*interspec* I) and
virus RNA-dependent DNA polymerase by protein
ification, physical properties, and immunological
lysis. The new antigen was not detected by com-
ition radioimmunoassay in uninfected mouse cells or
se cells productively infected with vesicular
matitis virus.

1 MEMBRANE CHANGES AND ADENOSINE TRIPHOS-
PHATE CONTENT IN NORMAL AND MALIGNANT
NSFORMED CELLS. *(E.)* Vlodavsky, I. (Dept.
etics, Weizmann Inst. Sci., Rehovot, Israel),
Inbar and L. Sachs. *Proc Natl Acad Sci USA*
6):1780-1784, 1973.

dies were undertaken to determine the role of
rgy-generating systems in the control of membrane
nges required for agglutination by concanavalin
Con A) and the difference between the control in
mal and transformed hamster cells. The increased
lutinability of high-density polyoma- or SV40-
nsformed cells was associated with a decreased
racellular ATP concentration as determined by
luciferin-luciferase method. The increased
lutinability could be reversed by growing the
ls in medium with glucose which increased in-
cellular ATP. Nonagglutinating transformed
ls could be induced to aggregate in the presence
Con A by exposure to inhibitors of glycolysis,
ctron transfer, or oxidative phosphorylation.
cose could prevent agglutination in the presence
electron transfer and oxidative phosphorylation
ibitors but not in the presence of inhibitors of
glycolysis. Normal cells seeded at low density showed no agglutination by Con A even though intracellular ATP levels were decreased by 90%. Depletion of ATP stores in high-density normal cell cultures resulted in agglutination which was not as extensive as that seen in high-density transformed cell cultures. Binding experiments with radioactively labeled Con A showed that similar numbers of Con A molecules bound to the membranes of normal and transformed agglutinating and nonagglutinating cells.

1942 EARLY DNA-RNA COMPLEX FROM THE ENDOGENOUS RNA-DIRECTED DNA POLYMERASE ACTIVITY OF UNINFECTED CHICKEN EMBRYOS. *(E.)* Kang, C.-Y. (McArdle Lab. Cancer Res., U. Wisconsin, Madison) and H. M. Temin. *Nature* [*New Biol*] 242(120):206-208, 1973.

A DNA-RNA complex has been isolated along with endogenous RNA-directed DNA polymerase activity from uninfected chicken embryos. DNA synthesized for 5 min in the presence of actinomycin D sedimented in linear sucrose gradients at 6S and 30S. The 30S complex was sensitive to RNase, alkali, and heat. Cs_2SO_4 equilibrium density centrifugation studies of a 5 min product of chicken endogenous DNA polymerase activity made in the presence of actinomycin D and of the same product pretreated with single-strand-specific S1 nuclease suggested that the early product contained RNA-DNA hybrid, some RNA covalently bound to DNA, and some double-stranded DNA. The complex thus presumably contained an RNA template and, possibly, an RNA primer.

1943 LYMPHOMA INDUCTION AND VIRAL ISOLATION FROM OWL MONKEY *(AOTUS TRIVIRGATUS)* INOCULATED WITH LYMPHOMA CELLS INDUCED BY *HERPERSVIRUS SAIMIRI*. *(E.)* Loeb, W. F. (Litton Bionetics, Inc., Kensington, Md.), M. G. Valerio, D. V. Ablashi and G. R. Armstrong. *Amer J Vet Res* 34(5):677-680, 1973.

The effect of whole-cell inoculation of neoplastic cells into a susceptible monkey was studied using an adult, male owl monkey weighing 1000 g. Lymphoma cells were harvested from the fused sublumbar and pelvic lymph nodes of another owl monkey *(Aotus trivirgatus)* inoculated with *Herpesvirus saimiri* (HVS). Free complete virus could not be demonstrated in this material by electron microscopy or by inoculation of cell-free extract, including clarified cell lysate, on susceptible cells. Cultivation of the lymphoma cells or cocultivation with susceptible cells was required to rescue the virus *in vitro*. The adult, male owl monkey was inoculated i.p. and i.m. with the lymphoma cells and developed a lymphoma involving the mesentery and intestine within a 9½-month period. Supravital studies of the peripheral blood from the inoculated monkey demonstrated actively motile lymphocytes in the late phase of the disease despite the absence of frank leukemia. *Herpesvirus saimiri* could not be isolated from blood samples collected during the first six months; however, HVS was isolated from blood samples collected during and after the 7th month and from mesenteric lymph nodes obtained at necropsy.

Thus, a phenomenon analogous to *in vitro* rescue of virus seems to have occurred *in vivo*. Because both the donor and recipient monkeys were male, karyotyping was not used to determine whether the neoplasm was of donor or recipient origin. However, its slow rate of development, concurrently with the appearance of virus and antibody, and the absence of neoplasm at inoculation sites indicates that the neoplasm was virally induced and of recipient origin.

1944 QUALITATIVE RESTRICTION OF TRANSCRIPTION IN HUMAN LEUKEMIC LYMPHOCYTES. *(E.)*

Flickinger, R. A., (Dept. Biol. State U., New York, Buffalo), F. M. Roche and L. V. Rodriguez. *Eur J Cancer* 9(5):363-366, 1973.

RNA from human normal lymphocytes and Burkitt lymphoma cells were compared with respect to the number of kinds of hybridizable RNA complementary to normal human DNA, and to the degree of similarity between normal and lymphoma RNA. Both saturation and competition experiments were performed with RNA uniformly labeled *in vitro* with dimethyl-H^3-sulfate and human placental DNA. Normal lymphocytes possess more kinds of RNA that hybridize with normal human DNA than the Burkitt lymphoma cells. Competition experiments revealed that some kinds of hybridizable species of RNA present in normal lymphocytes are not present in the leukemic cells. Although the differences observed in these competition experiments were small, they were detected in each of the three competition experiments. It should be remembered, however, that the recorded qualitative differences may only reflect a quantitative difference in some of the species of RNA that were transcribed in few copies.

1945 HERPES SIMPLEX VIRUS AS A SOURCE OF THYMIDINE KINASE FOR THYMIDINE KINASE-DEFICIENT MOUSE CELLS: SUPPRESSION AND REACTIVATION OF THE VIRAL ENZYME. *(E.)*

Davidson, R. L. (Harvard Med. Sch., Boston, Mass.), S. J. Adelstein and M. N. Oxman. *Proc Natl Acad Sci USA* 70(7):1912-1916, 1973.

The inheritance and expression of viral genetic information was studied using thymidine kinase (EC 2.7.121)-deficient mouse cells which were infected with inactivated herpes simplex virus; the "transformed" cells that produce viral thymidine kinase were then isolated and investigated. Shortly after transformation, the expression of the viral enzyme could be suppressed and reactivated with high efficiency. Suppression refers to the disappearance of enzyme activity and is not meant to imply any mechanism. On continued multiplication in nonselective medium, the proportion of cells producing the viral enzyme decayed exponentially. This decay seemed to represent a change in the expression of the viral gene for thymidine kinase rather than the loss of the gene from the cells, since the viral enzyme could be apparently reactivated in every cell, albeit at a very low frequency. This system appears to provide the first demonstration *in vitro* of a progressive transition of viral genetic information from a state in which it is expressed with a high efficiency to a state in which it is maintained in every cell but not expressed. It is uncertain whether the same mechanisms are involved in the high-frequency suppression and reactivation that occurs soon after transformation and the lengthy decay and low-frequency reactivation that occurs later.

1946 CELL CONTACT-DEPENDENT INCREASE IN MEMBRANE D-GALACTOPYRANOSYL-LIKE RESIDUES ON NORMAL, BUT NOT VIRUS- OR SPONTANEOUSLY-TRANSFORMED, MURINE FIBROBLASTS. *(E.)*

Nicolson, G. L. (Salk Inst. Biol. Studies, San Diego, Calif.) and M. LaCorbiere. *Proc Natl Acad Sci USA* 70(6): 1672-1676, 1973.

Ricinus communis agglutinin (RCA), a lectin specific for β-D-galactopyranosyl-like oligosaccharide residues, was used to study differences in lectin-binding sites on normal 3T3 and SV40-transformed 3T3 (SV3T3) and 3T12 cells grown to different cell densities. RCA agglutinability decreased after contact of SV3T3 cells reached a density where contact was made between cells. Agglutinability also decreased slightly after contact among 3T3 cells on the incubation plate was made, but the decrease was highly variable and occasionally was not observed Transformed cells (SV3T3 and 3T12) showed no density-dependent changes in the number of RCA-binding sites as determined by the extent of binding of ^{125}I-tagged agglutinin. However, confluent 3T3 cells bound 2.5 times more ^{121}I RCA/cell than did growing, sparsely populated 3T3 cells. Growing 3T3 cultures bound five times less ^{125}I-RCA/cell than did growing 3V3T3 or 3T12 cells.

1947 ULTRASTRUCTURAL AND COMPLEMENT FIXATION STUDIES ON SUSPENSION CULTURES DERIVED FROM HUMAN SOLID TUMORS. *(E.)*

Dalton, A. J. (Natl. Cancer Inst., Bethesda, Md.), U. Heine, J. M. Kondratick, D. V. Ablashi and E. A. Blackman. *J Natl Cancer Inst* 50(4):879-894, 1973.

The ultrastructure of cells in a series of 42 individual cell cultures from 55 separate biopsies of human solid tumors is described as are the results of complement-fixation tests on donor serum antibodies and in cell culture antigens. Ten of these cell line developed into suspension cultures at intervals of from five to seven days to three months after the cultures were started. Four of these lines were apparently derived from normal cells present in the stroma of the tumor. The derivation of the cells of the other six isolates is uncertain. In complement-fixation tests of donor serum, five were negative for cytomegalovirus (CMV) antibodies and five were positive for Epstein-Barr virus (EBV) antibodies. In eight of the ten isolates, herpes-type virions were identified by electron microscopy. In the two isolates in which herpes-type virions were not found the donor sera were positive for CMV antibodies and negative for EBV antibodies. The serum of the donor of one isolate in which herpes-type virions were found was anticomplementary. Tubuloreticular modifications of the endoplasmic reticulum were present in some of the cells of all ten isolates. The result

ggest that while EBV is the usual agent responsible
r the development of continous suspension cultures,
other instances CMV and possibly other factors
y be involved. There is no clear-cut evidence in-
cating that the infection of normal cells with EBV
ansforms them into malignant cells.

48 HL-A ANTIGENS OF HUMAN TUMOR-DERIVED
CELL LINES AND VIRAL TRANSFORMED FIBRO-
ASTS IN CULTURE. *(E.)* Kersey, J. H. (Dept. Path.,
diatrics, Lab. Med., U. Minnesota, Minneapolis),
J. Yunis, G. J. Todaro and S. A. Aaronson. *Proc*
c Exp Biol Med 143(2):453-456, 1973.

microtoxicity assay was used to compare HL-A re-
tivity of normal human diploid cells, human tumor
ne cells and cells transformed *in vitro* by on-
genic viruses. Skin punch biopsies were obtained
om individuals of varying racial backgrounds in
stern and Mid-Western U.S. Diploid skin fibro-
asts were transformed by the small plaque mutant
simian virus 40 (SV40) and by the Kirsten strain
murine sarcoma virus. Established human cell
nes included HeLa (human cervix carcinoma), KB
ral carcinoma), HEp-2 (laryngeal carcinoma),
vine-3 (breast carcinoma), and XC-114 (rhabdo-
rcoma). Histocompatibility (HL-A) antigens were
monstrated in 95% of 217 cultured adult human
broblast strains. Four of the eight lines es-
blished from human tumors showed no HL-A reactivi-
. By contrast, no loss of HL-A reactivity was
und in fibroblast strains transformed *in vitro* by
40 or murine sarcoma virus.

49 VIRIONS AND ANTIGENS OF HERPES VIRUS TYPE
2 IN CERVICAL CARCINOMA. *(E.)* Aurelian,
(Johns Hopkins U. Sch. Med., Baltimore, Md.).
ncer Res 33(6):1539-1547, 1973.

rpesvirus type 2 (HSV-2) has been associated with
uamous cancer of the human cervix on the basis of
roepidemiological data. Findings providing in-
rmation on the basis of the association of the
V-2 genome with cervical cancer cells are pre-
nted. A herpesvirus isolated from degenerated
rvical tumor cells grown *in vitro* is identified
a type 2 on the basis of biological and immuno-
gical properties. Surface antigens, HSV-2-speci-
c, are observed in a small proportion of cells
om a secondary culture of cervical tumor cells.
foliated but not biopsied tumor cells from patients
th cervical carcinoma contain HSV-2 antigens, but
rus particles are not observed. Finally, 90%
patients with cervical carcinoma have antibody to
HSV-2-induced early antigen different from neutral-
zing antigens. This antibody, present only in 10%
a matched control group, appears to be of prog-
stic significance. The results are discussed in
rms of the virus-host cell interaction. The
ta suggest that cervical tumor cells harbor the
ral genome in a partially repressed state char-
cterized by the expression of only some viral
nctions. Under conditions of stress, such a high
, complete expression of the viral genome may
cur resulting in the formation of viral com-
nents and/or infectious virus. The possibility
cannot be excluded that the specific cell harboring the virus, and the expression of those viral genes needed to transform cells, are singly or both determined by the genotype of the host.

1950 STRUCTURAL REARRANGEMENT AND SUBUNIT COMPOSITION OF RNA FROM RELEASED SOEHNER-DMOCHOWSKI MURINE SARCOMA VIRIONS. *(E.)* East, J. L. (U. Texas, M. D. Anderson Hosp., Tumor Inst., Houston), P. T. Allen, J. E. Knesek, J. C. Chan, J. M. Bowen and L. Dmochowski. *J Virol* 11(5):709-720, 1973.

A detailed study was undertaken to characterize the sedimentation behavior of native, genomic RNA and RNA subunit species of a murine sarcoma virus. Two types of genomic, high-molecular-weight RNA species were found in Soehner-Dmochowski murine sarcoma virions released from virus-induced rat tumor cells grown in tissue culture. The type of RNA species observed depended on the length of exposure of the tumor cells to radioactive precursor. Early RNA of virions labeled up to 4 hr with radioactive uridine had a sedimentation coefficient of 50*S*, and late RNA of virions labeled for 24 hr had a sedimentation coefficient of 58*S*. Thermal transitions of early and late RNA indicated a difference in the configuration or structure of these two types of RNA. The late RNA may represent either a different configurational state of the early RNA or an aggregate molecule of two early RNA components joined together. Heat dissociation revealed that the major subunit of both RNA types was a 28*S* species, which was not susceptible to degradation by the addition of micrococcal nuclease to virions. A transitional, intermediate RNA species with a sedimentation coefficient of 37 to 40*S* was detected when early RNA was dissociated by dimethyl sulfoxide or heat at temperatures suboptimal for complete conversion. No free RNA subunit components were detected in virions harvested at intervals as short as 30 sec or 5 min. A model for the assembly of genomic RNA from 28*S* RNA subunits is proposed but the process by which these RNA subunits are joined together to form the aggregate genome is not presently known. The possibility is suggested that a portion of the 4*S* RNA found in virions could be involved in the assembly of subunits by forming bridges between the molecules.

1951 PROSPECTIVE STUDIES OF THE ASSOCIATION OF GENITAL HERPES SIMPLEX INFECTION AND CERVICAL ANAPLASIA. *(E.)* Nahmias, A. J. (Emory U. Sch. Med., Atlanta, Ga.), Z. M. Naib, W. E. Josey, E. Franklin and R. Jenkins. *Cancer Res* 33(6): 1491-1497, 1973.

The various problems associated with prospective epidemiological studies, aimed at evaluating the risk of cervical anaplasia in women detected earlier with genital herpetic or herpes simplex virus type 2 (HSV-2) infection are identified. Preliminary results are included of ongoing follow-up studies in 871 women (herpes group) detected serologically, cytologically, or virologically with such infections and in 562 women (control group) in whom HSV-2 antibodies could not be detected serologically. The rate of cervical dysplasia was found to be greater than 2-fold and that of *in situ* carcinoma, 8-fold higher in the herpes group than the rate observed in the control group. There was also a greater than 2-fold higher rate of *in situ* carcinoma in women who were pregnant at the time of diagnosis of their genital herpetic infection, as compared to women in whom the genital herpetic infection was detected during a nonpregnant period. The rate of cervical anaplasia in women identified with a primary HSV-2 infection was less than that found when the infection was not identified as a primary one. These findings are discussed within an herpes simplex virus-cell interaction scheme which takes into account the important consideration of the known recurrences of herpetic cervicitis. These results should be viewed at present as only preliminary; further epidemiological variables, currently being obtained, may influence final interpretation as to causality of the herpesvirus in cervical carcinogenesis. Nevertheless, the results do point to women identified with a genital herpetic or HSV-2 infection as a special population that requires more careful Papanicolaou cervicovaginal screening for cervical cancer.

1952 AN ONCORNAVIRUS ISOLATED FROM HUMAN CANCER CELL LINE.

(E.) Ilyin, K. V. (Acad. Med. Sci., Moscow, USSR), A. F. Bykovsky and V. M. Zhdanov. *Cancer* 32(1):89-96, 1973.

Some morphological, biophysical, biochemical, and immunological properties of a virus isolated from a clone of continuous cell line that was derived from human cancer tissue (HEp-2 cells) were examined. The clone of HEp-2 cells retains human karyotype and has no heterogeneous contamination. Injection of the cells and the virus into mice of C3H/Sn, C3H/H2P, BaLB/c lines and into newborn hamsters did not produce tumors in animals. Intracellular virus possesses morphological characteristics of oncornaviruses type A and B; extracellular virus has the properties of oncornaviruses type B. The virus bands at $\rho = 1.16$ g/ml in sucrose density gradients, and the production of it is inhibited by actinomycin D. The virions contain reverse transcriptase, and the products of reaction are double-stranded DNA and RNA:DNA hybrids. The virus is immunologically distinct from mouse mammary tumor virus. These results lead to the conclusion that the virus isolated may be considered as an oncornavirus of human origin. Another explanation is that the virus was introduced into the culture with calf serum added to the cultural medium. However, the latter possibility is scarcely probable because the comparative study of several continuous human cell lines grown in the medium with calf serum showed that type C particles are associated only with leukemic cells, and type B particles were found in cancer cells.

1953 AN ADENOVIRUS, FAV-1, ISOLATED FROM THE KIDNEY OF A FROG (*RANA PIPIENS*).

(E.) Clark, H. F. (Wistar Inst. Anat. Biol., Philadelphia, Pa.), F. Michalski, K. S. Tweedell, D. Yohn, and R. F. Zeigel. *Virology* 51(2):392-400, 1973.

An adenovirus-like cytopathic agent, designated frog adenovirus 1 (FAV-1), was recovered from turtle (TH-1) cell cultures inoculated with cells of a granuloma-bearing kidney of a leopard frog, *Rana pipiens*. Virus replication, as indicated by cytopathic effect (including intranuclear inclusions) was limited to TH-1 cells; a variety of other cell types from amphibians, reptiles, fish, and mammals, were refractory to infection. The optimal temperature for replication was 30 C. Electron microscopic examination of infected TH-1 cells revealed intranuclear virus particles exhibiting a size and ultrastructure characteristic of adenoviruses. The virus is ether-resistant and a DNA nucleic acid type was inferred from observations of inhibition by 5-bromo-2'-deoxyuridine. The virus does not possess the group-specific, complement-fixing antigen of mammalia adenoviruses, and was not neutralized by hyperimmune antisera to the avian adenoviruses CELO, and GAL types I, II, and III. FAV-1 did not cause tumors when inoculated into the nephrogenic ridge of *R. pipiens* tadpoles. FAV-1 appears to be an "orphan" virus, and the first described adenovirus from a poikilothermic vertebrate host. It has a host range in cell culture, antigenic composition, and temperature requirement for replication that are unique among the members of the adenovirus group.

1954 STUDIES OF NONDEFECTIVE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID VIRUSES. VII. CHARACTERIZATION OF THE SIMIAN VIRUS 40 RNA SPECIES INDUCED BY FIVE NONDEFECTIVE HYBRID VIRUSES.

(E.) Levine, A. S. (Natl. Cancer Inst., Bethesda, Md.), M. J. Levin, M. N. Oxman and A. M. Lewis, Jr. *J Virol* 11(5):672-681, 1973.

Five nondefective adenovirus 2 (Ad2)-simian virus 40 (SV40) hybrid viruses were isolated and found to contain segments of SV40 DNA covalently linked to Ad2 DNA. The quantity of SV40 DNA present is a stable characteristic of each hybrid virus, and varies from less than 5% (in Ad2+ND$_3$) to more than 30% (in Ad2+ND$_4$) of the SV40 genome. The SV40 portions of these hybrids have been characterized by relating the SV40-specific RNA sequences transcribed in cells infected with each hybrid virus to those transcribed in cells infected with each of the other hybrid viruses and with SV40 itself. RNA-DNA hybridization-competition experiments indicate that the number of unique SV40 RNA sequences transcribed in infected cells is proportional to the size of the SV40 DNA segment contained within each hybrid and, in the case of the three hybrids which induce detectable SV-40 specific antigens, to the number of SV40 antigens induced. Furthermore, the SV40-specific RNA sequences transcribed from any one of the hybrids are completely represented in the RNA transcribed from all other hybrids with longer SV40 segments. Thus, the SV40 DNA regions in the five hybrid viruses appear to contain some nucleotide sequences in common. The SV40-specific RNA transcribed from Ad2+ND$_4$, the hybrid containing the largest SV40 segment, is qualitatively similar to the SV40-specific RNA transcribed early (i.e., prior to viral DNA replication) in SV40 lytic infection. Thus, it appears that no significant amount of late SV40 DNA is transcribed during infec-ion by any of the five nondefective Ad2-SV40 hybrid iruses. The presence of all of the early SV40 geneic information within the small segment of SV40 DNA ontained in Ad2+ND$_4$ suggests that the early genes of ild-type SV40 are localized in one portion of the ild-type SV40 DNA molecule.

955 REVERSION IN HYBRIDS BETWEEN SV40-TRANSFORMED HAMSTER AND MOUSE CELLS. *(E.)*

iblin, C. N. (Imperial Cancer Res. Fund Lab., Lonon, England) and I. Macpherson. *Int J Cancer* 12(1): 48-161, 1973.

he expression of the transformed phenotype was tudied in segregants from a hybrid cell line reulting from the fusion of SV40-transformed BHK21 yrian hamster cells (thioguanine resistant) and T3 mouse cells (bromodeoxyuridine resistant). Since he hamster cell parent was sensitive to bromodeoxyridine, segregants lacking some chromosomes could e obtained by "back-selection" in this drug. These egregants retained transformed characteristics, espite substantial loss of hamster chromosomes. elected segregants were treated further with cytooxic antibody produced against the hamster parent. he majority of the segregants isolated retained ll transformed characteristics, even when the amster chromosome complement was greatly reduced. ome derivatives showed properties intermediate etween a transformed and an untransformed state. wo revertant clones were also isolated which lacked ransformed characteristics and contained no etacentric (hamster) chromosomes. Unlike the ther segregants, no SV40 virus could be recovered rom these cells. The results obtained do not allow irm conclusions as to the location or number of ntegration sites for SV40 genomes in TG2 cells. ince all detectable hamster chromosomes must be liminated before loss of T-antigen and reversion of ransformed characteristics occurs, this might imply ultiple sites of insertion, all functional. A olygenic determination of transformed phenotype ould explain the finding that a/s 1-51 and its erivatives contain reduced amounts of T-antigen nd have lower efficiency of plating in agar and/or n plastic. It might be concluded that several virus ene copies are present, and threshold amounts of ene product are required for the full expression f all transformed characteristics. With a lowered ene product content, one or some characteristics re only partially expressed, or not at all.

956 STUDIES OF NONDEFECTIVE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID VIRUSES. V. ISO-ATION OF ADDITIONAL HYBRIDS WHICH DIFFER IN THEIR IMIAN VIRUS 40-SPECIFIC BIOLOGICAL PROPERTIES. *(E.)*

ewis, A. M., Jr. (Natl. Inst. Allergy, Infectious is., Bethesda, Md.), A. S. Levine, C. S. Crumpacker, . J. Levin, R. J. Samaha and P. H. Henry. *J Virol* 1(5):655-664, 1973.

our new nondefective adenovirus 2 (Ad2)-simian virus 0 (SV40) hybrid viruses have been isolated. Although hese viruses (designated Ad2+ND$_2$, Ad2+ND$_3$, Ad2+ND$_4$, nd Ad2+ND$_5$) were clonal derivatives of the same d2-SV40 hybrid population, they differ significantly from each other and from the previously isolated nondefective hybrid, Ad2+ND$_1$, in their biological properties or in the amount of SV40-specific RNA induced during lytic infection. Like Ad2+ND$_1$, Ad2+ND$_2$ and Ad2+ND$_4$ pass serially in both human embryonic kidney (HEK) and primary African green monkey kidney cells. In contrast, Ad2+ND$_3$ and Ad2+ND$_5$ pass serially only in HEK cells. Ad2+ND$_2$ is like Ad2+ND$_1$ in that it induces the SV40 U antigen, but no SV40 T antigen; however, in contrast to the perinuclear SV40 antigen induced by Ad2+ND$_1$, the SV40 antigen by Ad2+ND$_2$ is located peripherally in the cytoplasm as well as in the perinuclear region of infected cells. Ad2+ND$_4$ induces both the SV40 T and U antigens. Ad2+ND$_3$ and Ad2+ND$_5$ do not induce serologically detectable SV40 antigens and are distinguished from each other on the basis of the relative quantities of SV40-specific RNA which they induce. The induction of different SV40-specific functions suggests the incorporation of different segments of SV40 DNA within the genomes of the respective hybrid viruses.

1957 A TEMPERATURE-SENSITIVE MUTANT OF ROUS SARCOMA VIRUS THAT IS DEFECTIVE FOR REPLICATION. *(E.)*

Friis, R. R. (U. Southern California Sch. Med., Los Angeles) and E. Hunter. *Virology* 53(2):479-483, 1973.

The isolation and properties of *ts* 672, a temperature sensitive mutant of the Prague strain of Rous sarcoma virus which fails to replicate infectious progeny at 41 C, are described. Chick embryo cells infected with *ts* 672 are transformed, as may be seen by focus formation and increased rate of 2-deoxyglucose transport. *ts* 672 is defective for some function necessary to the maturation of infectious progeny. This function is complemented by nontransforming viruses such as the Rous associated viruses. *ts* 672-infected cells produce viral type-specific antigen which is recognized by antiserum specific to subgroup A, and which is effective in establishing interference against other viruses of subgroup A. The infected cells also produce physical particles that incorporate ^{3}H-uridine and cosediment with avian tumor viruses on sucrose density gradients in large numbers.

1958 A STUDY OF THE RELATIONSHIP OF RETICULOENDOTHELIOSIS VIRUS TO THE AVIAN LEUKOSIS-SARCOMA COMPLEX OF VIRUSES. *(E.)*

Halpern, M. S. (U. Southern California Med. Sch. Los Angeles), E. Wade, E. Rucker, K. L. Baxter-Gabbard, A. S. Levine and R. R. Friis. *Virology* 53(2):287-299, 1973.

The results of a study to make a direct comparison between reticuloendotheliosis virus (REV) and the avian leukosis-sarcoma complex of viruses (ALSV) for certain characteristic properties of the RNA tumor viruses are described. Stocks of REV contained no virus which produced interference with any subgroup of ALSV. REV was unable to complement various strains of Rous sarcoma virus (RSV) as measured by the absence of broadening of host range, "helper" virus activity with the defective Bryan high titer strain of RSV, or the induction of release of RAV-60 from chick embryo cells containing the chick helper factor. Hence, no biological interaction has been detected between REV and the avian leukosis-sarcoma complex of viruses. The RNA structure of the REV was studied using glycerol velocity gradients and polyacrylamide gel electrophoresis. The RNA of REV was distinctly like that of RAV-49, a subgroup C avian leukosis virus. The native RNA of REV recovered after phenol extraction cosedimented with the RNA of RAV-49, and after heat denaturation, both RNAs showed the reduced sedimentation velocity and increased rate of migration in polyacrylamide gel which is characteristic of the tumor virus RNAs. Proteins of REV were examined on SDS discontinuous gel electrophoresis. Five major proteins were detected, two of which were glycoproteins. Coelectrophoresis of REV with avian sarcoma virus B_{77} showed no polypeptides that were identical in electrophoretic mobilities. In addition, lactoperoxidase catalyzed iodination was employed to selectively label the surface proteins of REV. Two surface proteins were detected; these corresponded to the two viral glycoproteins.

1959 HUMAN EMBRYONIC LUNG CELLS ABORTIVELY INFECTED WITH HERPES VIRUS HOMINIS TYPE 2 SHOW SOME PROPERTIES OF CELL TRANSFORMATION. *(E.)* Darai, G. (Inst. Med. Virology, U. Heidelberg, W. Germany) and K. Munk. *Nature* [*New Biol*] 241(113): 268-269, 1973.

The properties of human embryonic lung cells abortively infected with Herpes virus Hominis type 2 (HVH-2) were studied. Infection was aborted by incubation of the cells at 42 C for eight days followed by maintenance at 37 C. Infected cell cultures began to show syncytium formation (i.e., morphological transformation) after the seventh passage. They had a longer *in vitro* life than noninfected cultures and were, to an extent, resistant to superinfection with HVH-2. Two different passages (37 and 38) spontaneously showed a cytopathic effect and started to produce HVH-2 suggesting that the viral genome had become integrated into the host cell genome. HVH-2 specific antigens were demonstrable by direct immunofluorescence in the cytoplasm of the abortively infected cells.

1960 INHIBITION IN THE JOINING OF DNA INTERMEDIATES TO GROWING SIMIAN VIRUS 40 CHAINS. *(E.)* Salzman, N. P. (Natl. Inst. Hlth., Bethesda, Md.) and M. M. Thoren. *J Virol* 11(5):721-729, 1973.

Viral DNA synthesis was inhibited for 1 hr by the addition of 5-fluorodeoxyuridine (FUdR) to simian virus 40 (SV40)-infected cultures at 28 to 30 hr postinfection. The subsequent addition of ^{3}H-thymidine to the inhibited cultures reverses the effect of the inhibitor, and during a 1-min labeling period there is rapid synthesis of SV40 DNA. By alkaline sedimentation analysis, it is observed that in FUdR-treated cultures there is synthesis of 4*S* SV40 DNA intermediates but there is a block in the joining of these intermediates to growing SV40 chain cultures. In addition to 4*S* fragments that are associated with replicating SV40 molecules, there is accumulation of SV40 DNA in the 6 to 8*S* region which is observed in neutral sucrose gradients. In an inhibited culture that is pulsed for 1 min with ^{3}H-thymidine and then chased for 10 min, accumulation of a Component II (Comp. II)-like material is observed. This Comp. II has the same neutral sedimentation characteristics and yields the same R_1 restriction endonuclease product as does authentic Comp. II. However, in alkali it is seen that it is composed of fragmented SV40 DNA. The basis for the failure of 4*S* fragments to join to growing SV40 chains is discussed. A model in which there is a requirement for two DNA polymerases and a ligase to permit SV40 DNA chain growth is proposed which is consistent with the data presented.

1961 RELATIONSHIP OF RETICULOENDOTHELIOSIS VIRUS TO THE AVIAN TUMOR VIRUSES: NUCLEIC ACID AND POLYPEPTIDE COMPOSITION. *(E.)* Maldonado, R. L. (Dept. Microbiol., U. Texas, Austin) and H. R. Bose. *J Virol* 11(5):741-747, 1973.

The RNA content and polypeptide composition of reticuloendotheliosis virus (REV) was compared to that of C-type RNA tumor viruses. Two RNA species with approximate sedimentation values of 64*S* and 4*S* were observed after sucrose gradient centrifugation of RNA extracted from purified REV. The high-molecular-weight RNA species of REV sedimented slightly faster than that of the Bryan strain of Rous sarcoma virus (RSV). Although these characteristics were consistent with those of other C-type RNA tumor viruses, significant differences were observed when the polypeptide composition of REV was compared with that of RSV possessing envelope determinants of Rous-associated virus RAV-2 and RAV-3. Five polypeptides of which two were glycosylated were resolved by polyacrylamide gel electrophoresis. The major nonglycosylated polypeptide of REV did not comigrate with that of RSV (RAV-2)-RSV(RAV-3). The majority of the group-specific antigen reactivity resides in this major nonglycosylated polypeptide of avian tumor viruses and comigrates when proteins of several avian tumor viruses are subjected to coelectrophoresis. This difference in the migration of the major polypeptide of REV and RSV(RAV-2)-RSV(RAV-3) may explain the absence of avian tumor virus group-specific antigen in REV. Unlike these other avian RNA tumor viruses, REV was isolated from a turkey rather than a chicken and probably posseses its own group-specific antigen.

1962 A COMPARATIVE EVALUATION OF THE DISTRIBUTION OF CONCANAVALIN A-BINDING SITES ON THE SURFACES OF NORMAL, VIRALLY-TRANSFORMED, AND PROTEASE-TREATED FIBROBLASTS. *(E.)* Rosenblith, J. Z. (Harvard Med. Sch., Boston, Mass.), T. E. Ukena H. H. Yin, R. D. Berlin, and M. J. Karnovsky. *Proc Natl Acad Sci USA* 70(6):1625-1629, 1973.

The topographical distributions of concanavalin A-binding sites on the surfaces of 3T3, protease-treated 3T3, and simian virus 40-transformed 3T3 cultured mouse fibroblasts appear to be different, as shown by a shadow-cast replica technique using concanavalin A (Con A) and a hemocyanin marker. However, chemical fixation of cells before labeling

h Con A and hemocyanin, or labeling exclusively
4 C, allows one to distinguish between inherent
ncanavalin A-binding site topography and potential
arrangement of sites induced by the action of the
ltivalent Con A molecule itself. The *inherent*
stribution of binding sites on 3T3, protease-
eated 3T3, and transformed cells is actually the
me on all cells, i.e., dispersed and random.
eatment of unfixed transformed or protease-
eated 3T3 cells, but not normal 3T3 cells, with
n A and hemocyanin at 37 C (or at 4 C with subse-
ent warming to 37 C), however, results in cluster-
g of binding sites, presumably due to crosslinking
neighboring lectin-binding sites by the
adrivalent Con A. Thus, the underlying difference
tween Con A-binding sites on normal as compared
th transformed or protease-treated normal cells
es not in the inherent topography of binding
tes, but rather in the susceptibility of the sites
aggregation by Con A. The later may reflect an
creased mobility of lectin-binding sites on trans-
rmed or protease-treated cells. An increased
uidity of the membrane could account for the
ility of the multivalent Con A to draw binding
tes into clusters.

53 THE INHIBITION OF THE AVIAN MYELOBLASTOSIS VIRUS DNA POLYMERASE BY POLY(U)
ACTIONS OF VARYING CHAIN LENGTH. *(E.)* Erickson,
J. (Molecular Biol. Dept., Miles Lab., Inc.,
khart, Ind.), B. Janik and R. G. Sommer.
ochem Biophys Res Commun 52(4):1475-1482, 1973.

e inhibitory activity of differing chain lengths
poly(U) toward purified avian myeloblastosis
rus (AMV) RNA-dependent DNA polymerase was studied
vitro by measuring the polymerization of dGMP.
nger lengths of poly(U) were significantly better
hibitors than shorter chain lengths. The inhi-
tory effect dropped sharply at chain lengths be-
w approximately 200 nucleotide residues/poly(U)
lecule. Oligonucleotides ($n < 20$) were not inhi-
tory. These data suggest that poly(U) is not
simple competetive inhibitor of AMV polymerase
previously shown for Rauscher murine leukemia
rus polymerase.

64 VIRUS-SPECIFIC MESSENGER RNA AND NASCENT POLYPEPTIDES IN POLYRIBOSOMES OF CELLS
PLICATING MURINE SARCOMA-LEUKEMIA VIRUSES. *(E.)*
cchio, G. (St. Louis U. Sch. Med., Mo.), N.
sochida, G. Shanmugan and M. Green. *Proc Natl
ad Sci USA* 70(7):2064-2068, 1973.

idence is presented that virus-specific RNA is
resent in polyribosomes of transformed cells re-
icating the murine sarcoma-leukemia virus complex
d that it serves as messenger RNA for the syn-
esis of viral-coded proteins. Both virus-specific
A (detected by hybridization with the [^{3}H]DNA
oduct of the viral RNA-directed DNA polymerase)
d nascent viral polypeptides (measured by pre-
pitation with antiserum to purified virus) were
und in membrane-bound and free polyribosomes.
embrane-bound polyribosomes contained a higher content of both virus-specific RNA and nascent viral polypeptides. From 60 to 70% of viral RNA sequences were released from polyribosomes with EDTA, consistent with a function as messenger RNA. Maximum amounts of both virus-specific RNA and nascent viral polypeptides were found in the polyribosome region sedimenting at about 350S. Thus, it is possible that the synthesis of RNA tumor virus proteins is initiated on free polyribosomes and that virus-specific complexes attach to membranes to a higher degree than do cellular polyribosomes. The attachment to membranes of virus-specific polyribosomes is necessitated by maturation of virion polypeptides, a process that may involve proteolytic cleavage and glycosylation and may require enzymes present on microsomes.

1965 INTRACISTERNAL A PARTICLES IN OVA AND PREIMPLANTATION STAGES OF THE MOUSE. *(E.)*
Calarco, P. G. (Dept. Anat. Biol., U. California, San Francisco) and D. Szollosi. *Nature [New Biol]* 243(124):91-93, 1973.

Studies were conducted on the ultrastructure of intracisternal A particles found in oocytes and preimplantation stages of several strains of mice. Intracisternal A particles were seen in small numbers in dictyate oocytes immediately after their release from ovarian follicles. They were enclosed within agranular endoplasmic reticulum (ER) and had fine strands which projected from their outer shell toward the inner wall of the ER. Intracisternal A particles were only rarely observed in oocytes after breakdown of the germinal vesicles began, and none was seen in metaphase II oocytes or in fertilized eggs. However, many budding particles became evident adjacent to agranular ER in late two-cell stages. A few fully formed particles were also seen in the cytoplasm and within the first polar body. Intracisternal A particles were most abundant in the four- and eight-cell stages and were only rarely encountered in the 16-cell (morula) and 32-plus-cell (blastocyst) stages. No other types of virus particles were seen at any stage examined.

1966 CONTACT INHIBITION OF MOVEMENT IN THE CULTURES OF TRANSFORMED CELLS. *(E.)*
Guelstein, V. I. (Acad. Med. Sci., USSR), O. Yu. Ivanova, L. B. Margolis, Ju. M. Vasiliev and I. M. Gelfand. *Proc Natl Acad Sci USA* 70(7):2011-2014, 1973.

Results of cell-cell collisions were studied with the aid of time-lapse microcinematography in primary cultures of normal mouse-embryo fibroblast-like cells and in cultures of transformed mouse cells of two types: (a) primary fibroblast-like cells transformed by Moloney mouse sarcoma virus; and (b) neoplastic fibroblasts of the CIM strain. Collisions of normal fibroblast-like cells and CIM cells in mixed cultures were also analyzed. Classification of the results of collisions were based on observation of the movements of the active cell edge during the first hour after the moment when this edge had contacted another cell. Three types of collision results were detected: halt of the active edge, overlapping, and underlapping. The relative number of overlappings was not higher and that of halts not lower in the cultures of transformed cells as compared with those of normal cells. Analysis of the collisions of normal fibroblasts with transformed cells gave similar results. Thus, the altered morphology of the cultures of these transformed cells cannot be explained by loss of contact inhibition of movement leading to increased ability of cells to move over the surfaces of other cells after collision. It is suggested that deficient attachment to the substrate may be very important. Abnormal attachment to the substrate may alter the cell shape and possibly modify certain characteristics of collisions, such as the degree of underlapping, frequency, and the degree of retraction.

1967 TEMPERATURE-SENSITIVE SIMIAN VIRUS 40-TRANSFORMED CELLS: PHENOMENA ACCOMPANYING TRANSITION FROM THE TRANSFORMED TO THE "NORMAL" STATE.

(E.) Renger, H. C. (New York U. Sch. Med., N.Y.) and C. Basilico. *J Virol* 11(5):702-708, 1973.

Temperature-sensitive simian virus (SV40)-transformed 3T3 cells (tsSV3T3), which express the transformed phenotype when growing at 32 C but not at 39 C, were used to study changes in growth behavior during shift-up or shift-down experiments. In cultures of tsSV3T3 cells which had reached or were beyond monolayer density at 32 C, DNA synthesis reached very low levels within 24 to 48 hr after shift-up. When cells which had been allowed to grow to high densities at 32 C were shifted to 39 C, not only cell growth stopped, but within two to three days the cultures shed a large number of cells into the medium. These cells were nonviable, and shedding stopped only when the number of cells attached had been reduced to that characteristic of the saturation density of 39 C. The remaining attached cells were viable and after the shift to 32 C were again able to grow from the monolayer to high cell densities. This behavior is compared with that of normal 3T3 and wild-type SV3T3 cells under different conditions. New tsSV3T3 lines have been isolated, using cells which had been infected with non-mutagenized wild-type SV40. This further demonstrates that the temperature sensitivity of these lines is due to a cellular rather than a viral mutation. Available data suggests that the temperature sensitivity could be due to changes in the surface or serum requirements. The mechanism of cell death which accompanies this phenomenon is still obscure. The data shows, however, that the host mutation carried by the tsSV3T3 cells affects a number of parameters of malignant transformation, and possibly all of them.

1968 INDEPENDENT SEGREGATION OF LOCI FOR ACTIVATION OF BIOLOGICALLY DISTINGUISHABLE RNA C-TYPE VIRUSES IN MOUSE CELLS.

(E.) Aaronson, S. A. (Natl. Cancer Inst., Bethesda, Md.) and J. R. Stephenson. *Proc Natl Acad Sci USA* 70(7):2055-2058, 1973.

Two genetic loci for induction of RNA C-type virus in BALB/c mouse embryo cells were found to segregate independently in backcross embryo cell lines. The viruses at these loci are shown to be biologically distinguishable. The first, described previously, codes for activation of a virus that grows preferentially in NIH Swiss embryo cells. The newly detecte locus codes for activation of a virus that grows poorly, if at all, in NIH Swiss or BALB/c cells, but replicates well in a rat cell line. Its serologic properties are different from those of viruses of th two major serologic subgroups of murine leukemia virus. The present findings provide support for the hypothesis that the loci for virus induction in BALB/c cells represent genetic information for C-typ viruses and that the endogenous viruses are subject to different cellular genetic controls. The relatio ship of these findings to the mechanisms of leukemia development *in vivo* has yet to be determined.

1969 ISOLATION AND CHARACTERIZATION OF A PRIMATE SARCOMA VIRUS: MECHANISM OF RESCU

(E.) Scolnick, E. M. (Natl. Cancer Inst., Bethesda, Md.) and W. P. Parks. *Int J Cancer* 12(1):138-147, 1973.

A clonal isolate of an RNA-containing sarcoma virus derived from a spontaneous woolly monkey fibrosarcom has been obtained. The woolly sarcoma virus transformed cells do not spontaneously yield infectious virus but do contain the woolly gs antigen. The primate sarcoma virus can be rescued with comparable efficiency by superinfection with either murine or primate helper type-C viruses. During rescue with murine helper type-C virus, the intracellular level of woolly monkey gs antigen does not rise in proportion to the production of helper gs antigen, or in proportion to the production of transmissible focus-forming virus. The results suggest that helper virus rescue of sarcoma viruses does not involve pri marily an increase in the production of sarcoma viru components but rather simply an assembly of sarcoma components into an infectious particle during the production of the helper type-C virus.

1970 RELATIONSHIP OF REPLICATION AND TRANSCRIPTION OF SIMIAN VIRUS 40 DNA. *(E.)*

Cowan, K. (Dept. Pharmacol., Case Western Reserve U., Cleveland, Ohio), P. Tegtmeyer and D. D. Anthony *Proc Natl Acad Sci USA* 70(7):1927-1930, 1973.

RNA produced by the Simian virus 40 (SV40) mutant tsA30 during lytic infection of kidney cells of African green monkeys was examined by RNA-DNA competition-hybridization. This mutant is temperature-sensitive in a function (gene A) that regulates synthesis of viral DNA. No detectable difference between mutant RNA synthesized at the permissive temperature (33 C) and wild-type viral RNA was found During continuous infection with the mutant at the restrictive temperature (41 C) only early viral RNA was produced. When mutant DNA and late RNA synthesis were initiated at the permissive temperature, a shift to the restrictive temperature rapidly terminated synthesis of viral DNA but not that of late viral RNA. Since late RNA synthesis was consistentl detected in infection by the mutant 2.5-6.5 hr after a temperature shift to 41 C, the replicative intermediate molecules are apparently not necessary for the continued transcription of late viral genes.

he patterns of RNA synthesis in lytic infection, n abortive infection, and in transforming infec- ion are different. It has not been determined hether a common mechanism regulating transcription s operating in each of these states. The data ndicate that the function of gene A is required efore synthesis of late viral RNA and that after nitiation, the production of late RNA continues ithout further expression of gene A or concomitant viral DNA synthesis.

971 PROSTAGLANDIN LEVELS IN NORMAL AND TRANSFORMED BABY-HAMSTER-KIDNEY FIBROBLASTS.

E.) Hammarström, S. (Dept. Chem., Karolinska Inst., tockholm, Sweden), B. Samuelsson and G. Bjursell. *ature [New Biol]* 243(123):50-51, 1973.

rostaglandin E_2 and $F_2\alpha$ levels were determined by uantitative mass spectrometry in normal and poly- ma virus-transformed baby hamster kidney (BHK) ibroblast cultures. BHK Cl 13 cells, which re- ained a normal response to growth controls *in vitro*, ad low intracellular prostaglandin levels and se- reted small amounts ($PGE_2 > PGF_2\alpha$) into the medium. ell density had no effect on prostaglandin produc- ion. Virus-transformed BHK cells produced 30-115 imes as much PGE_2 as the untransformed controls. inety-five-97% of the PGE_2 produced by transformed ells was present in the medium. BHK cells trans- ormed by a temperature-sensitive mutant produced 7- 2 times more PGE_2 than normal controls whether under ermissive or nonpermissive conditions.

972 FURTHER EVIDENCE FOR ONCORNAVIRUSES IN HUMAN MILK: THE PRODUCTION OF CORES.

E.) Feldman, S. P. (Coll. Phys. Surg., Columbia ., New York, N.Y.), J. Schlom and S. Spiegelman. *roc Natl Acad Sci USA* 70(7):1976-1980, 1973.

technique is described, using phospholipase C igestion followed by equilibrium density gradient entrifugation, for the preparation of cores, or ucleoids, from mouse mammary tumor virus and from uman milk particles. Cores from both sources ave a density of 1.26-1.27 g/ml and contain a 0-70 S RNA associated with an RNA-directed DNA poly- merase. These results provide further evidence of imilarity between human milk particles and animal NA tumor viruses. Use of the "core-isolation" echnique separates the cores from cell debris and ssociated enzymes which may inhibit RNA-directed NA polymerase and thus mask the presence of virus.

973 STUDIES OF NONDEFECTIVE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID VIRUSES. VI. HARACTERIZATION OF THE DNA FROM FIVE NONDEFECTIVE YBRID VIRUSES.

(E.) Henry, P. H. (U. Missouri Med. Ctr., Columbia), L. E. Schnipper, R. J. Samaha, C. S. Crumpacker, A. M. Lewis, Jr. and A. S. Levine. *J Virol* 11(5):665-671, 1973.

Certain biophysical characteristics of the DNA from each of the five nondefective adenovirus 2 (Ad2)-simian virus 40 (SV40) hybrid viruses ($Ad2^+ND_1$, $Ad2^+ND_2$, $Ad2^+ND_3$, $Ad2^+ND_4$, $Ad2^+ND_5$) have been determined. The guanine plus cytosine content varied from 55 to 57% and was not significantly different from that of nonhybrid Ad2 (56%), and the hybrid DNA molecules had mean molecular lengths which were similar to that of the standard, Ad2. The Ad2 and SV40 components of each hybrid were linked by alkali-resistant, presumably covalent bonds. The percentage of SV40 DNA in each hybrid virus was determined by hybridization with SV40 complementary RNA in a calibrated system. The results indicate that each hybrid virus DNA contains a different percentage of SV40 nucleotide sequences. The estimated size of the SV40 DNA component varies from 48,000 daltons for $Ad2^+ND_3$ to 840,000 daltons for $Ad2^+ND_4$, the latter being equivalent to between one-fourth and one-third of the SV40 genome.

1974 TRITIATED THYMIDINE UPTAKE BY *IN VITRO* SPLEEN LYMPHOID CELL CULTURES FOLLOWING STIMULATION BY GC MURINE LEUKEMIA VIRUS.

(E.) Abdel-Wahab, K. S. (Chicago Med. Sch., Ill.), T. Booden, R. A. Albach and D. A. Wilson. *Acta Virol (Praha)* 17(2):105-110, 1973.

In vitro ^3H-thymidine incorporation into acid-insoluble material was studied in spleen and thymus cells isolated from leukemic ARS/Ha/ICR mice infected with GC murine leukemia virus (MuLV). Based on the radioactivity per unit of cellular protein, leukemic cells incorporated more label than did normal, cultured thymic or splenic cells. However, unlike normal cells, leukemic cells showed little stimulation of ^3H-thymidine incorporation when cultured in the presence of either MuLV or phytohemagglutinin. This lack of stimulation was accompanied by a failure to show a further blastogenic response, suggesting that the virus-sensitized leukemic cells were in a state of impaired cell-mediated immunity.

1975 COMPARATIVE STUDY OF CYTOCHROMES BETWEEN VIRUS-TRANSFORMED AND UNTRANSFORMED CELLS.

(E.) Sato, N. (U. Pennsylvania Sch. Med., Pa.), B. Chance, K. Kato and W. Klietman. *Biochim Biophys Acta* 305(3):493-502, 1973.

A spectrophotometric analysis of the cytochrome contents of several tissue culture cell lines transformed by Simian virus 40 is compared with the untransformed cell lines. Tissue culture cells of virus-transformed and untransformed cell lines had low contents of cytochromes in the respiratory chain, measured per cell or per mg protein of the cells, in comparison to the cytochrome contents of liver cells *in vivo*. In the virus-transformed cells the contents of cytochromes aa_3, b and possibly c_1 were significantly lower than those in the untransformed cells, while the content of cytochrome c was found to be the same or even increased in the transformed cells. Thus, a markedly high ratio of cytochromes c/aa_3 was observed in the transformed cells. The decreased content of cytochromes could result in the decrease in the respiratory rate of tumors *in vivo*. Polarographic measurements of the oxygen uptake showed a generally low rate of both endogenous respiration and respiration in the presence of glucose and vitamin K_3 in the transformed cells. The observation of changes in the cytochrome composition of cells indicates that cytochrome aa_3 or cytochrome oxidase is most easily affected, and that cytochrome *c* is least affected, by environmental change or by pathological conditions. This may be due to differences in the biosynthetic process of each cytochrome. The *b* cytochrome with an alpha band at 557 nm was increased in amount by the malignant transformation of the cell.

1976 ADENYLATE CYCLASE ACTIVITY IS DECREASED IN CHICK EMBRYO FIBROBLASTS TRANSFORMED BY WILD TYPE AND TEMPERATURE SENSITIVE SCHMIDT-RUPPIN ROUS SARCOMA VIRUS. *(E.)*

Anderson, W. B. (Natl. Cancer Inst., Bethesda, Md.), E. Lovelace and I. Pastan. *Biochem Biophys Res Commun* 52(4): 1293-1299, 1973.

Adenylate cyclase levels were studied in normal and Schmidt-Ruppin strain Rous sarcoma virus (SR-RSV)-infected chick embryo fibroblasts by a radioassay. Cells infected with wild-type virus showed 40-50% less adenylate cyclase activity than normal cells. Change in growth temperature had no effect on enzyme levels. Cells infected with a temperature-sensitive mutant had decreased adenylate cyclase levels at the permissive temperature and normal levels at the nonpermissive temperature. Enzyme activity decreased slowly over a 24 hr period when nonpermissive cells were switched to the permissive temperature. The decreased enzyme activity was not due to an altered Km(ATP) nor to a change in the kinetics of Mg^{++} activation, and was not observed when the enzyme was assayed in the presence of NaF. Several of these properties of SR-RSV infected cells differed from those of cells infected by the Bryan high-titer strain RSV, and it was thus concluded that transformation by SR-RSV reduces enzyme activity by a different mechanism than that of the Bryan strain.

1977 INTERFERON INDUCTION IN NEW ZEALAND BLACK MICE BY MURINE LEUKEMIA VIRUS. *(E.)*

Siegel, B. V. (U. Oregon Med. Sch., Portland), M. Brown and J. I. Morton. *J Immunol* 111(2):644-646, 1973.

The interferon response to Rauscher leukemia virus (RLV) in the autoimmune New Zealand Black (NZB) model was investigated; for comparative purposes the C57BL/6 and BALB/c strains were also included. Mice were inoculated i.p. with 0.20 ml of a 10% suspension of RLV-infected spleen extract. The interferon response was compared in RLV-inoculated and uninoculated NZB controls of various ages. The animals were bled at 24 hr post-inoculation for interferon assay and sacrificed at 22 days for spleen weights. Both interferon production and viral leukemogenesis increased progressively with age. Thus, the production of higher levels of interferon with increasing age appears not to afford additional protection against infection by RLV. X-irradiation experiments indicate that the hemopoietic system is the origin of the circulating interferon induced in NZB mice by RLV. The enhanced capacity to form interferon with age may conceivably have been a consequence of the chronic autoimmune stimulation and concomitant increase in cell populations available for interferon production.

1978 VIROPEXIS OF TYPE B PARTICLES IN RETICULUM CELL SARCOMA OF RIII/DM STRAIN MICE. *(E.)*

Seman, G. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston) and L. Dmochowski. *Cancer Res* 33(6): 1238-1246, 1973.

Results of ultrastructural studies of generalized reticulum cell sarcomas in six virgin RIII/Dm strain female mice with s.c. transplants of isologous embryo tissues are presented. All six mice developed, at the site of inoculation, nodules that grew in two months to a size of about 15 mm and then partially regressed. Two of the mice were found to have considerable splenomegaly at 335 days after inoculation. Among the other four mice, two died tumor free at the age of 17 months and one developed a mammary adenocarcinoma at 17 months. The spleen tumors contained numerous type B and type C virus particles and, in smaller numbers, intracytoplasmic type A virus particles. Budding of virus particles was frequently seen. In the spleen tumors, type B virus particles were seen adsorbed on the plasma membrane of the reticulum tumor cells and penetrating into the cytoplasm of the cells by a mechanism described as viropexis. Nucleoids of the virions were apparently released in the cytoplasm of the tumor cells. Both type B and type C particles were phagocytized by macrophages in the spleen tumors. Adsorption of type B particles, budding of type C particles, and intracytoplasmic type A particles were observed in the same tumor cells. Type C particles and budding of type C particles, but not type B particles, were also seen in the lymph nodes and the intrathoracic tumors of these mice. Type B particles and their behavior in reticulum cell sarcoma of mice have not been previously recorded.

1979 HEXOSE TRANSPORT IN NORMAL AND IN ROUS SARCOMA VIRUS-TRANSFORMED CELLS. *(E.)*

Weber, M. J. (Dept. Microbiol., U. Illinois, Urbana) *J Biol Chem* 248(9):2978-2983, 1973.

Transport across the cell membrane of 3-*O*-methylglucose, a nonphosphorylatable glucose analogue, was measured in cultures of chick embryo fibroblasts which were either growing exponentially, contact-inhibited, or transformed by Rous sarcoma virus. Transport of this hexose was found to be 4 to 5 times faster in transformed cells than in normal cells growing at the same rate, and 30 times faster than in contact-inhibited cells. The rate of efflux was increased to the same extent as the rate of influx. 3-*O*-Methylglucose transport in these cells was found to be a saturable process which was not accompanied by post-transport phosphorylation and which did not result in accumulation of the hexose against a concentration gradient. It is concluded that a facilitated diffusion system for hexose transport is activated in the transformed cells. Kinetic measurements revealed that the K_m for transport of 3-*O*-methylglucose or of 2-deoxyglucose and the K_i for the phlorizin inhibition of 2-deoxyglucose transport were not significantly altered by malignant transformation. Only the V_{max} for hexose uptake showed consistent, significant change. It is suggested that a new hexose transport system is not synthesized in the transformed cells and that the activation of hexose transport occurs via either a modification or additional *de novo* synthesis of the normal cellular transport system. The results presented here are the clearest indication to date of a functional alteration in the plasma membrane associated with malignancy.

1980 THE ROLE OF HOST RNA POLYMERASE IN P1 PHAGE DEVELOPMENT. *(E.)* Myers, D. E. (Div. Biol. Med. Sci., Brown U., Providence, R.I.). *Virology* 51(2):521-524, 1973.

As part of an investigation of the mechanism by which expression of genome of the rifampicin-sensitive bacteriophage P1 is regulated, studies were conducted to determine whether P1 phage development is dependent at any time on an RNA polymerase coded for by the phage. P1 production in a rifampicin-sensitive strain of *E. coli* was inhibited when rifampicin was added during the first 30 min after infection. Complete phage particles continued to be produced in the rifampicin-sensitive host for 10 min after addition of the drug. This phenomenon was not a result of a vital phage-specific RNA polymerase which succumbs to rifampicin with kinetics different from those of host cell enzyme since phage development in a rifampicin-resistant *E. coli* strain was completely normal in the presence of rifampicin. Since rifampicin inhibited RNA synthesis equally well in both uninfected and infected sensitive hosts, the 10 min delay was also not due to rifampicin-resistant transcription. It was most likely a result of continued processing of RNA chains already initiated at the time of rifampicin addition. Thus, P1 lytic development is dependent on host RNA polymerase.

1981 INTRACISTERNAL TYPE A PARTICLES OCCURRING IN FOREIGN BODY-INDUCED SARCOMAS. *(E.)* Johnson, K. H. (Coll. Vet. Med., St. Paul, Minn.), . K. G. Ghobrial, L. C. Buoen, I. Brand and K. G. rand. *Cancer Res* 33(6):1165-1168, 1973.

ight sarcomas induced in mouse strains CBA/H, CBA/H-6, and C57BL/10ScSn (or their hybrids) by s.c. im- lantation of smooth or roughened plastic films or lass pieces were examined electron microscopically. ype A virus particles were detected within dilated rgastoplasmic cisternae of the sarcoma cells from ive mice. The doughnut-shaped particles measured 0 to 100 nm and were seen to bud from membranes of e granular endoplasmic reticulum. All five virus- ositive sarcomas developed in secondary hosts that ad received segments of cell-laden implants and/or urrounding tissue capsules transferred from the ori- inal implant carriers during the period of nonneo- lastic foreign body reaction. In all cases the tu- ors were derived from cells of the original host. No virus particles were found in three sarcomas that had developed directly in original implant carriers. Whether the type A particles demonstrated in this study are etiologically related to the foreign body-induced sarcomas or whether they are only passenger agents was not determined.

1982 ANTIGENIC PROPERTIES OF ENDOGENOUS TYPE-C VIRUSES FROM SPONTANEOUSLY TRANSFORMED CLONES OF BALB/3T3. *(E.)* Aoki, T. (Natl. Cancer Inst., Bethesda, Md.) and G. J. Todaro. *Proc Natl Acad Sci USA* 70(5):1598-1602, 1973.

During long-term tissue culture of spontaneously transformed clones derived from BALB/c 3T3 mouse-embryo cells, some clones spontaneously begin to produce high titers of endogenous murine type-C viruses. The antigenic properties of these viruses have been analyzed by indirect immunoelectronmicroscopy and can be classified into two distinguishable populations: (*a*) BALB/c murine myeloma-associated extracellular viruses that carry a specific envelope antigen, xVEA, different from the typical murine leukemia viral envelope antigens; and (*b*) previously uncharacterized type-C viruses that have neither xVEA nor the murine leukemia viral envelope antigens. The former produces PC1 antigen and the latter might induce a new cell-surface antigen. Neither of these two populations of BALB/3T3 endogenous type-C viruses was able to infect BALB/c cells but both could infect NIH Swiss cells. A single BALB/3T3 clone, then, can release infectious endogenous type-C viruses with at least two different antigenic properties. It is concluded that BALB/c somatic cells contain preexisting genetic information for production of at least two closely related but, nevertheless, distinct type-C viruses.

1983 DNA POLYMERASE ACTIVITY IN HUMAN SERUM: STUDIES WITH AUSTRALIA ANTIGEN. *(E.)* Loeb, L. A. (Inst. Cancer Res., Philadelphia, Pa.), R. O. Williams, A. I. Sutnick, A. O'Connell and I. Millman. *Proc Soc Exp Biol Med* 143(2):519-525, 1973.

The presence of DNA polymerase in normal human serum and its possible association with Australia antigen was investigated. DNA polymerase activity was determined in 135 samples of normal serum. Only three samples revealed appreciable amounts of activity. The addition of a variety of polynucleotide templates did not stimulate polymerase activity. Australia antigen is a particle associated with hepatitis and is reported to contain RNA. A DNA polymerase, "reverse transcriptase" might be required for replication of this RNA. DNA polymerase activity was found in only one of five preparations of purified Australia antigen. A similar activity was also found to be present in serum from certain normal volunteers. In either case, polymerase activity does not require nor is it stimulated by added polynucleotides suggesting that the enzyme may already be associated with a template such as RNA or DNA. A comparison between the amount of DNA polymerase activity in serum and the presence of Australia antigen in three series of patients with

Down's syndrome and hepatitis revealed at most a marginal positive association. Thus, DNA polymerase is probably not a part of Australia antigen and the association observed is probably of an indirect nature. Its presence in occasional normal sera must be considered when using this activity as an index for detection of oncogenic viruses.

1984 SPECIFICITY OF ANTIBODY TO THE RD-114 VIRAL POLYMERASE.

(E.) Long, C. (Flow Labs, Inc., Rockville, Md.), R. Sachs, J. Norvell, V. Huebner, M. Hatanaka and R. Gilden. *Nature* [*New Biol*] 241(109):147-149, 1973.

Hyperimmune guinea-pig antiserum was prepared against the purified RNA-dependent DNA polymerase (RDP) of RD-114, the candidate human C-type virus. Immune serum fractionated by ammonium sulfate precipitation and dialyzed against Tris-HCl in 0.1 M NaCl inhibited RDP activity from RD-114, woolly monkey and gibbon ape C-type viruses, and a pseudotype sarcoma virus (MSV (RD-114)) using either poly rA·oligo dT or poly d(A-T) as template. The antiserum had no effect on RDP activity from feline, mouse, hamster, rat chicken and viper viruses.

1985 ELECTRON MICROSCOPIC STUDY OF THE DISTRIBUTION OF CONCANAVALIN A RECEPTOR SITES AT THE SURFACE OF NORMAL CELLS AND CELLS TRANSFORMED BY ONCOGENIC VIRUSES.

(Fr.) Torpier, G. (Pasteur Inst., Paris, France) and L. Montagnier. *Int J Cancer* 11(3):604-615, 1973.

The concanavalin-peroxidase method was used to study the distribution of receptor sites for concanavalin A at the surface of normal and of polyoma, RSV or MSV transformed normal hamster fibroblasts (BKH 21/13) and fibroblasts transformed by polyoma and Rous or mouse sarcoma virus; results were correlated with agglutinability of these cells. The product of concanavalin-bound peroxidase activity had the same thickness in all the clones studied. A somewhat more irregularly distributed reaction was observed in some transformed clones than in normal, untransformed clones, but these changes were not necessarily accompanied by changes in agglutinability. Irregular distribution of receptor sites has also been observed in normal human and mouse cells. It is concluded that the surface change detected by the peroxidase method cannot be considered as a general transformation characteristic.

1986 BRAIN TUMOR TYPES INDUCED BY THE SCHMIDT-RUPPIN STRAIN OF ROUS SARCOMA VIRUS IN INBRED FISCHER RATS.

(E.) Wilfong, R. F. (Duke U. Med. Ctr., Durham, N.C.), D. D. Bigner, D. J. Self and W. Wechsler. *Acta Neuropathol* 25(3):196-206, 1973.

The induction and classification of autochthonous neoplasms of neuroectodermal and mesenchymal origin in CDF Fischer rats following the neonatal intracerebral inoculation of the Schmidt-Ruppin strain of Rous sarcoma virus was studied. The inoculation of 10^5 FFU of virus into the cerebrum of 2 day old rats was followed by 96% incidence of tumor growth after a latency period of 3 months. Twenty-four of twenty-five inoculated animals developed tumors, all of which were primary intracerebral neoplasms. They fell into three main groups: gliomas 60%, sarcomas 31%, and gliosarcomas 9%. Of the 34 gliomas induced, 28 (84%) were astrocytic, and there was one oligodendroglioma, 2 mixed gliomas, and 3 ependymoma-like tumors. The advantages of this brain tumor model are a high glioma incidence, and a short induction time in a commercially available strain of inbred Fischer rats previously shown to be susceptible to the neuro-oncogenic action of resorptive chemical carcinogens.

1987 CELLS, INTRACELLS AND SCHIZOGENY. IV. FURTHER STUDIES ON INTRACELL FORMATION AND SCHIZOGENY PHENOMENA IN HUMAN AMNION CELLS AND SV40 VIRUS-TRANSFORMED MOUSE EMBRYO CELLS *IN VITRO*.

(E.) Valladares, Y. (Natl. Inst. Oncology, City U., Madrid Y. Alvarez, J. Alvarez and E. Tabares. *Oncology* 27(5):437-458, 1973.

Schizogenic cells of the AV3/INO and ER-SV40 lines display numerous aberrations, including the production of intracells (endogenous microcells), whose morphological type, mechanism of production and fate are described and discussed. It is possible to distinguish the following types of genuine intracells: those (1) consisting of a nucleoid; (2) formed by a nucleoid and a small amount of cytoplasmoid; (3) constituted by a nucleoid within which there is a nucleoloid; and (4) as mature intracells possessing nucleoid, nucleoloid and cytoplasmoid. The whole population of the ER-SV40 line was abnormal during more than 60 serial subcultures, surviving against any prediction. Asynchronous polykaryocytes, containing regions in different phases of the cell cycle, and cells resembling several lower phylogenetic forms are frequently encountered. Some of the cell lines originally had contact with CV and SV40 viruses, and some others contained cells showing particles resembling mycoplasmas, although they did not grow in mycoplasma culture media. Due to these facts, the relation, if any, between the schizogenic phenomena and intracell formation on the one hand, and cell transformation due to genetic changes promoted by viral and mycoplasma effects on the other hand are being investigated.

1988 SEROCHEMOTHERAPY AGAINST A MOLONEY VIRUS-INDUCED LEUKEMIA.

(E.) Pearson, G. R. (Natl. Cancer Inst., Bethesda, Md.), L. W. Redmon and J. W. Pearson. *Cancer Res* 33(8):1854-1857, 1973.

This study was designed to determine whether high-titered immune sera would be successful in eradicating established transplantable Moloney leukemia virus-induced lymphoma (LSTRA) disease when given either alone or after drug therapy. Sera from BALB/c mice bearing progressively growing tumors induced by Moloney sarcoma virus (progressor serum) or from BALB/c x DAB/2 F_1 mice immunized with Moloney sarcoma virus-induced tumor cells of C57BL origin

ogeneic serum) were tested in immunotherapy ex-
ments. The progressor serum had previously been
rted to contain low antibody titers as deter-
d in indirect membrane immunofluorescence and
ralization assays while the allogeneic serum
ained high antibody titers in both assays. Mul-
e inoculations of the allogeneic but not the pro-
sor serum into mice with systemic LSTRA disease
lted in a significantly increased median survival
over controls, but there were no long-term
ivors. When single and multiple injections
llogeneic serum were administered to diseased
following high-dose 1,3-bis(2-chloroethyl)-1-
osourea therapy (25 mg/kg), there was 79 and
respectively, long-term survivors (still alive
r 100 days, at the termination of the study) as
sed to 34% in the group treated with drug alone.
iple injections of normal serum also increased
percentage of survivors to 54%. Following low-
1,3-bis(2-chloroethyl)-1-nitrosourea therapy
mg/kg), administration of the allogeneic serum
luced up to 40% survivors as opposed to none in
control groups. The results indicate that well-
ined immune sera should be considered in experi-
s investigating combined chemoimmunotherapy as
approach to the control of cancer.

9 ONCOGENIC POTENTIAL OF GUINEA PIG HERPES- AND C-TYPE VIRUSES. *(E.)*

Hsiung, G. D.
le U. Sch. Med., New Haven, Conn.), C. K. Y.
and P. A. Gross. *Cancer Res* 33(6):1436-
2, 1973.

erpes-like virus and a C-type virus were repeat-
isolated from leukemic strain 2 guinea pigs.
success of the isolation of these two viruses
itro depended upon the conditions to which
cultures were exposed. Although it is unclear
her there is causal relationship between these
viruses and guinea pig leukemia, the conditions
the activation of these viruses *in vitro* are
ilar to those of other oncogenic herpesviruses
C-type viruses of man and animals. Preliminary
a showed that 1-day-old guinea pigs exhibited
erplasia in spleens when inoculated with a
nea pig C-type virus carrying cell line infect-
with guinea pig herpesvirus. It appears that
h viruses are necessary for the manifestation
neoplastic disease *in vivo*.

0 CHANGES IN SPLEEN CELLULARITY AND ERYTHROCYTE ACCUMULATION FOLLOWING RAUSCHER VIRUS ECTION IN THE MOUSE. *(E.)*

Markoe, A. M.
legheny Gen. Hosp., Pittsburgh, Pa.) and J. P.
newick. *Scand J Haemat* 10(4):273-281, 1973.

nature of the relationship of the spleen enlarge-
t effected by Rauscher virus infection of SJL/J
e has been investigated with reference to cell
liferation in the spleen and to splenic red blood
l pooling and anemia. The gross splenic nucleated
l content was found to increase linearly with
een weight. The ratio of total spleen cells to
leated spleen cells was identical at a value of
5 to 1 for both normal and virus-infected mice
even though in the latter some spleen sizes were as high as 30 times normal. On a per milligram basis, the average splenic accumulation of ^{59}Fe-labelled normal red blood cells by normal mice was approximately twice as great as by 18-day leukemic mice, even though the leukemic spleens contained a greater fraction of the injected label. Anemia was seen to develop shortly after injection of the virus, reducing the hematocrit to values of less than 40 by 2 wk. Compensatory increase in reticulocyte number did not occur until after the major fall in venous hematocrit had already been achieved. Peripheral white blood cell counts increased with time after virus injection and these correlated positively with spleen size, the relation being logarithmic. The data confirm the assumption that splenomegaly is a direct result of the cellular proliferative effects of Rauscher virus rather than a consequence of increased red blood cell pooling in the spleen.

1991 EVIDENCE FOR VIRAL INVOLVEMENT IN MURINE AND HUMAN MAMMARY ADENOCARCINOMA. *(E.)*

Schlom, J. (Coll. Physicians Surgeons, Columbia U., New York, N.Y.) and S. Spiegelman. *Am J Clin Pathol* 60(1):44-56, 1973.

Particles which have several biochemical and biophysical properties diagnostic of ribonucleic acid (RNA) tumor viruses of animals have been isolated from specimens of human milk. The human particles have a density of 1.16 to 1.19 g per ml., a RNA-directed deoxyribonucleic acid polymerase, and a 60-70S RNA. The quantity of particles in particular milk samples is determined by hybridization of the polyadenylic acid regions of their 60-70S RNA to radioactive polyribouridylic acid of known specific activity. A method involving molecular hybridization with radioactive DNA complementary to the RNA of the mouse mammary tumor virus was used to detect homologous RNA in both murine and human malignant breast tumors.

1992 VIRUS IN PROSTATIC EPITHELIUM OF MAN. *(E.)*

Webber, M. M. (U. Colorado Sch. Med., Denver), O. G. Stonington and J. Lehman. *Urology* 1(6):561-567, 1973.

Experiments conducted to examine previous observations indicating the presence of a viral agent in human prostatic cells maintained *in vitro* are described. Prostatic tissue was obtained from patients undergoing surgery for benign prostatic hyperplasia. Cytopathic effects, consisting of vacuolated syncytial cells, nuclear inclusions, and marked increased in acid phosphatase activity at the cell membrane and in the cytoplasm around the nucleus have been observed with the light microscope. Virus-like particles and nuclear changes related to a viral infection have been noted with the electron microscope. Investigations to isolate and characterize this agent and to determine its role in the cause of prostatic neoplasia are continuing. Although the exact mode in which a virus might be transmitted to the prostate is not apparent, systemic infections by herpes-type viruses are known; these may originate from genital herpes lesions. Tissues may be infected latently with a virus, and when environmental conditions are altered, virus spontaneously appears in the cells. These altered conditions may be provided *in vivo* by changes in hormonal balance as in the aging male and by a tissue culture environment *in vitro*.

1993 ULTRASTRUCTURAL LOCALIZATION OF T-ANTIGEN IN SV40-TRANSFORMED KIDNEY CELLS OF A GREEN MONKEY BY THE PEROXIDASE-LABELED ANTIBODY METHOD.

(E.) Baba, T. (St. Marianna U., Sch. Med., Sugao, Takatsu-ku, Kawasaki, Japan), N. Yamaguchi, R. Ishida and I. Suzuki. *Gann* 64(2):173-177, 1973.

The T antigen was stained successfully by immunoelectron microscopic method applied directly on the ultrathin section of SV40-transformed kidney cells of an African green monkey embedded in water-soluble plastics (glycol methacrylate). The development of this technique made it possible to clarify the ultrastructural localization of the antigen in transformed cells, showing that the T-antigen is associated closely with the nuclear chromatin.

1994 GENETIC INTERACTIONS OF AVIAN RNA TUMOR VIRUSES AND THEIR HOST CELLS.

(E.) Friis, R. R. (U. Southern California Sch. Med., Los Angeles). *Am J Clin Pathol* 60(1):25-30, 1973.

Normal chick embryo cells have been shown to express various functions usually associated with the replication of avian RNA tumor viruses in infected cells. These functions have been detected by a variety of techniques which include complement fixation tests for the presence of the viral group specific antigens, physiologic complementation tests for the defective function of the Bryan high titer strain of Rous sarcoma virus, observation of spontaneous production of typical RNA tumor viral particles, and induction of RNA tumor viral particle production by superinfection of chick cells with a second, but distinguishable, RNA tumor virus, or by activation with carcinogenic agents. Results from these experiments indicate that the expression of the endogenous viral genetic information varies from chick embryo to chick embryo. Thus far no pattern has emerged except that, in general, the content of group specific antigen detected by the complement fixation test is a fairly reliable indicator of whether or not a cell will have chick helper factor activity.

1995 *IN VITRO* TRANSCRIPTION OF DNA I OF SV40 BY RNA POLYMERASE II OF CVI CELLS.

(Fr.) Krsmanovic, V. (Med. Res. Unit, U. Lille II, France) and S. M. Weissman. *Arch Int Physiol Biol* 81(1):189-191, 1973.

RNA polymerase II was isolated from host CVI cells (monkey kidney cells) using a slight modification of Herzberg and Winocour's technique. When incubated with DNA I from SV40, this enzyme synthesized RNA containing ATP at the 5 end. In order to study the number of initiation sites of RNA polymerase II from CVI cells on the DNA I of SV40, ^{32}P-ATP was used as the radioactive percursor of RNA. After incubation at 37°C for 20 minutes the synthesized RNAs were extracted with dodecyl sodium sulfate, pu fied by filtration on Sephadex G-100 and precipitat to effect hydrolysis with ribonuclease T_1. Two-dimensional chromatography revealed the presence of a major oligonucleotide on the 5'-end, indicatin that there are very few RNA polymerase II initiatic sites on the DNA I of SV40. In order to study the two RNA polymerases (I and II) extracted by a different method, a slight modification of Roeder Rutter's technique was used. By using ^{32}P-guanosin triphosphate and ^{32}P-ATP in the presence of DNA I of SV40, it was found that polymerase I incorporate guanosine triphosphate at the 5-end while polymeras II preferentially initiates RNA synthesis via ATP.

1996 COENZYMES Q LEVELS IN LIVER, SPLEEN, AND BLOOD OF MICE WITH FRIEND LEUKEMIA VIRUS INFECTION.

(E.) Casey, A. C. (New England Inst., Ridgefield, Conn.) and E. G. Bliznakov. *Cancer Res* 33(6):1183-1186, 1973.

Coenzyme Q levels of blood, spleen and liver of mal Swiss-Webster mice infected with Friend leukemia virus (FLV) were determined by a spectro-photometr technique at 10, 20, 30 and 40 days after infectio and were compared with those of controls. Blood levels of total coenzyme Q were significantly elev in FLV infected mice, reaching values up to five times those of controls after 30 days. This increa was accompanied by the appearance of coenzyme Q_{10} which is not present in the blood of normal animal A similar shift to a predominance of coenzyme Q_{10} was also observed in the livers of infected mice although total enzyme levels were unchanged from control values. Coenzyme Q levels in the spleens of FLV infected mice were quantitatively and qualitatively the same as those in spleens of the contr The increased coenzyme Q blood levels were presumably due to the increased numbers of circulating erythroblasts in FLV infected mice.

1997 TRANSCRIPTIVE FUNCTIONS OF SENDAI VIRUS NUCLEOCAPSID IN INFECTED CELLS.

(E.) Bukrinskaya, A. G. (USSR Acad. Med. Sci., Moscow). *Virology* 52(2):344-350, 1973.

The synthesis of virus-specific RNA in the cytoplas of Ehrlich tumor cells infected with Sendai virus was studied. Cellular RNA synthesis was selectivel inhibited by actinomycin D (10 μg/ml). Viral RNA synthesis, as assayed by ^{3}H-uridine incorporation, was initiated within the ^{32}P-prelabeled parental nucleocapsid structures which sedimented at 1.31 g and 1.38 g/ml in CsCl gradients and as 200S and 150S particles, resp., in sucrose gradients. The 1.31 nucleocapsid was no longer demonstrable two hr after infection, and the 1.38 nucleocapsid struc ture was converted to a polysome-containing complex with a buoyant density of 1.45 g/ml. Virus-

ecific RNA synthesis proceeded within this com-
lex. Cycloheximide had no effect on production of
e 1.45 complex or on the synthesis of virus-
ecific RNA, suggesting that the parental nucleo-
psid within this complex contained preformed
anscriptase. The 1.45 complex dissociated three
after infection and nascent RNA separated from
e parental RNA. These findings suggest that the
45 complex is a transcriptive complex which
ntains parental nucleocapsid, and that virus-
ecific RNA is transcribed within the nucleo-
psid by a virion transcriptase.

98 CYTOMEGALOVIRUS: CONVERSION OF NON-PERMISSIVE CELLS TO A PERMISSIVE STATE
R VIRUS REPLICATION. *(E.)* St. Jeor, S. (Coll.
d., Pennsylvania State U., Hershey) and F. Rapp.
ience 181(4104):1060-1061, 1973.

llowing the finding that treatment of human fibro-
lasts with 5-iodo-2'-deoxyuridine (IdU) enhanced
e *in vitro* replication of human cytomegalovirus
MV) in these cells, experiments were conducted to
termine if IdU would enable the *in vitro* replication
CMV in normally nonpermissive human epithelioid
lls. Trypsin-dispersed second-passage epithelioid
man embryonic kidney cells were first incubated for
hr in the presence of IdU, washed twice, and then
posed to either virus or growth medium. In control
ltures the IdU was not added. Replication of the
irus was tested by measurements of viral DNA and
llular DNA produced and also by periodic assays for
fectivity. The DNA was measured by labeling
fected and sham-infected cultures with tritiated
ymidine, precipitation of acid-insoluble DNA, and
paration of the cellular and viral DNA by isopycnic
nding in CsCl. The data showed clearly that prior
reatment with IdU rendered nonpermissive epithelioid
lls permissive to infecting CMV; treated cells
roduced more than 3 log U of CMV. The technique
lds promise in studying virus agents that have
roved difficult to replicate *in vitro* and in
vestigating the factors that render cells non-
rmissive to virus infection.

999 VIRUS AND CARCINOGENESIS IN EPIDERMODYSPLASIA VERRUCIFORMIS.
(E.) Yabe, Y.
Okayama U. Med. Sch., Japan) and H. Koyama. *Gann*
4(2):167-172, 1973.

wo cases of epidermodysplasia verruciformis (EV),
human precancerous condition, were studied by
ight and electron microscopy. Skin lesions were
istologically typical of EV. Virus particles were
bserved in all of five typical benign lesions tes-
ed. They were observed in the nucleus of cells in
he upper epidermal layers. Negative staining of
issue extracts revealed that virus particles were
0-60 nm in diameter and had the same surface struc-
ure as the virus of human common warts, with 72
apsomeres arranged in the right-handed form. A
esion on the upper thigh of one of the two cases
howed a histology of early squamous cell carcinoma.
this case its transition from the typical benign
esion was evident. A few virus particles were also
bserved in the negatively stained tissue extract of
his lesion showing the histology of early squamous
cell carcinoma. These results suggest that the skin lesions of EV induced by a virus identical with or related to the wart virus become malignant and that the sunlight is not essential though it accelerates the malignant transformation of the skin lesions of EV. Common warts developing in normal individuals regress sooner or later. On the contrary, wart-like skin lesions induced in genetically defective individuals such as EV patients, are disseminated over the body and do not regress, and eventually some of them progress to carcinoma, at which stage no virus particles are observed.

2000 CHARACTERIZATION OF GUINEA PIG C-TYPE VIRUS.

(E.) Gross, P. A. (VA Hosp., West Haven, Conn.), C. K. Y. Fong and G. D. Hsiung. *Proc Soc Exp Biol Med* 143(2):367-370, 1973.

A guinea pig C-type virus was successfully isolated from primary and passaged cells derived from leukemic and normal strain 2, and strain 2-Hartley hybrid guinea pigs. Activation of the virus required 5-bromo-2'-deoxyuridine (BUDR) treatment. Morphologically, both in infected cells and in negatively stained preparations, the guinea pig C-type virus resembles other members of the C-type virus group. Purified guinea pig C-type virus labeled with ^{3}H-uridine had an equilibrium density of 1.17 ± 0.01 g/ml in sucrose. The absence of RNA-dependent DNA polymerase and the GS-3 antigen in the guinea pig C-type virus preparations hampered final identification. It is possible that the concentration of this enzyme in the C-type virus preparations was not high enough for the detection method.

2001 PROTEIN AND MESSENGER RNA REQUIREMENTS FOR SUPERHELICITY OF POLYOMA VIRUS DNA.

(E.) Cheevers, W. P. (Cancer Res. Lab., U. Western Ontario, London, Canada). *Nature* [*New Biol*] 242(120):202-204, 1973.

Cycloheximide (10 μg/ml) stopped protein synthesis in polyoma virus-infected mouse embryo cells within 20 min and reduced DNA synthesis by 95% within 3 hr. Representative sedimentation profiles in neutral sucrose gradients were made of low molecular-weight DNA synthesized during a 2 hr period in untreated infected cells and in cells pretreated for 30 min with cycloheximide. Pulse-labelled DNA extracted from untreated cells was comprised primarily of 20S DNA. The sedimentation properties of DNA from treated cells were quite different. A prominent peak of labelled DNA sedimenting at about 16S was observed in addition to some 20S DNA. On the basis of alkaline sedimentation profiles, it was determined that the DNA of untreated cells, sedimenting at 20S at neutral pH is comprised predominantly of form I polyoma DNA, which sediments in alkali at 53S. The material sedimenting near the top of the gradient (~18S) contains primarily cellular DNA. Labelled DNA of cycloheximide-treated cells, sedimenting at 20S at neutral pH is also comprised of a mixture of form I viral DNA and linear cellular DNA. DNA isolated from the 13-18S region of neutral sucrose gradients after sedimentation of extracts untreated and cycloheximide-treated cells had very different alkaline properties. The

13-18S DNA of untreated cells was almost exclusively cellular in nature. In contrast, the 13-18S DNA of cycloheximide-treated cells contained a major component sedimenting at 53S. This indicated that, in cycloheximide-treated cells, a superhelical DNA component is formed which sediments more slowly than form I viral DNA. It is believed that both are similar in molecular weight and that the dissimilarity exhibited at neutral pH is due to decreased superhelicity of the component synthesized in the presence of cycloheximide. A stoichiometric relationship existed between the formation of form I polyoma DNA and the synthesis of protein. CsCl equilibrium centrifugation analysis of native DNA synthesized 24-26 hr after polyoma infection of cells pretreated with actinomycin D indicated that the mRNA which formed the protein required to maintain the normal superhelix density of polyoma DNA was much more stable (half-life = 5-6 hr) than the protein itself. These results are consistent with the view that superhelicity of newly synthesized polyoma virus DNA is not maintained by continuous transcription and is therefore not regulated at this level.

2002 CELLULAR DNA SYNTHESIS AND MITOTIC ACTIVITY OF RAT EMBRYO FIBROBLAST INFECTED WITH TUMORIGENIC ADENOVIRUS TYPE 12. *(Rus.)* Ageyenko, A. I. (P. A. Gertsen Sci. Res. Inst. Oncol., Moscow, USSR) and I. Ya. Kogan. *Vopr Onkol* 19(2):38-41, 1973.

Rat embryo fibroblasts, grown on No. 199 media containing 10% bovine serum, were inoculated with human adenovirus type 12 after 24 hr (preceding monolayer formation), 48 hr (in media with no bovine serum and after monolayer formation) and 5-6 days of cultivation to study their mitotic activity and DNA synthesis (^{3}H-thymidine incorporation) during the logarithmic, early stationary, and late stationary phases, resp. The mitotic index reached 25.5 and 51.5 at 24 and 120 hr after infection, resp. as compared to 21.5 and 23, resp. in the noninfected control cultures during the logarithmic phase. During the early stationary phase mitotic indices were 16.5 and 15.5 in the control and 30.5 and 31.5 in the test cultures 24 and 120 hr after infection, resp. Mitotic indices decreased to 6 in the control and to 10.5 in the test culture 24 hr after infection during the late stationary phase. Adenovirus type 12 stimulated the induction of DNA synthesis during the first 24 hr after infection in the logarithmic phase. Later the rate of DNA synthesis decreased and no DNA induction was observed during the stationary phase. The induction of mitosis appeared to be independent of the DNA synthesis induction.

2003 HERPESVIRUS TYPE 2 INDUCED, TUMOR-SPECIFIC ANTIGEN IN CERVICAL CARCINOMA. *(E.)* Aurelian, L. (Johns Hopkins U. Sch. Med., Baltimore, Md.), H. J. Davis and C. G. Julian. *Am J Epidemiol* 98(1):1-9, 1973.

A micro-quantitative complement fixation test was used to detect antibody to a tumor-specific herpesvirus type 2 induced antigen (AG-4) in sera from patients with cervical neoplasia. The prevalence of antibody to AG-4 in women with cervical carcinoma displayed the progression expected of the neoplastic lesion. Antibody was detected in 35% of 20 cases of atypia, 73% of 22 cases of carcinoma *in situ* and 89% of 27 cases of untreated invasive carcinoma. Antibody to AG-4 was virtually absent in women without cancer and matched to the carcinom patients for age, race and socioeconomic class. The frequency of antibody in the carcinoma patients appeared to be a function of the extent of the tumor. Antibody to AG-4 was absent in 23 cases with successful therapy prior to blood collection, and present in three cases with recurrent neoplastic disease following radiation or hysterectomy; it was not affected by age or socioeconomic class. Finally, AG-4 reactivity was present in 5 of 6 biopsies of squamous cervical carcinoma but not in a biopsy from a normal woman and one from a case of adenocarcinoma. The data suggest that AG-4 be of diagnostic and prognostic significance, thus flecting tumor growth and arguing in favor of an ac role for HSV-2 in cervical neoplasia.

2004 ELECTRON MICROSCOPIC STUDIES OF PINOCYTOS AND OF THE SYNTHESIS OF POLYOMA VIRUS IN CELL CULTURES. *(Ger.)* Fasske, E. (Inst. Exp. Biol Med., Borstel, W. Germany), R. Fetting, D. Rühland and H. Themann. *Beitr Pathol* 149(2):158-169, 1973.

The morphologic steps of viral synthesis in culture of 48-hour-old mouse embryo cells infected with BB/ 2 (Graffi) strain polyoma virus is described. The cultures were fixed at intervals with glutaraldehyde and OsO_4, embedded in Epon 812, sectioned, strained with uranyl acetate and lead citrate, and examined with an electron microscope. In the first phase, the virus particles became attached to small indentiations of cell surfaces, surrounded by a 5 µm membrane, incorporated into the cytoplasm, and c centrated in phagolysosomes where they disintegrate The cytopathogenic effect is seen 120 hr after initial infection, lytic alterations first occurring i the cytoplasm. In the second phase, the DNA synthesising apparatus of the host cell is activated, as expressed by recombination of virus particles in the nucleus and filling the nucleus with the particles. Subsequent nuclear lysis liberates the virus into the culture medium.

2005 SYMMETRICAL *IN VIVO* TRANSCRIPTION OF POLY DNA AND THE SEPARATION OF SELF-COMPLEMENT VIRAL AND CELL RNA. *(E.)* Aloni, Y. (Dept. Genetic Weizmann Inst. Sci., Rehovot, Israel) and H. Locker *Virology* 54(2):495-505, 1973.

Mouse kidney cells infected with polyoma virus were labeled for 20 min with [5,6-^{3}H]uridine late in inf tion. The rapidly-labeled RNA was extracted from w cells and from the Hirt SDS/high salt supernatant f tion. The RNA was self-annealed and became resista to ribonuclease digestion. The RNase-resistant RNA isolated by chromatography on Sephadex G-100 and sh to be double-stranded RNA by several different crit resistance to RNase and to the combined effects of and DNase, the characteristic buoyant density of do stranded RNA in Cs_2SO_4, a buoyant density increase denaturation to that of single-stranded viral RNA m an inability to hybridize with complementary DNA un

natured, and a high T_m value with a sharp transition
RNase sensitivity. Separated strands of the double-
randed RNA hybridized with high efficiency with com-
nent I of polyoma DNA. After self-annealing in for-
mide at low temperature, from 18 to 30% of the total
pidly-labeled viral RNA of infected cells sedimented
11 S; 11 S corresponds in size to about 30% of the
lyoma DNA. The extraction procedure for double-
randed viral RNA also yields double-stranded cellu-
r RNA, but the double-stranded viral RNA can be fur-
er purified by using the Hirt supernatant, the RNase-
sistance of the viral RNA coupled with its higher T_m,
s greater sedimentation coefficient. These observa-
ons indicate that, late in infection of mouse cells,
lyoma DNA is transcribed symmetrically over a con-
derable portion of its length, yielding self-comple-
ntary RNA that is distinguishable from the cellular
lf-complementary RNA.

006 NERVOUS SYSTEM INVOLVEMENT IN BURKITT'S LYMPHOMA IN KENYA. *(E.)* Singh, S. (Ken-
atta Natl. Hosp., Nairobi), P. Clifford and S. A.
atel. *Afr J Med Sci* 4(2):187-195, 1973.

007 THE NEUROLOGICAL PICTURE OF BURKITT'S LYMPHOMA IN IBADAN. *(E.)* Odeku, E. L.
Dept. Surg., U. Ibadan, Nigeria), A. Adeloye and
. O. Osuntokun. *Afr J Med Sci* 4(2):119-126, 1973.

008 MURINE ERYTHROBLASTOSIS BY A NOVEL VIRAL AGENT. *(E.)* Imamura, N. (Res. Inst.
uclear Med., Biol., Hiroshima U., Japan). *Gann*
4(2):121-131, 1973.

009 A RABIES-INDUCED SERUM FACTOR INHIBITING ROUS SARCOMA VIRUS IN CHICKS. *(E.)* Desai,
M. (Dept. Virology, Haffkine Inst., Bombay,
dia), P. B. Sehgal, A. N. D. Nanavati and M. V.
irodkar. *J Genet Virol* 19(part 3):285-293, 1973.

010 DIFFERENTIAL EFFECT OF CYTOCHALASIN B ON NORMAL AND TRANSFORMED MOUSE CELLS. *(E.)*
elly, F. (Cold Spring Harbor Lab., N.Y.) and J.
mbrook. *Nature [New Biol]* 242(120):217-219, 1973.

011 FACTOR IN ACIDIFIED SERUM TOXIC FOR MALIGNANT CELLS IN CULTURE. *(E.)* Paul, D.
Salk Inst., San Diego, Calif.). *Nature [New Biol]*
2(119):186-188, 1973.

012 TRANSFER RNA ACTIVITIES OF ROUS SARCOMA AND ROUS ASSOCIATED VIRUSES. *(E.)* Wang,
(Dept. Microbiol., Indiana U., Bloomington), R.
. Kothari, M. Taylor and P. Hung. *Nature [New*
ol] 242(118):133-135, 1973.

013 AMINO ACID COMBINATIONS OF SIMIAN VIRUS 40 STRUCTURAL PROTEINS. *(E.)* Greenaway, P.
. (Cold Spring Harbor Lab., N.Y.) and D. LeVine.
ochem Biophys Res Commun 52(4):1221-1227, 1973.

2014 CLINICAL FEATURES OF HERPES GENITALIS. *(E.)* Kaufman, R. H. (Baylor Coll. Med., Houston, Tex.), H. L. Gardner, W. E. Rawls, R. E. Dixon and R. L. Young. *Cancer Res* 33(6):1446-1451, 1973.

2015 HERPESVIRUS AND CERVICAL CANCER STUDIES IN EXPERIMENTAL ANIMALS. *(E.)* Sever, J. L. (Natl. Inst. Hlth., Bethesda, Md.). *Cancer Res* 33(6):1509-1510, 1973.

2016 HERPESVIRUS AND THE LUCKÉ TUMOR. *(E.)* Granoff, A. (St. Jude Children's Res. Hosp., Memphis, Tenn.). *Cancer Res* 33(6):1431-1433, 1973.

2017 ONCOGENICITY OF RABBIT HERPESVIRUS. *(E.)* Hinze, H. C. (Dept. Med. Microbiol., U. Wisconsin, Madison) and D. L. Wegner. *Cancer Res* 33(6):1434-1435, 1973.

2018 TEMPERATURE-SENSITIVE AVIAN SARCOMA VIRUSES: A PHYSIOLOGICAL COMPARISON OF TWENTY MUTANTS. *(E.)* Wyke, J. A. (U. Southern California Sch. Med., Los Angeles) and M. Linial. *Virology* 53(1):152-161, 1973.

2019 STUDIES OF SV40 DNA. VI. CLEAVAGE OF SV40 DNA BY RESTRICTION ENDONUCLEASE FROM *HEMOPHILUS PARAINFLUENZAE*. *(E.)* Sack, G. H., Jr. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and D. Nathans. *Virology* 51(2):517-520, 1973.

2020 RAPID, SELECTIVE PROCEDURE FOR ISOLATION OF SPONTANEOUS TEMPERATURE-SENSITIVE MUTANTS OF MOLONEY LEUKEMIA VIRUS. *(E.)* Wong, P. K. Y. (Cancer Res. Lab., U. Western Ontario, London, Canada), L. J. Russ and J. A. McCarter. *Virology* 51(2):424-431, 1973.

2021 AVIAN SARCOMA VIRUS TRANSFORMED HAMSTER CELLS RESISTANT TO BROMODEOXYURIDINE AND DEFICIENT IN THYMIDINE KINASE ACTIVITY. *(E.)* Altaner, C. (Cancer Res. Inst., Slovak Acad. Sci., Bratislava, Czechoslovakia), M. Hladka and H. Schlechte. *Arch Gesamte Virusforsch* 41(1-2):40-51, 1971.

2022 SPECIFIC INFECTIVITY OF DIFFERENT WOUND TUMOR VIRUS ISOLATES. *(E.)* Liu, H.-Y. (Dept. Botany, U. Illinois, Urbana), I. Kimura and L. M. Black. *Virology* 51(2):320-326, 1973.

2023 INDUCTION OF ARGINASE ACTIVITY WITH THE SHOPE PAPILLOMA VIRUS IN TISSUE CULTURE CELLS FROM AN ARGININEMIC PATIENT. *(E.)* Rogers, S. (Biol. Division, Oak Ridge Natl. Lab., Tenn.), A. Lowenthal, H. G. Terheggen and J. P. Columbo. *J Exp Med* 137(4):1091-1096, 1973.

2024 THE SELECTIVE ISOLATION OF TEMPERATURE-SENSITIVE MUTANTS OF ROUS SARCOMA VIRUS. *(E.)* Wyke, J. A. (U. Southern California Sch. Med., Los Angeles). *Virology* 52(2):587-590, 1973.

2025 BASE SPECIFIC METHYLASE ACTIVITY IN RNA TUMOR VIRUSES: AVIAN LEUKOSIS VIRION-ASSOCIATED RNA METHYLASE(S). *(E.)* Gantt, R. (Natl. Cancer Inst., Bethesda, Md.), G. H. Smith and B. Julian. *Virology* 52(2):584-586, 1973.

2026 ENUCLEATION OF CELLS MADE SIMPLE AND RESCUE OF SV40 BY ENUCLEATED CELLS MADE EVEN SIMPLER. *(E.)* Croce, C. M. (Wistar Inst. Anatomy, Biol., Philadelphia, Pa.) and H. Koprowski. *Virology* 51(1):227-229, 1973.

2027 EFFECTS OF 2-DEOXY-D-GLUCOASE ON HERPES SIMPLEX VIRUS REPLICATION. *(E.)* Courtney, R. J. (Baylor Coll. Med., Houston, Tex.), S. M. Steiner and M. Benyesh-Melnick. *Virology* 52(2):447-455, 1973.

2028 TEMPERATURE-SENSITIVE MUTANTS OF HERPES SIMPLEX VIRUS TYPE 1: ISOLATION, COMPLEMENTATION AND PARTIAL CHARACTERIZATION. *(E.)* Schaffer, P. A. (Baylor Coll. Med., Houston, Tex.), G. M. Aron, N. Biswal and M. Benyesh-Melnick. *Virology* 52(1): 57-71, 1973.

2029 STEM CELLS IN THE PERIPHERAL BLOOD OF RAUSCHER LEUKEMIC MICE. *(E.)* Seidel, H. J. (Dept. Clin. Physiology, U. Ulm, Donau, Germany). *Experientia* 29(5):607-608, 1973.

2030 INHIBITION OF CELL PROLIFERATION CAUSED BY ONCOGENIC DNA-POLYOMA VIRUS. *(E.)* Zobl, H. (Med. High Sch., Hannover-Kleefeld, Germany), A. Georgii, G. Siegismund, W. Lang and L. Herzog. *Experientia* 29(5):595-596, 1973.

2031 IDENTIFICATION OF A LARGE POLYPEPTIDE PRECURSOR OF AVIAN ONCORNAVIRUS PROTEINS. *(E.)* Vogt, V. M. (Swiss Inst. Exp. Cancer Res., Lausanne) and R. Eisenman. *Proc Natl Acad Sci USA* 70(6):1734-1738, 1973.

2032 OCCURRENCE OF AMINOACYL-tRNA SYNTHETASE IN AN RNA ONCOGENIC VIRUS. *(E.)* Trávníček, M. (Inst. Organic Chem., Biochem., Czechoslovak Acad. Sci., Prague) and J. Římán. *Nature* [*New Biol*] 241 (106):60-62, 1973.

2033 SUPERCOILING OF SV40 DNA CAN OCCUR INDEPENDENTLY OF REPLICATION. *(E.)* White, M. (Dept. Biochem., Glasgow U., Scotland) and R. Eason. *Nature* [*New Biol*] 241(106):46-49, 1973.

2034 AN IMPROVED METHODOLOGY FOR PREPARATION OF VIRUS-INDUCED DOUBLE STRANDED RNA. *(E.)* Mechali, M. (Pasteur Lab., Radium Inst., Paris, France), R. Perez-Bercoff, G. Carrara and E. Falcoff *Biochimie* 55(3):361-363, 1973.

2035 COLONY FORMATION BY CHICKEN HEMATOPOIETIC CELLS AND VIRUS-INDUCED MYELOBLASTS. *(E.)* Dodge, W. H. (VA Hosp., Gainesville, Fla.) and C. Moscovici. *J Cell Physiol* 81(3):371-386, 1973.

2036 ISOLATION AND *IN VITRO* CHARACTERIZATION OF A HERPESVIRUS FROM FIELD MOUSE (*MICROTUS PENNSYLVANICUS*). *(E.)* Melendez, L. V. (Harvard Med. Sch., Boston, Mass.), M. D. Daniel, N. W. King, F. C. Calvo, H. H. Barahona, R. W. Thorington, D. A. Jackman and J. Cadwallader. *Lab Anim Sci* 23(3):385-390, 1973.

2037 TEMPERATURE-DEPENDENT CELLULAR REGULATION IN INDUCTION OF CELLULAR DEOXYRIBONUCLEIC ACID SYNTHESIS BY BOVINE ADENOVIRUS TYPE 3. *(E.)* Nakajima, K. (Inst. Virus Res., Kyoto U., Japan) and K. Hamada. *Jap J Microbiol* 17(2):123-128, 1973.

2038 GROWTH OF HERPES SIMPLEX VIRUS IN TRANSFORMED GLIAL AND NEURONAL CELLS IN TISSUE CULTURE: ULTRASTRUCTURAL STUDIES. *(E.)* Schwartz, J. (Mount Sinai Sch. Med., City U. New York, N.Y.) and T. S. Elizan. *J Neuropathol Exp Neurol* 32(3): 303-312, 1973.

2039 THE MYSTIQUE OF THE HERPESVIRUSES. *(E.)* St. Geme, J. W., Jr. (Harbor Gen. Hosp., Torrance, Calif.). *Calif Med* 118(6):;9-23, 1973.

2040 DEFECTIVE-INTERFERING PARTICLES OF SINDBIS VIRUS. I. ISOLATION AND SOME CHEMICAL AND BIOLOGICAL PROPERTIES. *(E.)* Shenk, T. E. (Rutgers Med. Sch., Piscataway, N.J.) and V. Stollar. *Virology* 53(1):162-173, 1973.

2041 FAST SEDIMENTING DEOXYRIBONUCLEIC ACID IN BACTERIOPHAGE T7-INFECTED CELLS. *(E.)* Strätling, W. (Friedrich-Miescher-Lab., Max-Planck-Society, Tübingen, Germany), E. Krause and R. Knippers. *Virology* 51(1):109-119, 1973.

2042 ALTERED PATTERN OF VIRAL RNA SYNTHESIS IN CELLS INFECTED WITH STANDARD AND DEFECTIVE SINDBIS VIRUS. *(E.)* Eaton, B. T. (Dept. Microbiol Immunology, Queen's U., Kingston, Ontario, Canada) and P. Faulkner. *Virology* 51(1):85-93, 1973.

2043 CHARACTERIZATION OF MURINE LEUKAEMIA VIRUS SPECIFIC DNA PRESENT IN NORMAL MOUSE CELLS *(E.)* Gelb, L. D. (Natl. Inst. Hlth., Bethesda, Md. J. B. Milstein, M. A. Martin and S. A. Aaronson. *Nature* [*New Biol*] 244(133):76-79, 1973.

See also:

* (Rev): 1805, 1808, 1809
* (Chem): 1847
* (Phys): 1913
* (Immun): 2049, 2051, 2055, 2056, 2059, 2060,
2061, 2064, 2065, 2066, 2069, 2072,
2080, 2095, 2104, 2105, 2107, 2116,
2118, 2120, 2124, 2125, 2127
* (Epid-Biom): 2149

2044 MALIGNANT LYMPHOMA AND AMYLOIDOSIS FOLLOWING GRAFT-VERSUS-HOST REACTION. *(E.)*

Tanaka, M. (Nagoya U. Sch. Med., Japan), T. Shibata, Y. Ito and A. Kunii. *Nagoya J Med Sci* 35:151-155, 1973.

The graft-versus-host reaction was induced in 18-wk-old male (C57Bl/6J x DBA/aJ)F$_1$ mice by 2 or 4 weekly i.p. injections of approximately 80 x 10^6 male C57Bl/6J or C57Bl/Na mouse lymphoid cells. Some of the recipient mice were treated with amethopterin. The majority of mice dying soon after injection of parental lymphoid cells showed signs of runt disease; most animals recovering from the disease survived and were alive at the age of 24 months, at the end of the experiments. Histological studies of 94 mice revealed lymphomatous changes in ten and a moderate to extensive amyloidosis in 22 mice, with no abnormal changes in 15 untreated control mice. Nine out of ten lymphomas were Type B, only one being reticulum cell sarcoma, Type A. Five out of nine Type B lymphomas were associated with amyloidosis. Comparative analysis of mortality rates due to runt disease, and incidences of lymphoma and amyloidosis in each experimental group showed that the resulting lymphoma incidences were apparently indifferent to the severity of runt disease. The results suggest that the overt graft-versus-host reaction may not be a necessary precondition for increased lymphomagenesis in parental-F$_1$ hybrid chimeras and that factors other than the graft-versus-host reaction itself may also be involved.

2045 THETA ANTIGEN IN N-NITROSOBUTYLUREA LEUKEMOGENESIS OF THE MOUSE. *(E.)*

Hiai, H. (Aichi Cancer Ctr. Res. Inst., Nagoya, Japan), H. Shisa, Y. Matsudaira and Y. Nishizuka. *Gann* 64(2):197-201, 1973.

Changes in thymocyte susceptibility to anti-theta C3H serum in the process of N-nitrosobutylurea (NBU) leukemogenesis and the relationship between types of leukemia and the theta antigen were examined. Swiss mice of both sexes were continuously administered NBU dissolved in drinking water in a concentration of 0.04%, starting at 35 days of age, for 60 days. Groups of mice were killed at 15-day intervals and their thymus cells tested for susceptibility to anti-theta C3H serum by the cytotoxicity test. Leukemias of C57BL/6, 129, CBA, and BALB/c mice induced with NBU were also tested for the presence or absence of the theta antigen on their cell surface. In spite of the rapid decrease of the thymus weight after the start of NBU administration, the predominant cells in the thymus were small lymphocytes until the 45th day, but after the 45th day, small lymphocytes disappeared almost completely. Corresponding to these findings, no significant changes in thymocyte susceptibility to anti-theta C3H serum were noted until the 45th day. The first leukemia of thymic type was found on the 58th day. All 11 mice observed over 58 days developed thymic leukemias with a mean latency of 69.4 days. Susceptibility of the leukemic cells to the antiserum was reduced and showed marked variation. All the thymic leukemias possessed the theta antigen and non-thymic leukemias did not. Histological types of leukemias also corresponded well with the presence or absence of the theta antigen on the cell surface. Thymic leukemias consisted of relatively well-differentiated lymphocytic cells whereas non-thymic leukemias consisted of poorly differentiated cells. The former possessed the theta antigen and the latter did not. There were no exceptions. Therefore, a *sine qua non* classification may be possible for lymphatic leukemias of the mouse.

2046 STUDIES ON THE BINDING OF CONCANAVALIN A TO RAT LIVER MITOCHONDRIA. *(E.)*

Glew, R. H. (Sch. Med., U. Pittsburgh, Pa.), S. C. Kayman and M. S. Kuhlenschmidt. *J Biol Chem* 248(9):3137-3145, 1973.

The binding of concanavalin A (con A) to intact and detergent-solubilized fractions of rat liver mitochondria has been studied quantitatively using a turbidimetric agglutination assay and by a radioactive binding procedure employing [*acetyl*-^{3}H]con A. The reaction appears to involve oligosaccharide residues associated with glycoproteins of the mitochondrion since glycosides such as methyl-α-D-mannopyranoside and methyl-α-D-glucopyranoside, which are specifically bound by con A, will inhibit this interaction. Solvent extraction (10% water in acetone) did not affect the ability of mitochondria to bind [*acetyl*-^{3}H]con A. Based on gel filtration studies of Triton X-100 (1%) solubilized preparations of mitochondria, the majority (70%) of the con A-reacti components have molecular weights of 40,000 to 80,00 Since approximately 40% of the total [*acetyl*-^{3}H]con A-binding capacity can be demonstrated using intact mitochondria, it appears that a considerable number of binding sites are located on the outer surface of the mitochondrial outer membrane. Seventy per cent of the con A-reactive components are released in soluble form together with most (85%) of the intr cristate marker enzyme adenylate kinase by treatment with 0.108 mg of digitonin per mg of mitochondrial protein. The remainder of the con A-binding material is distributed equally between the inne and outer mitochondrial membranes. When the digitonin-solubilized intracristate space fraction is subjected to polyacrylamide gel electrophoresis five periodic acid-Schiff's base staining bands are observed. When the soluble, intracristate protein fraction is subjected to trypsin digestion or periodic acid oxidation, extensive destruction of [*acetyl* ^{3}H]con A-binding activity is observed. These observations indicate that con A may be a useful probe for investigating the function and distribution of glycoproteins in the mitochondrion.

2047 INDUCTION OF ACTIVE IMMUNE STATE BY MULTINUCLEATE TUMOUR CELLS IN MICE. *(E.)*

Lia S. K. (Dept. Anat., McMaster U., Hamilton, Ontario, Canada) and D. H. Carr. *Experimentia* 29(5):597-599, 1973.

Active immunity was induced in male A/Jax mice foll ing three injections over a 20 day period of Sendai virus-fused methylcholanthrene-induced syngeneic sa coma cells. Of 107 mice treated in this manner,

re than 70% did not develop tumors following re-
ated challenge with 1 x 10^5 live unfused sarcoma
lls over a three month period. All control mice
ed with tumors. Immune mice challenged with cells
om two syngeneic spontaneously arising parotid
livary gland myoepitheliomas developed tumors at
e same rate as did controls, indicating that
nunity to the sarcoma cells was specific. Immunity
uld not be induced by single unfused cells or by
ndai-induced interferon.

48 PRESENCE OF ANTIGEN RELATED TO THE CARCINOEMBRYONIC ANTIGEN IN FECES OF NORMAL ADULTS.

) Matsuoka, Y. (Osaka U. Med. Sch., Japan), M.
a, K. Takatsu and M. Kitagawa. *Gann* 64(2):203-206,
73.

racts from feces of normal adults prepared as a
ntrol in a clinical experiment were found to react
h antiserum to carcinoembryonic antigen (CEA)
racted from an adenocarcinoma of the colon. Then,
es of normal adults were extensively examined and
was found that 6 out of 7 extracts showed a
ong reaction with anti-CEA upon immunodiffusion.
of these normal adults were examined by X-ray
found to have no neoplastic changes in the di-
tive canal. Judging from the reactivities with
antiserum, it appears that feces of most normal
lts contain a fairly large amount of the antigen
ilar to the CEA extracted from colon carcinomas,
hough some fecal antigens showed partially var-
t specificity. It is suggested that the CEA
ecule may be composed of two antigenic moieties,
of which may be present only in extracts from
cerous tissues in digestive organs and the other
mon to both the cancerous and fecal antigens.
gin of the fecal antigen is unknown at present
no information about its chemical nature is
ilable yet.

49 IMMUNOGLOBULIN RESPONSE IN EXPERIMENTAL INFECTION WITH CELL-FREE AND CELL-
OCIATED MAREK'S DISEASE VIRUS.

(E.) Jakowski,
M. (Dept. Pathobiol., U. Connecticut, Storrs),
N. Fredrickson and R. E. Luginbuhl. *J Immunol*
(1):238-248, 1973

immunosuppressive effect of Marek's disease
pesvirus (MDV) on humoral antibody development
evaluated before the time of overt neoplasia.
-day-old line 7 chickens were tested for passively
uired MD agar gel precipitating (AGP) antibody and
ected intra-abdominally with cell-free or cell-
ociated MDV. Infected chickens were subsequently
culated with a sheep erythrocyte (SRBC) suspension
the humoral response to this antigen was evalua-
by following SRBC hemagglutination titer.
unosuppression of IgG anti-SRBC antibody was found
chickens infected with cell-associated MDV virus
ardless of whether maternal MD-AGP antibody was
sent. No significant suppression was found in
ilar chickens infected with cell-free MDV. IgM
i-SRBC antibody was unaltered in all of the
ected groups receiving cell-free or cell-associa-
MDV. At termination of the experimental period (40 days postinoculation MDV), MD-AGP antibody was found in all infected groups. The incidence of this antibody was lowest in birds negative for maternal MD-AGP antibody and infected with cell-associated MDV, reflecting the severe suppression of anti-SRBC antibody encountered in this same group during the experimental period. On the basis of these findings, humoral immunosuppression in MD appears to be a phenomenon associated with high doses of cell-associated virus. The effect primarily involves IgG antibody while IgM antibody appears to remain unaltered. Experimental conditions simulating more natural infection with a cell-free MDV inoculum were not found to alter significantly IgG or IgM antibody.

2050 STUDIES ON HISTOCOMPATIBILITY ANTIGENS AND TROPHOBLASTIC TUMORS.

(Ger.) Tatra, G. (2nd Gynecol. Clin. Inst. Immuno., Vienna U., Austria) and E. Wolf. *Arch Gynaek* 213(4):341-351, 1973.

ABO blood groups and human leukocyte antigen (HL-A) phenotypes were determined in 27 women with trophoblastic tumors (5 chorioepitheliomas and 22 hydatidiform moles) and in 26 of their husbands. The diagnosis was established on the basis of the clinical course and histological picture. Blood group B was found more frequently than expected in women with trophoblastic tumors. The distribution of the HL-A antigens was the same as in the general population of Vienna. None of the 26 couples investigated had the same HL-A phenotype. In only 2 patients with chorioepithelioma was it possible that they could have had and HL-A-compatible haplotype inherited from their fathers. Cytotoxic antibodies were found in the blood of only 6 of the 27 patients, but this may result from samples being taken some time after development of the disease.

2051 CELL-MEDIATED IMMUNITY AND ANTIBODIES TO *HERPESVIRUS HOMINIS* TYPE 1 IN ORAL LEUKOPLAKIA AND CARCINOMA.

(E.) Lehner, T. (Guy's Hosp. Med. Dent. Sch., London, England), J. M. A. Wilton, E. J. Shillitoe and L. Ivanyi. *Br J Cancer* 27(5): 351-361, 1973.

Cell-mediated and humoral immune responses to *Herpesvirus hominis* type 1 (HVH1) and *Candida albicans* were studied in 32 patients with leukoplakia, showing a histological spectrum of changes from epithelial keratosis (12) to acanthosis (13) and atypia (7) and in 13 patients with carcinoma. The results were ranked according to increasing values of stimulation indices of lymphocyte transformation to HVH1, and the corresponding macrophage migration inhibition indices, and complement fixing antibody titers of each patient were correlated. Most patients with epithelial atypia were clustered to that end of the spectrum which had the highest stimulation and migration indices to HVH1; this relationship was not evident with *C. albicans*. In patients with keratosis and acanthosis there was a significant lack of correlation between lymphocyte transformation and migration inhibition to both HVH1 and *C. albicans*. In carcinoma the indices of lymphocyte transformation and migration inhibition to HVH1 and *C. albicans* were depressed. Furthermore, a significant negative correlation was found between lymphocyte transformation and migration inhibition to HVH1, unlike the positive correlation in control subjects. Complement fixing antibodies to HVH1, HVH2, cytomegalovirus and adenovirus, and fluorescent antibodies to *C. albicans* failed to show a significant change in titer in any one group of subjects tested. The results suggest a cell-mediated immune defect in leukoplakia, with a dissociation between lymphocyte transformation and macrophage migration inhibition to HVH1 and *C. albicans* in cases of keratosis or acanthosis. A specific increase in cell-mediated immunity to HVH1 in leukoplakia with epithelial atypia and the sequential changes argue in favor of a possible participation of HVH1 in carcinomatous transformation of some leukoplakias.

2052 *IN VITRO* CYTOTOXICITY AND *IN VIVO* TUMOR ENHANCEMENT INDUCED BY MOUSE SPLEEN CELLS AUTOSENSITIZED *IN VITRO*.

Ilfeld, D. (Weizmann Inst. Sci., Rehovot, Israel), C. Carnaud, I. R. Cohen and N. Trainin. *Int J Cancer* 12(1):213-222, 1973.

The immuno-reactivity of C57BL mouse spleen cells previously sensitized *in vitro* against syngeneic fibroblasts or syngeneic 3LL Lewis tumor cells was investigated. The sensitized spleen cells were assayed *in vitro* to test their cytotoxicity against the 3LL tumor cells. In addition, spleen cells autosensitized against syngeneic fibroblasts were mixed with 3LL tumor cells and injected into syngeneic recipient mice in order to ascertain their influence on tumor growth. The *in vitro* assay indicated that lymphoid cells specifically sensitized against either fibroblasts or tumor cells were equally cytotoxic against the 3LL target cells. On the other hand, in the *in vivo* experiments lymphoid cells sensitized against syngeneic fibroblasts promoted the growth of the 3LL tumor. The 3LL carcinoma which was used as a target for autosensitized lymphocytes contains the normal cell antigens of the C57BL strain as well as tumor-specific antigens. It is probable that lymphocytes sensitized against self-antigens. It is probable that lymphocytes sensitized against self-antigens present on fibroblasts specifically recognize the same antigens on the tumor cells and consequently mediate a cytotoxic reaction against the target cells. In the case of direct sensitization against the 3LL tumor cells, recognition and triggering of reactive lymphocytes could have been caused by either normal cell antigens or by tumor-specific antigens, both types of antigens being present on the sensitizing monolayer. The present data do not permit a distinction between sensitization against normal self-antigens and tumor-specific antigens.

2053 REGRESSION OF POLYOMA TUMOR METASTASIS BY COMBINED UNBLOCKING AND BCG TREATMENT--CORRELATION WITH INDUCED ALTERATIONS IN TUMOR IMMUNITY STATUS.

(E.) Bansal, S. C. (Dept. Immunol. Pacific Northwest Res. Fdn., Seattle, Wash.) and H. O. Sjogren. *Int J Cancer* 12(1):179-193, 1973.

Immunotherapy, in the form of inoculations of "unblocking" antitumor sera alone or combined with BCG and splenectomy, was tried in a metastasis model in animals. W/Fu rats were exposed to progressively growing polyoma tumor isografts which were subsequen excised, and then rechallenged with an isograft of the same tumor as a "metastasis". The immunological manipulation was initiated at the time of rechalleng and, although it was not capable of preventing tumor outgrowth, it caused a complete regression of the outgrowing tumor nodules within a few weeks in 7 out of 7 and 5 out of 16 rats in two different experimen An attempt was also made to inhibit well-established spontaneous tumor metastasis to the lung by repeated inoculations of unblocking antitumor serum. Prolong survival was obtained in 4 out of 9 rats and one of these animals was free of tumor when killed after 4 months. The spread of the tumor to various organs was significantly restricted by the therapy, indicating partially inhibited tumor growth. An attempt was made to establish correlations between inhibiti of tumor growth *in vivo* and certain alterations in the antitumor immunity as analyzed by *in vitro* techniques. Disappearance of serum blocking activity and appearance of rat complement-dependent cytotoxicity in serum was seen in all rats with regressing tumors but not in the other animals, although they had received the same treatment. The lymphocyte cytotoxicity was increased to about the same extent in regressor and progressor rats. It may therefore be seen from these studies that tumor regressions occurred when the animals were treated so that: 1) the blocking activity disappeared from their serum, 2) the cytotoxic antibodies (with rat complement) appeared, and 3) their lymphocyte cytotoxicity was maintained or increased.

2054 BIOCHEMICAL, MORPHOLOGICAL, AND IMMUNOLOGICAL OBSERVATIONS OF LEUKEMIC RETICULOENDOTHELIOSIS.

(E.) Burns, C. P. (U. Iowa Col. Med., Iowa City), R. D. Maca and J. C. Ho *Cancer Res* 33(7):1615-1624, 1973.

The biochemical, immunological, and morphological characteristics of the abnormal mononuclear cells from four patients with leukemic reticuloendotheliosis are described. Two patients had a homogeneo population of mononuclear cells with elevated thymidine incorporation, normal uridine incorporation, suboptimal or delayed response to phytohemagglutinin and a characteristic ultrastructure. Glucose utilization by these cells was somewhat greater than that by normal lymphocytes, but considerably less than that by normal monocytes and macrophages. The leukemic cells from both of these patients had surface-bound immunoglobulin. In contrast, the disease of the other two patients was characterized by a heterogeneous population of mononuclear cells with normal thymidine and uridine incorporation, normal or increased response to phytohemagglutinin, and a paucity of surface-bound immunoglobulin. All four patients had low levels of serum muramidase. The aforementioned two patients had elevated leukocyte counts while the latter two patients had normal or low values. Attempts to establish long-term cultures were unsuccessful. All four patients appeared to have lymphoproliferative processes.

5 RADIOIMMUNOASSAY OF MAMMALIAN TYPE-C POLYPEPTIDES. IV. CHARACTERIZATION OF WOOLLY (EY AND GIBBON VIRAL ANTIGENS. *(E.)* Parks, W. P. :l. Cancer Inst., Bethesda, Md.), E. M. Scolnick, C. Noon, C. J. Watson. *Int J Cancer* 12(1):129- , 1973.

approximately 30,000 dalton woolly and gibbon e-C viral polypeptides were characterized by ioimmunoassay. Antibody prepared in rabbits to woolly and gibbon "gs" protein show only low- el cross-reactions with other mammalian type-C al gs antigens and their antisera. The woolly gibbon approximately 30,000 dalton polypeptides highly related on the basis of both serum and igen titrations, confirming previous studies with viral reverse transcriptases. The woolly gs ay using a double antibody radioimmunoassay can ect less than one nanogram of purified woolly or oon viral protein, the same protein also reacts the interspecies of gs-3 radioimmunoassay indi- ing that the primate "gs" proteins also contain h species-specific and interspecies reactivities the same polypeptide. To establish that the woolly gibbon type-C viruses represented distinct iso- es with a close immunological relationship and to minate the possibility of laboratory contamina- n, original woolly and gibbon material was studied antigen. Both the original woolly monkey fibro- coma and two separate gibbon lymphoid tumors tained high levels of type-C interspecies and lly viral antigen by radioimmunoassay, indicating t natural tumors from distinct species of primates tain immunologically related viral antigen. Thus, findings in the present work suggest that previ- concepts of one immunological group of type-C us per species may not be directly applicable to mates.

5 CELL-MEDIATED IMMUNE REACTION AGAINST TUMORS INDUCED BY ONCORNAVIRUSES. III. DIES BY MIXED LYMPHOCYTE-TUMOR REACTION. *(E.)* ik, A. (St. Louis Hosp., Paris, France), E. Gomard, Plata and J. P. Levy. *Int J Cancer* 12(1):233-241, 3.

ne-way mixed lymphocyte-tumor-cell reaction (MLTR) applied to the study of anti-tumor cell-mediated unity in murine sarcoma virus (MSV)-infected b/c and C57Bl/6 mice. No stimulation of normal, -immune lymphocytes by tumor cells was observed. phoid cells which can be stimulated *in vitro* by ious tumor cells of the same antigenic specificity MSV-induced tumor cells were detected in the spleen lymph nodes of MSV-infected mice. These "responder ls" were only found before the appearance of the or, and at a time (3 to 7 days after MSV inocula- n) when no cytotoxic cells can be detected in the omium-51 release test (CRT). The reactivity of ponder cells declines rapidly and remains undetec- le thereafter, both when palpable tumors and cyto- ic cells are present and after tumor rejection, n none or very few CRT-killer cells are active. inverse relationship between responder cells in R and killer cells in CRT was also detected after ogeneic immunization. Two hypotheses are proposed explain this lack of correspondence between the two types of lymphoid cells. 1) MLTR responder cells and CRT killer cells are one and the same, but they correspond to two different phases of the immune cell development; and 2) MLTR responder cells and CRT killer cells are two distinct types of cells.

2057 CELL-MEDIATED IMMUNITY AND BLOCKING FACTORS IN PATIENTS WITH TUMOURS OF THE CENTRAL NERVOUS SYSTEM. *(E.)* Kumar, S. (Immunol. Lab., Manchester, England), G. Taylor, J. K. Steward, M. A. Waghe and P. Morris-Jones. *Int J Cancer* 12(1):194-205, 1973.

A detailed study of immune responses against central nervous system tumors was undertaken using 18 human glial tumors (9 astrocytomas, 7 medulloblastomas, and 2 ependymomas). The results of the study show evidence of the presence of cellular and humoral immunity in the individuals bearing these tumors. Lymphocytotoxicity was specifically directed against the neoplastic component of the tumor. The source of tumors was either fresh biopsy material or specimens that had been stored in a freezing medium containing DMSO in liquid nitrogen for up to 7 yr. These tumors were confronted with autologous or homologous lymphocytes derived from: 1) patients with the same type of tumor; 2) patients with other histological types of tumors; and 3) healthy donors. The results indicate that the lymphocytotoxicity is tumor type-specific rather than "brain tumor" specific. The degree of lymphocyte-mediated tumor-cell killing did not decrease with the duration of apparent clinical cure. Higher degrees of tumor-cell killing were found in the autochthonous as compared with the allogeneic combinations. Serum samples from 3 of 15 patients with central nervous system tumors had blocking activity. Of the 12 patients without blocking factor, one had clinically active disease and 11 had been completely symptom-free for up to 14 yr. Absence of blocking factor, therefore, would seem to be to some extent associated with apparent cure.

2058 RAPID DEVELOPMENT OF A CANCER-LIKE ANTIGEN IN NORMAL TISSUE *IN VITRO*. *(E.)* Field, E. J. (Newcastle Gen. Hosp., Newcastle upon Tyne, England), D. Hughes and E. A. Caspary. *Br J Cancer* 27(6):427-433, 1973.

The antigenic properties before and after various periods in culture of normal human embryonic tissue with respect to lymphocytes from cancer patients were studied. Freshly excised human embryonic tissue used as antigen to test lymphocytes from a cancer bearing patient gave a "normal tissue" result of about 10% in the macrophage electrophoresis migration (MEM) test. When it was grown *in vitro* and then used as an antigenic stimulant to cancer lymphocytes, a "cancer-like" result (about 15%) was produced. These new antigenic determinant(s) similar to those associated with cancer basic protein appear rapidly (within 5.5 hours) *in vitro*. Cultures of "normal" cells are thus antigenically different from the same cells in context *in vivo*. In view of the short time needed for these antigenic determinants to appear, it is suggested that the changes may be associated with the onset of mitotic division.

2059 RELATION OF CHROMOSOME 4 (LINKAGE GROUP VIII) TO MURINE LEUKEMIA VIRUS-ASSOCIATED ANTIGENS OF AKR MICE.

(E.) Ikeda, H. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), E. Stockert, W. P. Rowe, E. A. Boyce, F. Lilly, H. Sato, S. Jacobs and L. J. Old. *J Exp Med* 137(4):1103-1107, 1973.

The locations of genes coding for the expression of three murine leukemia virus (MuLV)-associated antigens—G_{IX} (cell-surface antigen), GCSA (Gross cell-surface antigen), and gs (group specific viral antigen)—have been determined from results of segregating crosses of young AKR mice (an Fv-1^{a} strain) with different Fv-1^{b} mouse strains. The results indicate that the genes specifying or controlling the expression of all three antigens are located in linkage group (LG) VIII of chromosome 4 of the AKR mouse. The data strongly indicates that the locus specifying G_{IX} is about 19 map units from the *Fv-1 (Gpd-1)* region. Whether the gs phenotype is an expression of virus titer regulated by *Fv-1* or an independent mendelian trait governed by a discrete locus in LG VIII could not be determined. The G_{IX} and GCSA phenotypes of the AKR backcross segregants were invariably associated, indicating a close linkage between their controlling genes. The G_{IX}-determining gene in LG VIII of AKR mice is apparently equivalent to *Gv-1*, which controls expression of the same antigen in strain 129 mice, but which in 129 mice is located in LG IX. Since the estimated distance of *Gv-1* from the *H-2* locus in 129 mice is considerable (37 units), the possibility of pseudolinkage cannot be excluded in this case.

2060 TWO DISTINCT ANTIGENIC COMPONENTS IN AN EPSTEIN-BARR VIRUS-RELATED EARLY PRODUCT INDUCED BY HALOGENATED PYRIMIDINES IN NON-PRODUCING HUMAN LYMPHOBLASTOID CELLS.

(E.) Sugawara, K. (Sch. Med., Hokkaido U., Sapporo, Japan) and T. Osato. *Nature [New Biol]* 243(128):209-210, 1973.

Epstein-Barr virus (EBV) early antigen (EA) production was studied in nonproducer NC-37 lymphoblastoid cells following stimulation with 5'-bromodeoxyuridine (BUdR) or 5'-iododeoxyuridine (IUdR). Two distinct antigenic components of EA, diffuse (D) and restricted (R), previously demonstrated in abortively infected cells, could be demonstrated in both EBV infected and IUdR- or BUdR-treated cells by indirect immunofluorescence using specific antisera. It is suggested that the repressed EBV genome might be fully responsible for the complete early viral functions which could be associated with EBV carcinogenesis.

2061 CONTROL OF GROUP-SPECIFIC ANTIGEN SYNTHESIS BY THE DEFECTIVE GAZDAR MURINE SARCOMA VIRUS GENOME.

(E.) Sarma, P. S. (Natl. Cancer Inst., Bethesda, Md.), T. Log and A. F. Gazdar. *Virology* 52(2):568-573, 1973.

Previous experimental results had indicated that, unlike other presently known murine sarcoma virus (MSV) strains, the newly isolated Gazdar strain (Gz-MSV) could direct synthesis of the mouse Gs-1 antigen in the absence of demonstrable infectious murine leukemia virus (MuLV). To test this hypothesis, attempts were made to rescue the defective MSV genome and gs-1 antigen from Gz-MSV hamster tumor cells with a heterologous feline helper leukemia virus (FeLV). Infection of co-cultivated hamster tumor and feline embryo fibroblast (FEF) cell cultures with FeLV resulted in the rescue of a focus-forming virus with the properties of FeLV pseudotype of MSV [MSV(FeLV)]. This virus could transform feline embryo but not hamster embryo cells. Serological typing of the MSV(FeLV) envelope antigens showed the same specificity as the helper FeLV used in the rescue. The Gz-MSV(FeLV) pseudotype which was propagated in FEF cultures contained excess nontransforming FeLV and gave a two-hit focus titration pattern indicating a requirement for helper virus. The Gz-MSV (FeLV) pseudotype contained species specific gs-1 antigens of both mouse and cat C-type virus. These results thus support recent studies which concluded that defective Gz-MSV virions in hamster tumor cells have the genetic capacity to control synthesis of the mouse C-type virus gs-1 antigen.

2062 CELL-MEDIATED IMMUNITY TO TUMOR ANTIGENS IN PATIENTS WITH RENAL CELL CARCINOMA.

(E.) Cummings, K. B. (UCLA Sch. Med., Los Angeles, Calif.), J. B. Peter and J. J. Kaufman. *J Urol* 110(1):31-35, 1973.

Cell-mediated immune response against renal cell carcinoma was observed with a microassay technique in disposable tissue culture plates. Peripheral blood lymphocytes from 8 of 13 patients with renal cell carcinoma exhibited cytotoxic activity against renal cell carcinoma target cells. The tumor cells from four renal cell carcinoma tumor lines studied were susceptible to this cytotoxicity. Consistent cytotoxicity of high magnitude was observed in the autochthonous setting, whereas only 5 of 12 allogeneic combinations were cytotoxic. By contrast no cytotoxicity for renal cell carcinoma was observe in the control group which consisted of lymphocyte donors with tumors of histologic type different from renal cell carcinoma and normals. Tumor target cell other than renal cell carcinoma were not reduced by lymphocytes from patients with this disease. The data suggest that when speaking of a tumor-specific antigen for renal cell carcinoma, one is, in effect, speaking of a finite group of tumor-related antigens some of which cross-react. It is suggested that renal cell carcinoma does not have a single oncogeni stimulus but shares sufficient common antigenicity between tumor lines to permit cross-reactivity of reasonable magnitude in the allogeneic setting.

2063 HUMORAL AND CELLULAR IMMUNE FACTORS IN TH SYSTEMIC CONTROL OF ARTIFICIALLY INDUCED METASTASES IN C3Hf MICE.

(E.) Vaage, J. (Massachusetts Gen. Hosp., Boston). *Cancer Res* 33(8):1957-1965, 1973.

Active antitumor immune resistance was compared in different anatomical locations and the effectiveness of humoral and cellular immune factors in supres-

on of neoplastic growth was tested. All animals
ed in these experiments were 12-wk-old female mice
inbred strain C3Hf/Bu. Active and passive immune
sistance to implanted syngeneic sarcoma cells was
pressed more effectively in visceral organs reached
intravascular implantation than in i.m. and s.c.
plantation sites. The protective effect of immune
rum was also most evident when tumor challenge
s via vascular routes. One explanation for this
fect is that immune resistance factors are encoun-
red immediately and maybe also more abundantly by
mor cells injected intravascularly. Passive
ansfer of serum from tumor-cured as well as tumor-
aring donors gave resistance to unsensitized
blethally radiated recipients partially restored
th transferred normal lymph node cells. These
servations may indicate that immune destruction of
tigenic neoplastic cells is most effective in the
ood and that antitumor antibodies may be primarily
particularly effective in preventing metastatic
ssemination of tumor growth.

64 ANTIBODY REACTIONS TO HERPESVIRUS SAIMIRI
(HVS)-INDUCED EARLY AND LATE ANTIGENS (EA
D LA) IN HVS-INFECTED SQUIRREL, MARMOSET AND OWL
NKEYS. *(E.)* Klein, G. (Dept. Tumor Biol.
rolinska Inst. Stockholm, Sweden), G. Pearson,
Rabson, D. V. Ablashi, L. Falk, L. Wolfe, F.
inhardt and H. Rabin. *Int J Cancer* 12(1):270-
9, 1973.

me of the antibody responses to *Herpesvirus Saimiri*
tigen in natural and experimental hosts were com-
red with particular emphasis on antibody response
early and late antigens (EA and LA) in the course
acute infection in 3 primate species and in anti-
dy-positive, healthy squirrel monkeys. Vero cells
fected with *Herpesvirus saimiri* and EA and LA viral
tigens distinguished by inhibiting viral DNA syn-
esis with cytosine arabinoside (Ara C), or al-
wing it to proceed uninhibited until CPE was
sible. The difference in the antigenic specifi-
ty of EA and LA was confirmed by direct fluorescence
sts, in combination with blocking experiments.
ra that were anti-EA positive according to the in-
rect test were capable of blocking the EA staining
action with direct EA+ conjugates, whereas anti-EA+
- sera were unable to do so. Healthy adult
uirrel monkeys had antibodies to late antigens
A) but not to early antigens (EA), as judged by
munofluorescence tests. Isolation-reared, anti-
dy-free squirrel monkeys responded with anti-EA
d anti-LA antibodies after HVS inoculation. Un-
oculated, cage-mate squirrel monkeys became in-
cted by horizontal transmission and responded with
and EA antibodies approximately 2 months after
ing housed together with the infected animals.
multaneously isolated controls remained antibody-
ee. HVS-inoculated, tumor-bearing marmoset and
l monkeys developed antibodies to both LA and EA
the majority of the cases studied, although in
me owl monkeys there was only LA and no EA anti-
dy development. Two different patterns of EA
aining could be distinguished, trabecular and
nctate. There was a conspicuous difference between
e species with regard to the timing of antibody
velopment. Squirrel monkeys, the natural host species of the virus, but resistant to its oncogenic action, responded with antibody development within 14-17 days after inoculation. Marmoset and owl monkeys did not respond until 28-80 days. Conceivably, the natural host species has been selected for a high degree of genetic responsiveness against virus-determined antigens. This may be partly or entirely responsible for its resistance to the oncogenic effect of this virus.

2065 COMMON CELL-SURFACE ANTIGEN ASSOCIATED WITH MURINE AND FELINE C-TYPE RNA LEUKEMIA VIRUSES. *(E.)* Yoshiki, T. (New York Hosp.-Cornell U. Med. Coll., N.Y.), R. C. Mellors and W. D. Hardy, Jr. *Proc Natl Acad Sci USA* 70(6):1878-1882, 1973.

A new common cell-surface antigen associated with murine and feline C-type RNA leukemia viruses was demonstrated by the use of rabbit antiserum against feline leukemia virus and the indirect membrane immunofluorescence test. Common cell-surface antigen was found in all leukemias of all strains of mice tested, in normal lymphoid tissues of Gross-positive (high incidence of leukemia) mouse strains AKR, AKR/H-2^b, C58, and NZB, in cultures rat fibroblasts infected with Rauscher virus, in cultured feline fibroblasts infected with feline leukemia virus, and in spontaneous feline lymphosarcoma. The antigen was not demonstrable in normal adult and fetal tissues of Gross-negative mouse strains or in tissues and cultured fibroblasts derived from normal rats and normal cats. The immunoferritin study of murine leukemia cells revealed that the antigen was located on the cell surface in discrete areas; budding and C-type RNA viral envelope was not labeled as antigen site. The distribution of common cell-surface antigen on murine and feline leukemias, as well as on normal lymphoid tissues of Gross-positive mouse strains, indicates the presence of an antigen distinct from any cell-surface antigen heretofore shown to be associated with, or specified by, mammalian C-type RNA viruses. The hypothesis that the cells of many, perhaps all, vertebrates contain C-type RNA viral genetic information, usually in covert form but with inducible expression has been proposed elsewhere. Common cell-surface antigen is apparently an antigenic expression of this genetic information and possibly indicates incipient sites of virus synthesis.

2066 SPECIFIC ANTIGENICITY OF TUMORS AND IMMUNOLOGICAL TOLERANCE IN THE RAT INDUCED BY FRIEND, GROSS, AND RAUSCHER VIRUSES. *(E.)* Kobayashi, H. (Hokkaido U. Sch. Med., Sapporo, Japan), N. Kuzumaki, E. Gotohda, N. Takeichi, F. Sendo, M. Hosokawa and T. Kodama. *Cancer Res* 33(7):1598-1603, 1973.

The relationship of the transplantation antigens that are induced in the rat by Friend, Gross, and Rauscher viruses was studied. Adult WKA/Mk rats that had been given i.p. and s.c. injections of undiluted murine leukemia virus within 48 hr after birth were used as tolerant rats. Friend and Rauscher virus-tolerant rats (2- to 3-month old adult) rats received neonatal injections of Friend and Rauscher virus; Gross virus-tolerant rats were only 1-2 months old and had received injections of Gross virus at birth. It was found that several lines of rat tumors grew only in Friend, Gross, and Rauscher virus-tolerant rats. When the tumors were transplanted into the virus-tolerant rats that had received injections of the other types of murine leukemia virus, they grew initially, and then regressed. Tumors did not grow in normal rats. The same is true of the methylcholanthrene-induced tumors that had been given injections of murine leukemia viruses. It is suggested from these results that the transplantation antigen of tumor cells that were induced by infection with Friend, Gross, and Rauscher viruses may contain an antigen common to all three viruses and also an antigen that is individually specific, and that the host's immunological tolerance (induced by the neonatal injection of either Friend, Gross, or Rauscher virus) may consist of the tolerance common to all three viruses and, also, of the tolerance that is individually specific.

2067 DEMONSTRATION OF ANTIBODIES AGAINST 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED RAT MAMMARY TUMORS BY IMMUNOFLUORESCENCE.

(E.) Kellen, J. A. (Dept. Pathol. Chem., U. Toronto, Canada) and A. C.-H. Lo. *Oncology* 27(4):315-323, 1973.

The value and importance of immunofluorescent staining to detect antibodies against mammary tumors was studied. Tumor-specific antisera were prepared by immunizing rabbits with a non-particulate fraction of rat mammary tumors induced by three consecutive i.v. injections of 2 mg 7,12-dimethylbenz(a)anthracene on the 50th, 53rd, and 56th day of age to female Sprague-Dawley rats. After extensive absorption with lyophilized rat serum and organ preparations, the removal of precipitating antibodies to 'normal' antigens was confirmed by double-immuno-diffusion in agar gels and immunoelectrophoresis. The antiserum was conjugated with fluorescein isothiocyanate and sections from mammary tumors and normal organs were treated with this preparation. The sections were examined by darkground fluorescent microscopy and the specificity of the immuno-fluorescent stain was established by its limitation to tumor specimens. The results obtained suggest the ability of this approach to detect cancer cells in dubious histological specimens.

2068 CARCINOMA *IN SITU:* BEHAVIOR PATTERNS AND CYTOIMMUNOLOGY. INDUCED REGRESSION BY AN IMMUNOACTIVE COUPLED TUMOR PROTEIN ANTIGEN.

(E.) Ayre, J. E. (Natl. Cancer Cytology Ctr., Melville, N.Y.), R. Narvaez, J. J. LoGerfo, J. M. LeGuerrier and M. M. Modica. *Oncology* 27(4): 294-304, 1973.

The long-term effect of administration of a coupled tumor protein antigen (CTPA) was studied in two series of patients with *in situ* (stage 0) cervical carcinoma. Response was monitored by repeated studies using exfoliative cytological techniques. The CTPA was prepared from pooled multiple autologous tumor proteins and each patient received two or three intracutaneous inoculations. Combining the results of the two series of eight and six patients, resp., all 14 patients showed cytological evidence of varying degrees of regression over a 6-1 month period. Five patients showed total regression to benign states (e.g., chronic cervicitis, hyperkeratosis, dysplasia). The patients, who were followed for up to 3 yr, showed no signs of progression During treatment, all patients had shown a local delayed hypersensitivity response. There was no evidence of renal, hepatic, or blood toxicity. Lymphocytosis (30-60%) was consistently observed during CTPA administration.

2069 INTRACELLULAR LOCALIZATION OF MAMMALIAN TYPE C VIRUS SPECIES-SPECIFIC (gs-1) AND INTERSPECIES-SPECIFIC (gs-3) ANTIGENIC DETERMINANTS WITH THE INDIRECT IMMUNOPEROXIDASE TECHNIQUE AND LIGHT MICROSCOPY.

(E.) Girardi, A. (Wistar Inst. Anat. Biol., Philadelphia, Pa.), B. Hampar, K. C. Hsu, S. Oroszlan, E. Hornberger, G. Kelloff and R. V. Gilden. *J Immunol* 111(1):152-156, 1973.

The indirect immunoperoxidase staining technique was used for localizing by light microscopy the intracellular species-specific (gs-1) and interspecies-specific (gs-3) antigenic determinants in several murine and hamster type C virus-containing cell lines, and was compared to the immunofluorescent technique. The gs antigens were isolated by electrofocusing and specific antisera were prepared in quinea pigs and rabbits. The results from immunoperoxidase staining agreed with previously published results from direct immunofluorescence. The technique was specific for the particular gs antigen. Staining of infected cells was localized in the cytoplasm. No staining was observed in uninfected cells. Mixed cultures of infected and uninfected cells showed staining only of the infected cells. The results indicated that the immunoperoxidase test is more sensitive in detecting the gs-1 antigen than the immunofluorescence test. Both tests were equally sensitive in detecting the gs-3 antigen.

2070 CELL-MEDIATED AND HUMORAL IMMUNE REACTIONS TO HUMAN TUMOURS.

(E.) Baldwin, R. W. (Cancer Res. Campaign Lab., Nottingham, England), M. J. Embleton, J. S. P. Jones and M. J. S. Langman. *Int J Cancer* 12(1):73-83, 1973.

Peripheral lymphocytes from cancer patients were tested for immune reactivity against cultured target cells derived from carcinomas of colon, rectum, breast, kidney and lung, and a melanoma by microcytotoxicity in plastic plates. Lymphocytes from colon or rectum carcinoma patients were cytotoxic for allogeneic colon or rectum carcinoma cells in 61% of tests; no difference in reactivity could be detected between pre- or post-operative patients. Breast carcinoma patients' lymphocytes were cytotoxic for breast carcinoma cells in 50% of cases tested. Autochthonous lymphocytes were reactive against melanoma cells, but no lymphocyte cytotoxicity could be demonstrated on cells of a renal

rcinoma or a lung carcinoma. None of the lympho-
te preparations tested reacted positively against
lls of a tumor different from that of the lympho-
te donor, and none reacted against cells from
rmal colon. Complement-dependent cytotoxic
moral antibody was regularly detected against
east carcinomas, but only one sample of colon
rcinoma serum, from a post-operative patient, was
active against colon carcinoma cells. Sera from
lanoma patients likewise were only reactive
ainst melanoma cells in two post-operative cases.
om these observations on carcinomas of colon and
east and the limited tests with one melanoma cell
ne, the microcytotoxicity test was judged to be
itable for evaluating humoral factors inhibiting
mphocytes cytotoxicity to these tumors. The value
such tests in evaluating a patient's immunologi-
l responsiveness to his tumor is less clear; the
esent studies all indicate that positive lympho-
te cytotoxicity can be detected in tumor-bearing
tients as well as those clinically free of di-
ase.

71 A TUMOR-ASSOCIATED ANTIGEN IN HUMAN NEPHROBLASTOMAS. *(E.)* Burtin, P.
nst. Sci. Res. Cancer, Villejuif, France) and M. C.
ndron. *Proc Natl Acad Sci USA* 70(7):2051-2054, 1973.

tisera prepared against extracts of human nephro-
astomas (Wilms' tumors) allowed the characteriza-
on in these extracts, and in a few extracts of
mors of other organs, of a tumor-associated
tigen that is apparently absent from normal
ult and fetal tissues. The antigen, named W
for Wilms), is different from all previously
scribed antigens. W antigen was found in many,
t not all, the nephroblastomas studied. Negative
sults could be explained by a lack of sensitivity
the immunochemical methods. It is possible that
more sensitive technique, such as radioimmuno-
say, could have detected traces of W antigen in
re nephroblastomas, and perhaps also in control
gans, either adult or fetal. Fetal kidneys were
wever studied with special care and no W antigen
tected. Two hypotheses are presented for the
igin of W antigen: 1) that W antigen is synthesized
der the action of an exogenous and possibly viral
nome; and 2) W antigen is the marker of a passenger
rus which has a special tropism for nephroblastomas.

72 THE HORIZONTAL DEVELOPMENT OF CELL-MEDIATED TUMOR-SPECIFIC IMMUNITY IN ROUS
RCOMA VIRUS TUMORIGENESIS IN RATS. *(E.)* Jonsson,
(Inst. Pathol. U. Lund, Sweden). *Int J Cancer*
(1):162-168, 1973.

e horizontal development of lymphoid cell-mediated
ecific antitumor immunity was studied in R/F rats,
oculated with the Schmidt-Ruppin strain of Rous
rcoma virus (SR-RSV) during the neonatal period.
mph-node cells (LNC) were harvested from individ-
l animals on consecutive occasions and used in
lony inhibition tests for the detection of LNC-
diated immunity to the Rous-specific tumor-associ-
ted transplantation antigen(s). None of the rats
sted, which had been SR-RSV inoculated within 6
days after birth had any demonstrable LNC-mediated immunity when tested within 4 wk after virus inoculation. At the same time 68% of the rats SR-RSV-inoculated at a higher age disclosed a clear-cut immunity. In the former group sarcomas appeared after short latency periods (average 5 wk), while the latter group developed sarcomas with long latency periods (average 11 wk) or remained tumor-free. Also in tests performed later after SR-RSV-inoculation, rats remaining tumor-free or developing very late tumors tended to develop an LNC-mediated immunity more rapidly than rats with tumors appearing earlier. The majority of both tumor-positive and tumor-negative animals developed at some time an LNC-mediated immunity, which often persisted for several months in negative animals but sometimes disappeared in rats with a large tumor burden. The results indicate that the kinetics of the LNC-mediated antitumor immune response can be of importance for the development of primary Rous sarcomas.

2073 LYMPHOCYTE CYTOTOXICITY IN BLADDER CANCER, NO REQUIREMENT FOR THYMUS-DERIVED EFFECTOR CELLS? *(E.)* O'Toole, C. (Dept. Immunology, Wenner-Gren Inst., Stockholm, Sweden), P. Perlmann, H. Wigzell, B. Unsgaard and C. G. Zetterlund. *Lancet* (7812):1085-1088, 1973.

Purified peripheral lymphocytes from nine patients with transitional-cell carcinoma of the urinary bladder were studied in fractionation experiments on glass-bead columns. Two columns were used in parallel for each preparation. One contained beads coated with human IgG (Ig column), in the second column IgG was reacted with rabbit anti-human-Ig serum in excess (anti-Ig column). These columns have been shown to selectively remove lymphoid cells possessing surface immunoglobulin and/or receptors for immunoglobulin (Fc receptors) in human systems. After passage through the glass-bead columns, lymphocytes previously specifically cytotoxic for the tumor cells in question lost all such activity. This was observed in lymphocyte preparations obtained from five patients before treatment and from three after local radiotherapy to tumor. Passage of lymphocyte preparations through columns coated only with human immunoglobulin produced no change in existing cytotoxicity. Cytotoxicity was measured concurrently with comparable results in the microcytotoxicity assay and by chromium-51 release from labelled target cells. These results show that nonthymus-derived lymphoid cells are necessary for the *in vitro* expression of cell-mediated immunity to this tumor.

2074 MALIGNANT IMMUNOBLASTOMA: IMMUNOGLOBULIN SYNTHESIS AND THE PROGRESSION TO LEUKEMIA IN HETEROTRANSPLANTED ACUTE LYMPHOBLASTIC LEUKEMIA, CHRONIC LYMPHATIC LEUKEMIA, LYMPHOMA, AND INFECTIOUS MONONUCLEOSIS. *(E.)* Adams, R. A. (Harvard Med. Sch., Boston, Mass.), L. Pothier, E. E. Hellerstein and G. Boileau. *Cancer* 31(6):1397-1407, 1973

The heterotransplantation of cells from human subjects with overt lymphoid neoplasia or infectious mononucleosis into newborn Syrian hamsters treated with antilymphocyte serum results in serially transplantable malignant tumors. These tumors are composed of cells retaining human species-specific antigen at the cell surface, and hence represent progeny of the implanted human cells rather than tumor cells induced in the hamster by an oncogenic agent. The tumors appear to fall into two categories on the basis of their behavior in the hamster. Category A consists of tumors derived solely from subjects with acute lymphoblastic leukemia secondary to lymphosarcoma, which in the hamster progress to acute leukemia and apparently do not secrete immunoglobulins. Category B consists of tumors derived from all of the remaining diagnostic categories thus far investigated and from acute lymphoblastic leukemia in remission as well, which in the hamster secrete immunoglobulins of human type and do not progress to acute leukemia. The descriptive term "malignant immunoblastoma" is provisionally proposed for the category B tumors, to emphasize their experimental derivation as well as their relationship to cells of the immunoglobulin-secreting type. The hypothesis is advanced that the category A acute leukemias and the category B malignant immunoblastomas may be related to T and B lymphocytes, respectively. The oncogenicity in hamsters of directly injected peripheral blood buffy coat cells from subjects with infectious mononucleosis raises the possibility that this lymphoproliferative disease may be a benign, self-limiting form of lymphoma. These observations with infectious mononucleosis may provide inferential evidence for the existence in man of host-regulatory, cancer-preventing mechanisms of perhaps substantial importance.

2075 THE EFFECT OF SPLENECTOMY ON THE GROWTH OF SARCOMA AFTER SYNGENEIC TRANSPLANTATION IN MICE.

(Rus.) Shcherbakov, V. Ya. (Smolensk Med. Inst., USSR). *Biull Eksp Biol Med* 75(4):79-81, 1973.

The effect of splenectomy on the growth of syngeneic sarcomas A-3174 in F_1 hybrid mice (CBA x C57Bl/6) was studied. Splenectomy performed 1-2 hr or 10 days before or 3 days after s.c. inoculation of tumor cells significantly suppressed natural immunity to syngeneic tumors in recipient mice, and increased the death rate. Tumors appeared earlier only when splenectomy was performed 1-2 hours before or 3 days after inoculation of tumor cells. Splenectomy performed 10 days after injection of tumor cells did not have any effect on tumor growth.

2076 BINDING OF CONCANAVALIN A BY NORMAL AND TUMOR CELLS.

(Rus.) Gel'fand, V. I. (Protein Res. Inst., Pushchino, USSR). *Biull Eksp Biol Med* 75(4):82-84, 1973.

A hemadsorption method is proposed for evaluation of concanavalin A binding by monolayer cultures of tumor cells. The advantage of the method is that concanavalin A strongly agglutinates erythrocytes on tumor cells previously fixed with glutaraldehyde but not on normal cell cultures. Cultures of mouse sarcoma induced by polyvinylchloride, mouse embryonic cells transformed with SV40 virus or methylcholanthrene, and spontaneously transformed hamster embryonic cells obtained from tumors induced by Rous sarcoma, SV40, or polyoma viruses were purified, fixed with glutaraldehyde, treated with concanavalin A and guinea pig erythrocyte suspension, and then dried. Hemadsorption of erythrocytes on these cells was evaluated visually. The use of adsorption, rather than agglutination, on living cells increases the sensitivity of the reaction and rules out the possibility of artefacts being produced by treatment of living cells.

2077 CYTOTOXIC LYMPHOCYTES IN THE ABSENCE OF DETECTABLE ANTIBODY.

(E.) Berke, G. (Duke U. Med. Ctr., Durham, N.C.) and D. B. Amos. *Nature* [*New Biol*] 242(121):237-239, 1973.

Ascitic EL4 leukemia cells from C57Bl/6 male mice were injected i.p. into B6C-H-2 mice, a mutant strain. Immune peritoneal exudate and spleen cells cytotoxic *in vitro* to ^{51}Cr-labeled EL4 cells could be isolated from graft recipients which rejected their tumors (20/26 males and 23/38 females More than 90% of these cytotoxic cells were small-to-medium sized lymphocytes possessing the θ C3H antigen. No cytotoxic or agglutinating antibody against EL4 cells could be detected before, during, or after rejection of the grafts by B6C-H-2 mice. Moreover, no antibody was detected in the sera of mice after six weekly injections of EL4 cells. The phenomenon was not due to a peculiarity of the malignant cells since cytotoxic and agglutinating antibodies were produced by injecting EL4 cells into other incompatible mouse strains. The lack of antibody was also not due to the presence of serum blocking factors. These results indicate that the formation of antibody by a graft recipient is no a requirement for subsequent rejection of the graft.

2078 EPIDERMAL ANTIGENS IN EXPERIMENTAL KERATOACANTHOMA AND SQUAMOUS CELL CARCINOMA.

(E Muller, H. K. (Monash U. Med. Sch., Melbourne, Australia) and G. R. Flannery. *Cancer Res* 33(9):2181-2186, 1973.

Keratoacanthomas, squamous cell carcinomas, and squamous papillomas were induced in the skin of albino rabbits and male DA strain rats with 7,12-dimethylbenz(a)anthracene (DMBA). The rabbits were painted twice weekly for five months on the inner surface of the left ear auricle and on the left flank with 1% DMBA. The rats were painted once weekly for five months on their dorsal trunk skin and flanks with 1% DMBA. Antigenic patterns in these lesions were assessed by indirect immunofluorescence using (a) pemphigus vulgaris sera, which stain specifically th intercellular areas and cell membranes of stratified squamous epithelium, and (b) bullous pemphigoid sera which react specifically with the basement membrane zone of stratified squamous epithelium. Keratoacanthomas showed normal intercellular immunofluorescenc and the basement membrane staining was intact but frequently thickened. Squamous cell carcinomas mainly showed diminished or absent immunofluorescent staining with both sera, whereas squamous papillomas showed normal staining patterns. These findings sup rt previous observations of depletion of tissue-ecific antigens in malignant neoplasia. The exces-ve bullous pemphigoid antigen observed in the kera-acanthoma may reflect the hyperplastic activity of is lesion. Histological assessment of the basement mbrane using silver impregnation demonstrated frag-ntation and loss of basement membrane reticulin in e squamous cell carcinomas and areas of reticulin ickening in the keratoacanthomas. However, the eas of reticulin change did not correlate with al-red basement membrane antigenicity and it is likely at the bullous pemphigoid antigen is produced by e overlying epidermis.

079 EFFECTS OF PYRAN COPOLYMER ON LEUKEMOGENESIS IN IMMUNOSUPPRESSED AKR MICE. *(E.)* irsch, M. S. (Harvard Med. Sch., Boston, Mass.), . H. Black, M. L. Wood and A. P. Monaco. *J Immunol* 11(1):91-95, 1973.

ale and female AKR mice which received twice weekly .p. injections of anti-lymphocyte serum (ALS) from ge 4 months had a higher, although not statistically ignificant, incidence of leukemia than control imals. Leukemias also appeared earlier in ALS-reated mice than in controls. Monthly i.p. injec-ions of Pyran copolymer markedly reduced the inci-ence of leukemias both in immuno-suppressed and in tact AKR mice. Animals treated with both ALS and ran had an incidence of leukemias intermediate etween that of animals treated with ALS alone or ran alone. Assays of spleens from control and reated mice by the cytotoxicity test revealed no ifferences at 5 and 6 months of age. These studies rovide further evidence for the absence of immuno-ogic tolerance to viral or tumor antigens in AKR mice d for the efficacy of Pyran copolymer as a pro-ylactic agent against virus-associated oncogenesis immunosuppressed hosts.

080 TUMOUR-ASSOCIATED TRANSPLANTATION ANTIGENS OF NEOPLASMS INDUCED BY A NATURALLY OCCUR-ING MURINE SARCOMA VIRUS (FBJ-MSV). *(E.)* Jones, . B. (Robert Jones, Agnes Hunt Orthopaedic Hosp., swestry, Shropshire, England) and M. Moore. *Br J ancer* 27(6):415-426, 1973.

BJ osteosarcoma virus (FBJ-MSV) isolated originally rom a spontaneously arising osteosarcoma in a CF1 ouse is the only known naturally occurring murine arcoma virus (MSV). The capacity of tumor cells ransformed *in vivo* by this agent to elicit specific ransplantation immunity in syngeneic hosts was in-estigated. The animals used in this study were ale and female CBA*T6T*6 and CBA(H) mice, maintained y brother-sister mating. A low level of resistance 10^4-10^5 cells) was consistently induced by implanta-ion of x-irradiated (15,000 rad) tumors or surgical xcision of developing subcutaneous grafts. By con-rast i.p. inoculation of virus containing cell free xtracts of FBJ-MSV sarcoma was a far less effective mmunization procedure. Confirmatory evidence for he antigenicity of these neoplasms was obtained in tests in which preincubation of tumor cells with lymphoid cells from specifically immune donors inhibited *in vivo* outgrowth of the FBJ-MSV cells in untreated syngeneic recipients. The induction of host resistance to FBJ-MSV cells by immunization with identical and independently-induced FBJ-MSV tumors established that FBJ-MSV cells possess common cell surface antigenic specificities in a manner analogous to those of experimental neoplasms induced by other oncogenic DNA and RNA viruses. The nature of the FBJ-MSV tumor-rejection antigen(s) could not be elucidated. The transplantation methodology as employed in this study was incapable of establishing whether the rejection phenomenon in this tumor-host system is mediated primarily by new virus-determined cellular antigens, or because FBJ-MSV cells release infectious virus, by components of the viral capsid incorporated into the cell surface membrane. The antigenic weakness of FBJ-MSV cells in syngeneic hosts is comparable with that of virus-induced murine leukemias of the Gross (G) or "wild" type subgroup to which category FBJ-MSV also belongs. These features suggest that FBJ-MSV exemplifies naturally occurring sarcomagenic viruses more closely than those of the Friend-Moloney-Rauscher-Graffi (FMRGr) subgroup which in general induce highly antigenic neoplasms.

2081 QUALITATIVE AND QUANTITATIVE INVESTIGATIONS ON THE REACTIONS OF NORMAL AND MYELOMA IgG WITH ANTISERA TO IgG. *(E.)* Morell, A. (Inst. Clin. Exp. Cancer Res., U. Berne, Switzerland), F. Skvaril and S. Barandun. *Clin Exp Immunol* 13(2):293-302, 1973.

Ten different antisera specific for the γ-chain of IgG were tested in gel diffusion tests and radioimmuno assays with isolated normal IgG and myeloma proteins of all four IgG subclasses. In Ouchterlony analyses, eight antisera revealed antigenic deficiency of several myeloma proteins compared to normal IgG. The most pronounced deficiency was associated with IgG4 myeloma proteins. Only two antisera gave reactions of antigenic identity between normal IgG and all myeloma proteins. Radial immunodiffusion plates were prepared with four of the antisera. When normal IgG and myeloma proteins were applied at the same concentrations (1 mg/ml), areas of the precipitin discs of most myeloma proteins were larger than the discs obtained with normal IgG. The largest discs were obtained with IgG4 myeloma proteins. The same four antisera were used for a solid phase radioimmunoassay. Binding of ^{125}I-labelled normal IgG to the insolubilized globulin fractions of the antisera was inhibited by normal IgG and myeloma proteins at various concentrations. Binding inhibition curves for normal IgG, IgG1 and IgG2 myeloma proteins were similar. However, pronounced differences in binding inhibition capacity were found among the IgG3 and IgG4 myeloma proteins. It is concluded that antisera to IgG can detect immunochemical differences between normal and myeloma IgG on the one hand and between various IgG myeloma proteins on the other, and that several myeloma proteins, particularly of the IgG4 subclass, lack some of the antigenic determinants of normal IgG and other myeloma proteins.

2082 REGULATION OF THE IMMUNE RESPONSE WITH ANTIGEN SPECIFIC IgM ANTIBODY: A DUAL ROLE.

(E.) Wason, W. M. (Argonne Cancer Res. Hosp., U. Chicago, Ill.). *J Immunol* 110(5):1245-1254, 1973.

The effect of IgM a-SRBC antibody given before and after antigen both *in vivo* and *in vitro* were compared. The latter approach permitted control over variables such as antigen distribution, antibody half life, and selection of circulating cells. IgM a-SRBC antibody administered before antigen produced an augmented response to a suboptimal dose of sheep erythrocytes (SRBC) in the mouse; however, this effect was not observed in two stains of rats tested. The same dose of IgM a-SRBC antibody which elicited an augmented response when given before the SRBC caused a significant suppression of the immune response when given 1 or 2 days after the SRBC. *In vitro* IgM a-SRBC antibody suppressed the response of cultured mouse spleen cells to all doses of SRBC, whether given before or after immunization. In the intact mouse, IgM a-SRBC antibody apparently augments the primary immune response to low doses of antigen by increasing the number of SRBC localized in the spleen. Antibody must be present at the time of injection of the antigen for this effect to occur. However, IgM antibody can also act to suppress antibody-forming cells after antigen localization has occurred. Thus IgM antibody appears to play a dual role in the regulation of the immune response in the mouse.

2083 RADIOIMMUNOELECTROPHORETIC BINDING ASSAY FOR THE DETECTION OF CARCINOEMBRYONIC ANTIGEN.

(E.) Coller, J. A. (Hosp. U. Pennsylvania, Philadelphia), R. W. Crichlow and L. K. Yin. *Cancer Res* 33(7):1684-1688, 1973.

A new radioimmunoassay (RIA) method, using radioimmunoelectrophoretic binding (REB) technique has been applied to the carcinoembryonic antigen system. In contrast to the RIA method that uses perchloric acid extraction, in the REB technique the requirement for lengthy and possibly biologically degrading procedures on relatively large volumes of sera has been eliminated. It involves simultaneous electrophoretic binding and separation with only one-five hundredth (10 µl) as much serum as is required by RIA; the assay can be completed in 2 hr rather than in 5 days. While the sensitivity of REB in the detection of serum carcinoembryonic antigen is similar, there appears to be a a different but related specificity, compared to the RIA method.

2084 TRANSPLANTABILITY AND ANTIGENICITY OF TWO SUBLINES OF THE TA3 TUMOR.

(E.) Sanford, B. H. (Massachusetts General Hosp., Boston), J. E. Codington, R. W. Jeanloz and P. D. Palmer. *J Immunol* 110(5):1233-1237, 1973.

Loss of specificity in the strain A mammary ascites carcinoma subline TA3-Ha appears to be related to decreased immunogenicity rather than to increased immunoresistance. Allogeneic hosts could be induced to reject potentially lethal inocula of TA3-Ha cells by addition of cells from the more specific TA3-St subline or by presensitization with skin grafts from strain A mice. The nonspecific TA3-Ha subline absorb markedly less activity from H-2 antisera than strain-specific TA3-St cells; however, the absorption capacity of nonspecific TA3-Ha cells for H-2 antisera was noticeably increased by lyophilization, suggesting decreased exposure of H-2 antigens at the cell surface of intact TA3-Ha cells. It is suggested that inaccessibility of histocompatibility antigens on the TA3-Ha cell may be related to presence of a unique sialoglycoprotein at the cell surface in this tumor subline. This mucinlike glycoprotein was fractionate and shown to contain galactose, N-acetylgalactosamine N-acetylglucosamine, and N-acetylneuraminic acid in the approximate molar proportion 4:2:2:1. Amino acid analysis showed a high concentration of serine and threonine in the protein core. With the strain-specific TA3-St subline, lyophilization increased H-2k antigenicity while decreasing H-2d activity. The magnitude of these changes, however, was much less than that observed with either H-2d or H-2k antigenicity after lyophilization of TA3-Ha cells.

2085 SPECIFIC ANTIGENS IN HUMAN ALVEOLAR CELL CARCINOMA.

(E.) Nordquist, R. E. (Oklahoma Med. Res. Fdn., Oklahoma City). *Cancer Res* 33(7):1790-1795, 1973.

Immunofluorescence and immunodiffusion techniques were used to study tumor-specific antigens of several continuous cultures established from human alveolar cell carcinomas. Previous studies had revealed the presence of virus-like material in the nucleus of these tumor cells. Immunodiffusion tests using specific antisera prepared in rabbits and absorbed to normal cellular antigens resulted in a single precipitation line against the original tumors, tumor cell cultures, and infected WI38 and BHK-21 cells but not against normal tissues or control tissue cultures. Diffusion detectable antigens could be recovered from the medium of tumor cell cultures and infected indicator cells after the third or fourth passage. Tumor cells from all patients tested contained the antigen which could be detected in the cytoplasm of malignant cells by immunofluorescence. No fluorescence was observed in normal human lung cells, lung tumor cells of other types, or nonneoplastic diseased lung cells.

2086 PROTECTION OF ADULT MICE AGAINST TUMOR CHALLENGE BY IMMUNIZATION WITH IRRADIATED ADULT SKIN OR EMBRYO CELLS.

(E.) Bendich, A. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), E. Borenfreund and E. H. Stonehill. *J Immunol* 111(1):284-285, 1973.

Female adult C57BL mice inoculated with three weekly s.c. injections of isogeneic X-irradiated adult skin or 17-day embryo cells were protected against subsequent challenge with cells from an isogeneic methylcholanthrene-induced tumor. Half of 10 mice immunized with embryo cells and one-fifth of 10 mice immunized with adult skin developed no tumors. The tumors in the remaining animals developed slowly and some regressed. Mice injected with irradiated adult liver, spleen, kidney, or lung cells developed rapidly, progressively growing tumors.

37 A TUMOR ANTIGEN IN TISSUE CULTURES DERIVED FROM PATIENTS WITH HODGKIN'S EASE.

(E.) Long, J. C. (Huntington Mem. Hosp., ton, Mass.), A. C. Aisenberg, M. V. Zamecnik l P. C. Zamecnik. *Proc Natl Acad Sci USA* 70(5): 0-1544, 1973.

llets obtained from supernatant fluids of mono- yer cultures of cells from patients with Hodgkin's sease were fractionated by isopycnic density sedi- ntation. Material in a peak of specific gravity 15-1.21 g/ml from two Hodgkin's disease cultures s used to immunize rabbits, and the antisera tained in this manner were reacted by agar-gel ffusion and immunoelectrophoresis with antigens om the purified peaks and the unfractionated pel- ts of centrifuged culture medium from all the ltures. The antisera reacted with material from of 10 lines derived from spleens of patients with dgkin's disease, 2 of 8 cell lines from histologi- lly negative spleens from patients with Hodgkin's sease and with 3 of 6 lymphoma cell lines not agnosed as Hodgkin's disease. The antisera did t react with 12 cell cultures prepared from normal ult and fetal spleen and thymus. The antigen from ltures from patients with Hodgkin's disease was t found in material sedimenting at lower specific avities; it resisted Tween-ether solubilization, d migrated as a single band by immunoelectrophore- s. The antigen was not found in disrupted, non- ltured tumor cells from patients with Hodgkin's sease, and an antiserum against noncultured, minced mor tissue did not react with the Hodgkin's disease ssue-culture material. No immunological relation- ip was found between the tissue culture antigen d Epstein-Barr, RD-114, or Rauscher murine leukemia ruses. The Hodgkin's disease antigen may be a mor-related antigen or a component of an oncogenic rus. It is also possible that this antigen is a n-viral tumor antigen which is masked in normal lls or even that it reflects a quantitative dif- rence in cellular constitution of normal and mphoma cultures.

088 IMMUNOGENICITY AND IMMUNOSENSITIVITY OF URETHANE-INDUCED MURINE LUNG ADENOMATA, IN ELATION TO THE IMMUNOLOGICAL IMPAIRMENT OF THE RIMARY TUMOUR HOST.

(E.) Menard, S. (Natl. Inst. tudy Cure Tumors, Milan, Italy), M. I. Colnaghi and . Cornalba. *Brit J Cancer* 27(5):345-350, 1973.

he depression of the immunological status of BALB/c ice treated during infancy with two different doses f urethane, alone or combined with cortisone, was valuated by counting the number of plaque forming ells at 30 or 50 days of age. The incidence of ung adenomatous nodules was directly related to the egree of immunological impairment at 50 days of age. wenty-seven lung adenomata were tested in an *in itro* system involving spleen cells immune against he same single tumor used as target cell. Eighty- ix percent of tumors in the most immunodepressed roup of mice were positive compared with 20-40% n the less immunodepressed groups. Syngeneic cross- eaction tests showed that non-immunogenic tumors ere immunosensitive since 66% positive tests were btained when target cells belonging to the less immunodepressed groups were tested with spleen cells of mice immunized with immunogenic adenomata. This suggests that tumor-associated antigens might be present on the cell membrane of all lung adenomata but in different quantities and that the quantitative expression was related to the level of immunocompetence of the primary tumor hosts.

2089 CELL-MEDIATED IMMUNITY TO HUMAN ORAL LEUKOPLAKIA DEMONSTRATED BY THE LEUKOCYTE MIGRATION TEST.

(E.) Roed-Petersen, B. (U. Hosp. Copenhagen, Denmark), J. Roed-Petersen and G. Bendixen. *Int J Cancer* 12(1):66-72, 1973.

The occurrence of cellular hypersensitivity against antigenic components of leukoplakial tissue in patients with leukoplakial lesions of the oral mucosa was studied using the leukocyte migration technique (LMT). Tissue extracts from 11 oral leukoplakias and from normal oral mucosa of the same patients were tested in the LMT against autologous leukocytes. Five patients showed inhibition of the migration with leukoplakial tissue and one showed inhibition with normal mucosa. The migration of leukocytes from matched controls was not influenced by the tissue extracts, which accordingly had no non-specific toxicity. It is concluded that oral leukoplakia is associated with a state of tissue-specific, cellular hypersensitivity against antigenic components of the leukoplakial tissue.

2090 CORRELATION BETWEEN VARIOUS CELL-SURFACE ANTIGENS INDUCED BY MURINE LEUKEMIA VIRUS IN THE RAT: SEROLOGICAL ANALYSIS.

(E.) Kuzumaki, N. (Hokkaido U. Sch. Med., Sapporo, Japan), N. Takeichi, F. Sendo, T. Kodama and H. Kobayashi. *Int J Cancer* 11(3):575-585, 1973.

Analysis based on the cytotoxicity test using antisera produced in rats indicated the presence of various cell-surface antigens on the tumors in inbred Wistar-King-Aptekman/MK (WKA/Mk) rats induced or artificially infected by Friend, Rauscher or Gross viruses. Each line of the tumor was found to have a common antigen, namely group-specific cell-surface antigen. A Gross type-specific and Friend and Rauscher type-specific cell-surface antigens were also detected, in keeping with previous observations in the mouse system. In addition, the present experiments strongly suggested that a Friend strain-specific antigen was detected only on the cell-surface of the tumors in WKA/Mk rats induced or artificially infected by Friend virus. These results were in parallel with the data of previously reported transplantation studies.

2091 AUTOIMMUNE DISORDERS AND MALIGNANT LYMPHOMA.

(E.) Jones, S. E. (Stanford U. Med. Ctr., Calif.). *Cancer* 31(5):1092-1098, 1973.

Autoimmune disorders of the Coombs' positive hemolytic anemia (AIHA) and idiopathic thrombocytopenic purpura (ITP) types were found in 11 (2.1%) of

515 patients with the non-Hodgkin's lymphomas. Four additional patients with Hodgkin's disease and similar disorders were included for analysis. In only two instances did the autoimmune disorder precede the lymphoma. AIHA was frequently associated with splenomegaly, constitutional symptoms, and, in eight of nine instances, far-advanced lymphomas of the diffuse histiocytic and diffuse histiocytic-lymphocytic types. In contrast, three of four patients who developed ITP had no evidence of active lymphoma at the onset of thrombocytopenia. The hemolysis of AIHA was most often controlled by splenectomy or corticosteroids with concurrent chemotherapy, but in all instances of either AIHA or ITP the prognosis clearly related to the underlying lymphoma and not the autoimmune disorder. A comparison of survival reveals it to be identical to that observed in patients with comparably advanced diffuse lymphomas without AIHA. No patient died of anemia, thrombocytopenia, or complications thereof. Based on this review, therapeutic guidelines for the management of these patients are proposed.

2092 ON THE MODE OF ACTION OF BCG. *(E.)* Mitchell, M. S. (Yale U. Sch. Med., New Haven, Conn.), D. Kirkpatrick, M. B. Mokyr and I. Gery. *Nature* [*New Biol*] 243(128):216-218, 1973.

The mechanism of action of Bacillus Calmette-Guérin (BCG) was studied in male CD-1 mice previously challenged by i.p. injection of a suboptimal immunizing dose of allogeneic L-1210 leukemia cells. Mice were inoculated i.v. with BCG 1-4 days after challenge with L-1210, and spleen cell-mediated immunity (CMI) was assayed by the *in vitro* cytotoxicity test 10-17 days after challenge using the DBA mastocytoma P-815Y as the target. BCG significantly augmented CMI in suboptimally immunized mice above levels in the controls. In addition, BCG alone induced almost as much CMI as did inoculation with an optimal dose of L-1210 cells. BCG produced a two- to four-fold increase in the number of splenic macrophages which, in turn, elaborated two to three times more lymphocyte-activating factor (LAF) than cells from normal controls. Spleen cells from BCG-treated mice incorporated eight to ten times more ^{3}H-thymidine than cells from untreated controls. The cells from BCG-treated mice, however, were relatively unresponsive to *in vitro* stimulation by phytohemagglutinin. BCG-induced augmentation of CMI can be ascribed at least in part to the stimulation of T cells via the macrophage, probably by the elaboration of LAF or a similar factor.

2093 RESTRICTION OF SPECIFICITY IN THE PRECURSORS OF BONE MARROW-ASSOCIATED LYMPHOCYTES. *(E.)* Lafleur, L. (Ontario Cancer Inst. U. Toronto, Canada), R. G. Miller and R. A. Phillips. *J Exp Med* 137(4):954-966, 1973.

The physical properties, differentiative potential, and kinetics of differentiation of the immediate progenitors of B lymphocytes (PB cells) in adult (C57BL/6 x DBA/2)F$_1$ mice sensitized to sheep red blood cells (SRBC) were studied *in vitro*. PB cells could be differentiated from mature B cells and hematopoetic stem cells on the basis of size, density, tissue distribution and sensitivity to cytotoxic antisera. Comparison of sedimentation profiles of cells stained with fluorescein-conjugated anti-Ig (Fl-anti-Ig) verified that PB cells sediment faster than B cells and that they have surface Ig receptors. PB cells were present with a frequency of about 5-10% of the nucleated cells in spleen and bone marrow, but were absent from the lymph node population. Comparison of the size of the total pool of PB cells, as determined by Fl-anti-Ig binding, with the population of PB cells responding the SRBC in a functional assay indicated that only 0.1% of the total population reacted, suggesting that PB cells are not pluripotent but are restricted in specificity. The differentiation to mature B cells *in vivo* involved at least one intermediate stage in which cells larger than mature B cells were active functionally. These B cells were present in irradiated mice less than one wk after transplantation of PB cells. By 20 days, however, most of the B cells were typical small lymphocytes.

2094 MONOSPECIFICITY OF BONE MARROW-DERIVED LYMPHOCYTES. *(E.)* Raff, M. C. (Dept. Zool., U. Coll. London, England), M. Feldmann and S. de Petris. *J Exp Med* 137(4):1024-1030, 1973. 1024-1030, 1973.

A fluorescent antibody technique was used to determine the specificity of the membrane binding sites of B lymphocytes from the spleens of mice immunized with polymerized flagellin (POL). Incubation of sensitized cells with POL followed by exposure to fluorescein-conjugated anti-POL in the cold and/or the presence of azide produced a ring distribution of fluorescence about the cell membrane. At 20 or 37 C, however, as many as 90% of the cells showed fluorescence confined to one pole of the cell. Thus, POL could "cap" immunoglobulin (Ig) on POL-binding cells. This observation, along with the finding that immunoabsorbent-purified anti-Ig could completely inhibit POL binding, indicated that the B cell receptors were Ig. The ability of POL to cap all of the Ig detectable by immunofluorescence on most POL-binding sites from both normal and immunized mice suggested that all of the Ig on an individual B cell had the same specificity. The most likely explanation of these findings is that individual virgin and primed B cells display only one type of receptor, implying that only one Ig variable region gene per light or heavy chain is expressed.

2095 HYBRIDIZATION OF A HUMAN LYMPHOID LEUKEMIA PERMANENT CELL LINE WITH MOUSE FIBROBLAST CELLS. *(E.)* LeBorgne de Kaouël, C. (Ctr. Res. Blood Dis., Paris, France), C. Billardon, J. F. Doré, D. Sylvestre, L. Marholev, N. V. Cong, D. Weil, J. Y. Picard, R. Rebourcet and J. Frézal. *Biomedicine* 18:59-69, 1973.

Attempts to fuse six permanent cell lines derived from leukemia patients with a permanent mouse cell line with the aid of UV-inactivated Sendai virus resulted in the establishment of one hybrid line (L-OL) which has been maintained in culture for 17 months. That L-OL cells were true human-mouse hybrids was confirmed by electrophoretic analyses of cellular enzymes and by karyotypic analysis. The modal chromosome number was 53 with apparent retention of six to eight human chromosomes. Electron microscopic observations indicated that L-OL cells had retained the morphological characteristics of the parental mouse cells. Herpes-type viruses were not visible by electron microscopy; however, intracisternal A-type particles and C-type particles were both seen and were apparently more numerous than in the parental mouse cells. Serologically, L-OL cells showed persistence of a membrane antigen associated with human lymphoblastic leukemia, as well as the appearance of detectable Burkitt's lymphoma membrane antigens after eight months in culture. It is thus possible that the L-OL hybrid cells contain the Epstein-Barr virus genome.

2096 DETECTION OF TUMORANTIGENS OF HYPERNEPHROID CARCINOMA. *(Ger.)* Bichler, K. H. (Urol. Clin., U. Marburg, Germany), C. Kirchner and F. Porzsolt. *Urol Int* 28(3-5):184-186, 1973.

Serum and urine of patients with hypernephroid carcinoma were studied by immunological, electrophoretic and gel chromatographical methods. Nine of 12 tumor urines contained up to 4 antigens, which could not be neutralized by normal urine or, with one exception, by normal serum. The antigens seem to be protein in nature. In agar gel electrophoresis they show up in the α_2-γ-globulin range. These tumor-specific proteins differed from α_1-, β- and γ-fetoproteins.

2097 ATTEMPTS TO PROPAGATE HUMAN TUMOR EXPLANTS IN MICE INOCULATED WITH HUMAN THYMUS ANTISERUM. *(E.)* Anigstein, L. (U. Texas Med. Branch, Galveston), D. M. Anigstein and F. J. Unterharnscheidt. *Texas Rep Biol Med* 31(20): 177-188, 1973.

Attempts were made to propagate tissue, from 31 human malignant tumors, in Swiss mice treated with heterologous thymus antiserum (human: HUTHAS; rat; RATHAS) produced in rabbits. Chloramphenicol was included in some of the protocols. Experimental procedures varied as to treatment before and after tumor explants. Although propagation of human tissue in mice failed, in general, it was observed that HUTHAS administered to mice influenced responses to implanted tumor tissue (principally central nervous system tumors) as to frequency and time of nodule formation at explant sites, and prolongation of tissue rejection time. HUTHAS-treated mice, particularly those given HUTHAS and chloramphenicol, responded 50-100% with early (3-6 days) nodule formation, and often retained palpable nodules beyond 60 days. It is assumed that the combined immunosuppressive properties of both agents together with nonspecific growth-enhancing properties of human tissue antiserum may be responsible for the prolongation of tissue rejection time, and early nodule formation. Mice treated with RATHAS gave results similar to those obtained in untreated mice or mice treated with normal rabbit serum; that is, 0-30% showed nodule formation in 5-7 days and rejection at 10-16 days. One mouse treated with HUTHAS developed a transplantable mouse tumor (MEN I) at the site of an explant from human meningioma tissue.

2098 EVIDENCE FOR THE LINKAGE OF THE I$_G$C$_H$ LOCUS TO A GENE CONTROLLING THE IDIOTYPIC SPECIFICITY OF ANTI-*P*-AZOPHENYLARSONATE ANTIBODIES IN STRAIN A MICE. *(E.)* Pawlak, L. L. (U. Illinois Coll. Med., Chicago), E. B. Mushinski, A. Nisonoff and M. Potter. *J Exp Med* 137(1):22-31, 1973.

Anti-*p*-azophenylarsonate (anti-Ar) antibodies raised in strain AL/N mice and in the congenic strain BALB/c.AL/N IgC$_H$ BC9 (BC9) share a cross-reacting idiotypic determinant whereas antibodies raised by a similar procedure in BALB/c mice do not. Since the BC9 stock was derived by introgressively backcrossing the IgC$_H$ complex locus of strain AL/N mice onto the BALB/c background, these findings indicate that genes controlling the anti-Ar idiotype are linked to the IgC$_H$ locus, which controls allotypic markers in the Fc regions of the heavy chains of mouse immunoglobulins. The anti-Ar idiotype was absent on anti-Ar antibodies induced in many other mouse strains. On the average, the anti-Ar antibodies of congenic and AL/N mice were quantitatively equivalent in their ability to displace I-labeled anti-Ar antibodies of A/J mice from their anti-idiotypic antibodies. Since idiotype must be a function of V-region sequences, these results suggest a close linkage of the V$_H$ and C$_H$ genes. The cross-reactive idiotype was found in nearly all F$_1$ mice (C57BL x A/J or BALB/c x A/J) tested, indicating that the failure to observe the idiotype in the BALB/c or C57/BL strains was not due to the presence of a suppressive factor.

2099 *IN VITRO* LYMPHOCYTE RESPONSE TO PHYTOHEMAGGLUTININ AND POKEWEED MITOGEN IN HODGKIN'S DISEASE. AN ELECTRON MICROSCOPIC AND FUNCTIONAL STUDY. *(E.)* Cohnen, G. (Dept. Med., U. Essen, West Germany), S. D. Douglas, E. König and G. Brittinger. *Cancer* 31(6):1346-1353, 1973.

Possible abnormalities in the circulating lymphocyte populations in Hodgkin's disease were studied by incubating lymphocytes from eight patients with Hodgkin's disease with phytohemagglutinin (PHA) or pokeweed mitogen (PWM) over seven days. Thymidine incorporation into DNA and ultrastructural features of transformed cells were studied. Response to these mitogens was either normal or diminished and/or delayed. In seven patients lymphocyte response to PHA was paralleled by a corresponding response to PWM. PHA-transformed lymphocytes showed fine structural features similar to transformed normal cells. After PWM stimulation, blast cells and plasmacytoid cells in various stages of differentiation were observed. The number of transformed cells corresponded to the magnitude of thymidine incorporation and in cultures with normal PWM response, plasmacytoid cells occurred with almost normal frequency. If the specificity of the mitogens is as postulated, then patients with Hodgkin's disease do not have a selective loss of T lymphocytes. Furthermore, the findings suggest that in some patients there may be a functional impairment of both T and B lymphocytes.

2100 DEFICIT OF SPECIFIC THYMUS-DEPENDENT LYMPHOCYTES IN TRANSPLANTATION TOLERANCE IN THE RAT. *(E.)* Elkins, W. L. (Dept. Path., U. Pennsylvania, Philadelphia). *J Exp Med* 137(4):1097-1102, 1973.

Sex chromosome markers were used to identify the origins of proliferating lymphocytes in local renal graft-vs-host reactions (GVHR) induced by cells from intact and thymectomized donor rats (Lewis-Lewis/Brown Norway) in which transplantation tolerance had been abolished adoptively. A deficit of specifically reactive lymphocytes, while tolerance was in effect, was revealed by the continuing absence of autochthonous specifically reactive cells after tolerance was abolished in adult thymectomized chimeras. These findings are consistant with Burnet's hypothesis of the cellular basis of tolerance, but apply only to T lymphocytes of donor origin which constitute the normal proliferating population in GVHR.

2101 INTERACTION OF HISTOCOMPATIBILITY (HL-A) ANTIBODIES AND COMPLEMENT WITH SYNCHRONIZED HUMAN LYMPHOID CELLS IN CONTINUOUS CULTURE. *(E.)* Ferrone, S. (Scripps Clin., Res. Fdn., La Jolla, Calif.), N. R. Cooper, M. A. Pellegrino and R. A. Reisfeld. *J Exp Med* 137(1):55-68, 1973.

HL-A antigenic expression was investigated on the surfaces of synchronized human lymphoid cells during different phases of their growth cycle using, as criteria, sensitivity to lysis in the complement-dependent cytotoxicity test, ability to activate the complement effector system, and quantitative absorption of HL-A alloantisera. No cell cycle-dependent differences in cell-surface HL-A antigen expression were detected by any of the above tests. The constancy of HL-A antigen expression throughout the growth cycle suggests that these cell surface markers are an essential part of membrane cytoarchitecture and could play a critical role in determining the normal function of the cell membrane.

2102 REGULATION OF THE SECONDARY ANTIBODY RESPONSE *IN VITRO*. II. CHEMICAL PROPERTIES OF AN ANTIBODY INHIBITORY MATERIAL (AIM) PRODUCED IN ANTIGEN-STIMULATED RABBIT LYMPH NODE ORGAN CULTURE. *(E.)* Ambrose, C. T. (Harvard Med. Sch., Boston, Mass.). *J Exp Med* 137(6):1369-1392, 1973.

The chemical properties of an inhibitor of antibody synthesis (AIM) found in serum-free medium of rabbit lymph node organ cultures undergoing a secondary response were studied. AIM, which could also be extracted from lymph node fragments as late as 4 wk after initial culturing, was most active in inhibiting the early productive phase of the secondary *in vitro* response (i.e., during the second wk). AIM was immunologically nonspecific since that isolated from cultures undergoing a specific given immune response could inhibit the same as well as different responses. Ultrafiltration studies indicated a molecular wt for AIM of 10,000-50,000 daltons AIM was not an antibody and could be separated from antibodies in the cultures by CM-cellulose chromatography. AIM was insensitive to protease digestion; however, inhibitory activity was rapidly and dramatically decreased upon RNase digestion. The susceptibility to RNase raised the possibility that the immunological system under study may be regulated by a species of RNA.

2103 SUBCELLULAR LOCALIZATION OF MURINE HISTOCOMPATIBILITY ANTIGENS IN TUMOR CELLS. LOCALIZATION OF H-2 ANTIGENS. *(E.)* Molnar, J. (U. Illinois, Sch. Basic Med. Sci., Chicago), G. Klein and S. Friberg. *Transplantation* 16(2):93-102, 1973.

Subcellular localization of murine histocompatibility antigens (H-2) was studied in the ascitic forms of the SEWA sarcoma ($H\text{-}2^s$ specificity), YAC lymphoma ($H\text{-}2^a$), and the TA3/Stockholm and TA3/Hauschka ($H\text{-}2^a$) carcinoma sublines. Live cells were radioiodinated specifically at their cell surfaces by the lactoperoxidase method and the protein-bound radioactivity was used as a quantitative marker for the cell membrane content at each subcellular fraction. The H-2 antigens were assayed quantitatively by cytotoxic inhibition tests. The results showed that the H-2 antigens are almost exclusively localized in the cell membrane. The TA3/Hauschka cells are characterized by a low expression of H-2 antigen sites available for antibody binding on the cell surface of intact cells. Following homogenization, however, all of the antigenic sites became available, reaching about one-third of the level characteristic for the fully antigenic TA3/Stockholm line. The H-2 antigen of the TA3/Hauschka cells was entirely restricted to the cell membranes as well, and it must be therefore concluded that its reactivity is impaired by the steric arrangement in the living cells.

2104 IMMUNO-ELECTRON MICROSCOPIC STUDIES ON CELL SURFACE ANTIGENS OF FRIEND VIRUS-INDUCED TUMOR IN THE RAT. *(E.)* Kodama, T. (Hokkaido U. Sch. Med., Japan), N. Kuzumaki and N. Takeichi. *Gann* 64(3):274-276, 1973.

The virus-induced cell surface antigen and the histocompatibility antigen on the cells of a transplantable Friend virus-induced tumor in the rat (RFT) were examined by the indirect immuno-ferritin method. Both antigens were present on the RFT cell surface, but absent on the envelope of Friend virus morphologically identified as type-C virus particles which were produced from the RFT cell membrane.

105 IMMUNOLOGICAL STUDIES OF TUMORS INDUCED BY HIGH ONCOGENICITY VARIANTS OF THE ROUS VIRUS ADULT MICE. *(Rus.)* Kryukova, I. N. (N. F. maleya Inst. Epid. Microbiol., Moscow, USSR), I. B. ukh and O. V. Babkova. *Biull Eksp Biol Med* (9):83-86, 1972.

e presence of the membrane-specific antigen (MSA) d other immunological features of tumors, induced adult mice by recently isolated, highly oncogenic riants of the Karr-Zilber strain (K-Z) of Rous sarma virus (RSV) were studied and compared with those tumors induced in newborn mice by the Schmidt-ppin strain. Transplantation and immunofluoresence tests indicated that there were no appreiable immunological differences between the tumors vestigated. Apparently, the increased RSV morigenicity is not associated with inhibition f cell membrane antigen synthesis.

106 SERUM COMPLEMENT LEVEL IN HODGKIN'S DISEASE. *(Fr.)* Hamladji, R. M. (Inst. asteur, Algiers, Algeria), M. Belhani, J. runberry, M. Benabadji and P. Colonna. *Nouv Rev Fr ematol* 12(5):673-681, 1972.

otal activity of serum complement (C) and of the $_3$ component were determined in 35 patients with odgkins disease (13 women and 22 men; 28 in relapse nd 7 in remission). Significantly higher levels f C (>125 CH 50 U in 10, 100-125 CH 50 U in 8) and f C_3 (>190 mg/100 ml in 13, 164-190 mg/ml in 7) ere found in patients than in controls (98 CH 50 U nd 164 mg/100 ml resp.). The values, especially of , usually increased during relapse and decreased uring remission. Significant correlations were bserved between C and fever, WBC, α_2-globulin evels, elevated ceruloplasmin levels and diameter f tuberculin skin reaction (induced by injection f 10 U of tuberculin). Similar correlations xisted between C_3 and plasma fibrinogen, serum ron, ceruloplasmin, α_2-globulin levels and diameter f tuberculin skin reaction. The possible ignificance of elevated levels of C and C_3 in odgkins disease is briefly discussed.

107 EFFECT OF VARIOUS INHIBITORS ON THE EXPRESSION OF POLYOMA VIRUS-INDUCED SURFACE ANTIEN IN BHK21 CELLS. *(E.)* Birg, F. (Res. Unit, Natl. nst. Hlth. Med. Res., Marseille, France) and G. eyer. *J Gen Virol* 20, Pt. 1:89-95, 1973.

tudies were made of the effects of various inhibitors f macromolecular synthesis on the appearance and isappearance of S antigen in polyoma virus abortivey infected BHK21 hamster cells. Drugs which block NA synthesis (5-bromodeoxyuridine, 5-fluorouracil, ytosine arabinoside, mitomycin C) also blocked ppearance, but could not prevent disappearance, of antigen as detected by immunofluorescence. Actiomycin D, on the other hand, had no effect on ppearance of S antigen but effectively prevented ts disappearance. Cycloheximide delayed and puroycin inhibited appearance of S antigen, possibly y their indirect effect on DNA synthesis which was nhibited by 40-50%.

2108 ALTERATION OF BINDING SPECIFICITY OF MURINE MYELOMA PROTEIN-315 TO 2,4-DINITROPHEHYL AND 2,4,6-TRINITROPHENYL LIGANDS. *(E.)* Hsia, J. C. (Fac. Med., U. Toronto, Canada), L. T. L. Wong, K. Pryse and J. R. Little. *Immunochemistry* 10(8):517-520, 1973.

Using spin-labeled hapten titration techniques, the thermodynamics of binding of a homogenous mouse immunoglobulin (the murine A-myeloma protein-315) to 2,4-dinitrophenyl (DNP) and 2,4,6-trinitrophenyl (TNP) ligands were investigated. It has previously been shown and confirmed in this study, that protein-315 has a higher affinity for TNP-ε-aminocaproate than for DNP-ε-aminocaproate. However, studies of the binding of the corresponding TNP and DNP derivatives of 3-amino-methyl-1-oxyl-2,2,5,5-tetramethylpyrrolidine spin label (methylene-SL(5)) resulted in a reversal of specificity of the protein-315 with a resulting higher affinity for the spin-labeled DNP than for the TNP ligand. These results are incompatible with the existence of a rigid site in which the ligands have the same positions and orientations. The possible explanations that can accommodate the experimental observations are: 1) the binding site is rigid and the ligands (DNP vs TNP) have different orientations within the site; or 2) the binding site is flexible and can fold differently around the ligands (that is, both the conformation of the combining site and the relative orientation of the ligand in the site have changed).

2109 RECEPTORS FOR IMMUNOGLOBULIN Fc IN HUMAN MALIGNANT TISSUES. *(E.)* Tonder, O. (U. Bergen, Sch. Med., Norway) and S. Thunold. *Scand J Immunol* 2(3):207-215, 1973.

Human malignant tumors were investigated for hemadsorption of erythrocytes coated with antibodies of various classes and specificities. Cryostat sections of ten malignant human tumors of various types strongly adsorbed erythrocytes sensitized with IgG antibodies. Normal spleen and lymph nodes showed medium strong and thymus weak reactions. The receptors in tissue have affinity for the Fc-portion of Ig since none of the tumors bound erythrocytes sensitized by $F(ab')_2$ fragments, and (phospho) lipids are common constituents. Contrary to IgG, IgA did not mediate hemadsorption while IgM was active with some of the tumors. An antiserum to a lymphoma inhibited receptor activity of all tissues. Spleen and tumor tissue removed this inhibiting activity. Tentatively, the malignant tissue seems to carry membrane receptor(s) similar in nature to receptor(s) on B lymphocytes.

2110 DEMONSTRATION OF CELL-MEDIATED AND BLOCKING IMMUNE RESPONSES TO TUMOR ANTIGENS IN CANCER PATIENTS WITH THE COLONY-INHIBITION-IN-GEL TEST. *(E.)* Cummings, F. J. (Division Biol., Med. Sci., Brown U., Providence, R.I.), G. H. Heppner, L. Stolbach and P. Calabresi. *Isr J Med Sci* 9(3):308-316, 1973.

2111 DISTRIBUTION OF MICROSOMAL ANTIGEN IN VARIOUS TYPES OF THYROID TUMOUR. *(E.)* Doniach, I. (London Hosp., England) and J. Perrin. *Clin Exp Immunol* 14(1):77-90, 1973.

2112 THE EFFECTS OF SURGERY ON LYMPHOCYTE TRANSFORMATION IN PATIENTS WITH CANCER. *(E.)* Watkins, S. M. (Roy. Free Hosp., London, England). *Clin Exp Immunol* 14(1):69-76, 1973.

2113 MEMBRANE DYNAMICS OF HL-A-ANTI-HL-A COMPLEXES OF HUMAN NORMAL AND LEUKAEMIC LYMPHOCYTES. *(E.)* Menne, H.-D. (Dept. Clin. Physiology, U. Ulm, Donau, Germany) and H.-D. Flad. *Clin Exp Immunol* 14(1):57-67, 1973.

2114 IMMUNOLOGICAL STUDIES OF THE RELATIONSHIP BETWEEN HL-A AND SPECIES-SPECIFIC ANTIGENIC SITES ON THE HUMAN LYMPHOCYTE SURFACE. *(E.)* Bachvaroff, R. (New York U. Med. Ctr., N.Y.) and F. T. Rapaport. *Transplantation* 15(6):586-593, 1973.

2115 LYMPHOCYTE INTERACTIONS IN THE PRESENCE OF MITOGENS. NEW POSSIBILITIES OF EXPLORING HISTOCOMPATIBILITY IN THE MOUSE AND MAN. *(E.)* Tridente, G. (Inst. Anatomical Path., U. Padua, Italy), L. Yung, G. Mok, E. Lupatin-Tridente and L. Fiore-Donati. *Transplantation* 15(6):614-618, 1973.

2116 EVIDENCE FOR MEMBRANE ANTIGEN(S) SPECIFIC FOR HUMAN B LYMPHOBLASTS. *(E.)* Thomas, D. B. (Middlesex Hosp. Med. Sch., London, England) and B. Phillips. *Clin Exp Immunol* 14(1):91-96, 1973.

2117 EFFECTS OF PROLONGED RES STIMULATION ON THE FREQUENCY OF MALIGNANT TUMORS AND LYMPHOMATOUS DISORDERS IN MICE. *(E.)* Isliker, H. (Swiss Inst. Exp. Cancer Res., Lausanne), C. Leuchtenberger and U. Kliem. *J Reticuloendothel Soc* 13(5):459-466, 1973.

2118 HOST RANGE CONVERSION OF THE MURINE SARCOMA-LEUKAEMIA COMPLEX. *(E.)* Yoshikura, H. (Inst. Med. Sci., U. Tokyo, Japan). *J Gen Virol* 19(Part 3):321-327, 1973.

2119 CELL-BOUND ANTIGLIAL IMMUNITY IN PATIENTS WITH MALIGNANT TUMORS OF THE BRAIN. *(E.)* Wahlström, T. (III Dept. Path., U. Helsinki, Finland), E. Saksela and H. Troupp. *Cell Immunol* 6:161-170, 1973.

2120 CONCANAVALIN A-INDUCED CHANGES OF TRANSFORMED CELL TRANSPLANTABILITY. *(Fr.)* Micco, Ph. De (Natl. Inst. Hlth. Med. Sci. Res., Marseille, France), C. De Micco-Pagis and G. Meyer. *C R Acad Sci [D] (Paris)* 276(7):1233-1236, 1973.

2121 RADIOIMMUNOASSAY OF ALPHA FETOPROTEIN. *(Ja*
Nakamura, M. (Sch. Med., Tohoku U., Japan)
Sawai and M. Abe. *J Nucl Med* 10(1):17-26, 1973.

2122 ABO BLOOD GROUPS IN PRIMARY CARCINOMA OF THE LIVER. *(E.)* Chew, B. K. (Dept. Med., U. Singapore), C. S. Seah, Y. W. Ong and L. Tan. *Aust NZ J Med* 3:129-130, 1973.

2123 ASSESSMENT OF STATISTICAL DISTRIBUTION OF THE NUMBER OF ANTIBODY-FORMING CELLS IN THE NONIMMUNE AND IMMUNIZED MICE. *(Rus.)* Pevnitsky, L. A. (N. F. Gamaleya Inst. Epidemiology, Microbiol. USSR). *Biull Eksp Biol Med* 75(5):122-124, 1973.

2124 FAILURE OF ACTINOMYCIN D TO INHIBIT THE INDUCTION OF SV40 VIRAL ANTIGEN IN BSC_1 TRANSFORMED CELLS. BRIEF REPORT. *(E.)* Margalith, M. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel), E. Margalith and N. Goldblum. *Arch Gesamte Virusforsch* 41(1-2):127-131, 1973.

2125 IMMUNOFERRITIN STUDIES OF MAREK'S DISEASE VIRUS DIRECTED INTRACELLULAR AND MEMBRANE ANTIGENS. *(E.)* Nazerian, K. (Poultry Res. Branch, U.S. Dept. Agriculture, East Lansing, Mich.) and J. H. Chen. *Arch Gesamte Virusforsch* 41(1-2):59-65, 197

2126 SELECTIVE CYTOTOXICITY OF PERITONEAL LEUCOCYTES. A PRELIMINARY REPORT. *(E.)* Holt
mann, O. A. (Roswell Park Mem. Inst., Buffalo, N.Y.)
B. A. Lisafeld and E. Klein. *J Med* 3(6):359-362, 197

2127 INCIDENCE OF ANTIBODY TO ENVELOPE ANTIGEN OF HERPES SIMPLEX VIRUS TYPE 2 AMONG PATIENTS WITH CERVICAL CARCINOMA AND MATCHED CONTROLS. *(E.)* Feorino, P. M. (Ctr. Dis. Control, Atlanta, Ga.) and E. L. Palmer. *J Infect Dis* 127(6):732-735, 1973.

2128 HL-A ANTIGENS IN RETINOBLASTOMA. *(E.)* Bertrams, J. (Division Med. Virology, Immunology, Eye Clin., U. Essen, Germany), P. Schildberg, W. Höpping, U. Böhme and E. Albert. *Tissue Antigens* 3(2):78-87, 1973.

2129 FUNCTIONAL EFFICIENCY OF ANTITUMOR CYTOTOXIC LYMPHOID CELLS. *(E.)* Wunderlich, J. R. (Natl. Inst. Hlth., Bethesda, Md.), W. J. Martin and J. MacDonald. *Isr J Med Sci* 9(3):317-323, 1973.

See also:

* (Rev) 1808, 1813
* (Chem): 1871
* (Phys): 1913
* (Viral): 1947, 1948, 1949, 1977, 1988,
1993, 2003, 2022

30 METABOLIC CONTROLS IN PRECANCEROUS LIVER- III. FURTHER STUDIES ON THE CONTROL OF PID SYNTHESIS DURING *N*-2-FLUORENYLACETAMIDE FEEDING.

.) Horton, B. J. (Dept. Animal Physiology, U. elaide, Australia) and J. R. Sabine. *Eur J Cancer* 1):1-9, 1973.

rious aspects of the control of cholesterol and tty-acid synthesis in the livers of 2 to 3 month d, male, hooded-Wistar rats fed the hepatocarcino- n, N-2-fluorenylacetamide, were examined both *in vo* and *in vitro*. Within 1-4 wk of beginning car- nogen treatment, "feedback" control of cholesterol nthesis (inhibition of cholesterol synthesis by olesterol feeding) was partially or completely st. Other controls over lipogenesis, however— sting inhibition of cholesterol synthesis and etary control of fatty-acid synthesis (fasting, feeding after fasting, high-fat diet)—were re- ined. The normal diurnal rhythm of hepatic choles- rol synthesis was missing from carcinogen-treated imals, but this was due to an alteration in their ily pattern of voluntary food intake. The loss of eedback" control of cholesterol synthesis was found be not due to the reduction in food intake that companied carcinogen feeding. This work is fur- er evidence in support of the hypothesis that a eakdown in "feedback" control of cholesterol syn- esis is an important feature in the development liver cancer. Two possible general explanations e offered: 1) for any given liver cell the carcin- enic process is dependent at some stage upon the ss of feedback control of cholesterol synthesis, d this process cannot proceed until control has en lost; and 2) a fundamental change which is es- ntial to the carcinogenic process invariably gives se to loss of feedback control of cholesterol nthesis, but as a side effect and not directly in- lved in tumor development.

31 PASSAGE OF CYTOPLASMIC LIPID INTO INTERPHASE NUCLEI IN PRENEOPLASTIC RAT VER.

(E.) Karasaki, S. (Notre-Dame Hosp., ontreal, Canada). *J Ultrastructure Res* (5-6):463-478, 1973.

ectron microscopic examinations of the prolif- rating cells in preneoplastic rat liver during o dye carcinogenesis revealed the presence of pid droplets both in the cytoplasm and nucleus. ntranuclear lipid inclusions were found mostly ree in the nucleoplasm but were occasionally sur- ounded by a single membrane. The inner membrane the nuclear envelope exhibited deep invagina- ions, forming intranuclear canaliculi, and were ften associated with the lipid droplets. The nterspace of intranuclear canaliculi was continu- us with the perinuclear space of the nuclear en- elope, which, in turn, was confluent with the isterns of the endoplasmic reticulum. Lipid roplets were also present in the lumens of both he endoplasmic reticulum and the nuclear en- elope. Since lipids were apparently synthesized n the endoplasmic reticulum, the confluent embrane-bound system reported here would facili- ate the passage of lipid material from the cyto- lasm into the nucleoplasm through an intact nuclear envelope. The accumulation of lipid droplets within the nucleus appears to represent an aberration of the cellular function perhaps associated with the neoplastic process. However, the cell type described here constitutes an actively proliferating cell population within preneoplastic liver parenchyma and the possibility exists that such a canalicular system would support metabolic activity in the nucleus by supplying the transport system for other macromolecules.

2132 GROWTH KINETICS OF TRANSPLANTABLE HEPATOMAS WITH DIFFERENT DEGREES OF DIFFERENTIATION.

(Rus.) Bogdanov, G. N. (Chem. Phys. Inst., Chernogolovka, USSR), V. M. Shmonina and N. M. Emanuel. *Biull Eksp Biol Med* 75(3):87-89, 1973.

The growth kinetics of three transplantable hepatomas with different growth rates and different degrees of differentiation was studied. Primary hepatomas were induced by o-aminoazotoluene in C3HA mice and were then transplanted s.c. into mice of the same line. Curves for the increase in the average volume and weight of growing transplanted hepatomas were S-shaped. The curves could be described by curves for first-order autocatalysis equations. In contrast to slightly differentiated hepatoma 22A, highly differentiated hepatomas had a significantly increased induction period and increased growth rates to the inflection point. Growth curves for hepatoma 22A also resembled curves for first-order autocatalysis equations.

2133 AN ANALYSIS OF SPONTANEOUS MAMMARY TUMORS IN DOGS.

(Rus.) Ponomar'kov, V. I. (Inst. Exp. Clin. Oncol., Moscow, USSR) and N. Ye. Osipov. *Vopr Onkol* 18(10):67-72, 1972.

Spontaneous mammary tumors were studied in 168 dogs by anamnestic data, clinical and laboratory examinations, biopsy and autopsy, and x-ray examinations. Tumors occurred most often in dogs between 8 and 10 yr old; the average age of dogs with tumors was 9.6 yr. There was an inverse relationship between the number of litters borne and the incidence of tumors: 51 dogs with mammary tumors had 0 litters, while one dog with a mammary tumor had 7 litters. A total of 113 dogs (67.3%) developed multiple encapsulated fibrous nodes of the mammary glands. Seventy percent of the ovaries of 104 dogs used in biopsy studies showed cystic degenerations. More than half of the dogs autopsied (number unspecified) had enlarged adrenals with nodular formations in the cortical layer, characteristic of adenomas. A total of 34 (56%) of 62 dogs studied at autopsy had metastases to other organs: 24 to the liver and 20 to the lungs. Seventy-five percent of the tumors studied were malignant; most were adenocarcinomas and 8.9% were mixed tumors (mesenchymal tumors with ectopic formations of malignant bones and cartilaginous tissue). Dyshormonal factors were important in the pathogenesis of mammary tumors in most dogs. Hyperestrogenism and suppressed and disturbed lactation were also implicated in tumor development. Adenomas of the adrenal cortex are apparently unrelated to mammary tumors, since they were found in female dogs with other tumors and in male dogs.

2134 HUMAN PRELEUKEMIA. IDENTIFICATION OF A MATURATION DEFECT *IN VITRO*. *(E.)*

Golde, D. W. (Cancer Res. Inst., U. California, San Francisco) and M. J. Cline. *N Engl J Med* 288(21): 1083-1086, 1973.

The proliferation and maturation of bone-marrow cells from four patients with preleukemia were studied *in vitro* with the use of a liquid culture technic. Viable cell counts and ^{3}H-thymidine labeling indexes were similar in preleukemia, acute myelogenous leukemia and normal marrow cultures. Cellular maturation, however, was delayed in the patients with preleukemia and delayed or absent in acute leukemia, resulting in the appearance of a morphologically homogeneous population of immature cells early in culture. Maturation was normal in cultures of bone marrow from three patients with refractory anemia and from three with chronic myelocytic leukemia. These data provide evidence supporting a concept of acute myelogenous leukemia as a primary disturbance of cellular maturation and indicate that a maturational defect can be observed in bone-marrow cultures from patients with preleukemia. The technic of bone-marrow culture in liquid medium may be useful in identifying the preleukemic state in man.

2135 THE METASTATIC POTENTIAL OF EXPERIMENTAL PULMONARY METASTASES. *(E.)*

Ketcham, A. S. (Natl. Cancer Inst., Bethesda, Md.), H. Wexler, and P. B. Chretien. *J Surg Res* 15(1): 45-52, 1973.

The potential of metastasizing tumors to develop further metastases was explored in a series of experiments using C57BL/6N mice. These studies demonstrated that immunization to an immunogenic methylcholanthrene-induced (MCA) tumor does not decrease the production of tumors by tumor cells circulating in the vascular tree. On the other hand, the survival of circulating tumor cells in the tumor-immune host is shown to be dependent upon factors which seem to be serum-mediated and which are produced by tumor immunization. Pulmonary metastases behave in a similar manner to the primary tumors in that tumor cells shed from either the metastases or the primary can colonize elsewhere, particularly when facilitated by serum factors which are shown to be elicited by tumor immunization. The experimental data, therefore, give no support for the hypothesis that circulating tumor cells are destroyed by specific host immunity.

2136 METABOLISM OF ^{3}H-TESTOSTERONE AND ^{3}H-5α-DIHYDROTESTOSTERONE IN THE NORMAL PROSTATE AND PROSTATE ADENOMA. *(Ger.)*

Becker, H. (Urol. Clin, U. Hamburg, Germany), H. Klosterhalfen and K. D. Voigt. *Urol Int* 28(3-5):350-355, 1973.

Thirty min before prostatectomy either ^{3}H-testosterone or ^{3}H-5α-dihydrotestosterone (^{3}H-5α-DHT) (doses unspecified) were injected i.v. into men with benign prostate hypertrophy. Radioactivity was counted in specimens of blood, skeletal muscle, prostate, and different parts of prostate adenomas, and the androgens were separated by thin-layer chromatography in several systems. In patients with benign prostate hypertrophy the endogenous plasma concentrations of testosterone and 5α-DHT were in the normal range. Testosterone and 5α-DHT accumulate to a greater extent in prostate than in muscle tissue. 5α-DHT is the main metabolite in prostate tissue and the 5α reduction depends on the predominant type of tissue present. In muscle no 5α reduction of testosterone occurs, whereas exogenous 5α-DHT is efficiently metabolized to androstanediols and androsterone.

2137 MONOCLONAL ORIGIN OF ACUTE TRANSFORMATION OF CHRONIC MYELOGENOUS LEUKEMIA. EVIDENCE FROM AN ACQUIRED SEX CHROMOSOME MOSAIC. *(E.)*

Motomura, S. (Fac. Med., Kyushu U., Fukuoka, Japan), K. Ogi and M. Horie. *Acta Haematol* 49(5):300-305, 1973.

In a case of chronic myelogenous leukemia with an acquired sex chromosome mosaic (45, XO, Ph^{1}/46, XY, Ph^{1}), variant cells, developed after blast crisis, were shown to have been originated from a 46, XY, Ph^{1}+ clone because of the constant presence of a Y chromosome in them. This strongly suggests that acute transformation has occurred selectively in a single clone.

2138 A STUDY OF THE MECHANISM BY WHICH ANTICOAGULATION WITH WARFARIN INHIBITS BLOOD-BORNE METASTASES. *(E.)*

Brown, J. M. (Stanford U. Sch. Med., Calif.). *Cancer Res* 33(6):1217-1224, 1973.

The effect of warfarin-induced anticoagulation on the ability of KHT sarcoma cells to metastasize in syngeneic C3H mice was studied. Addition of warfarin to the drinking water following tumor cell implantation into the thigh or prior to i.v. tumor cell inoculation significantly and effectively reduced the number of metastases to the lungs of mice autopsied 14 days later. The effect was not due to any cytotoxic action of warfarin. Simultaneou administration of vitamin K with warfarin produced complete inhibition of both the elevated prothrombin time and the decreased number of lung metastases. Experiments using ^{125}I-IUdR labeled tumor cells showed that the lower number of metastases in warfarin-treated mice was a result of an increased clearance of tumor cells from the lungs of anticoagulated mice between 6-24 hr after injection. These results are consistent with the hypothesis that warfarin reduced blood-borne metastases by inducing anticoagulation which prevents the formation of thrombi around metastatic cells, thus keeping these cells vulnerable to the normal cell-dependent defense mechanisms.

2139 GRANULE ASSEMBLY IN PRECURSORS OF HUMAN LEUKEMIA GRANULOCYTES. *(E.)*

Zeya, H. I. (Dept. Microbiol., U. North Carolina, Chapel Hill) and J. Laszlo. *Am J Pathol* 71(3):467-474, 1973.

The distribution of five granule enzymes and cationic proteins in the subcellular particles of pure human myeloblasts were examined and the incorporation of these enzymes in the granule-bearing precursors of chronic myelogenous leukemia was traced. The granule enzymes β-glucuronidase, myeloperoxidase and acid phosphatase were associated with light particles of varying densities that were separable from each other by means of zonal density gradient centrifugation. In more mature granulocytic cells of chronic myelogenous leukemia the three enzymes merged within a single group of denser particles; such particles were absent in myeloblasts. Myeloblast particles had two to three times higher activity of β-glucuronidase and acid phosphatase, but only one-tenth of the myeloperoxidase activity. Some of the cationic proteins and lysozyme were not found in leukemic myeloblasts but were present in particles of chronic myelogenous leukemia; alkaline phosphatase was absent from both types of leukemic cells. The merger and the movement of granule enzymes in late precursors of polymorphonuclear leukocytes implies that granulogenesis proceeds by progressive fusion of smaller subunits of enzyme-carrying particles into a larger and heavier granule entity.

2140 CELL ALTERATIONS OF THE GASTRIC MUCOSA IN BENIGN DISEASES AND MALIGNANT TUMORS. *(E.)* Martuzzi, M. (G. B. Morgani Hosp., Forli, Italy). *Pathol Eur* 7(3-4):263-272, 1972.

Histologic sections and/or cytologic smears of gastric washings were obtained from patients with gastric carcinomas, ulcers, polyps and gastritis and were examined to determine the incidence and alterations of cell types in benign and malignant disease. Areas of carcinoma showed a significant increase, compared with control tissues, of telophase and interphase cells with a decrease in the frequency of early prophase cells. The nuclear/cytoplasmic and nucleolar/nuclear ratios and the degree of chromasia were significantly greater in carcinomatous areas than in areas of benign disease. The cellular changes seen primarily in cases of carcinomas (79%) fit the criteria of Nieburgs for "malignancy associated changes" (MAC). MAC were noted in only 16% of the benign lesions.

2141 STUDIES ON THE INFLUENCE OF ULTRAVIOLET LIGHT ON INITIATION IN SKIN TUMORIGENESIS. *(E.)* Stenback, F. (U. Nebraska Med. Ctr., Omaha), H. Garcia and P. Shubik. *J Invest Dermatol* 61(2):101-104, 1973.

The effect of shortwave ultraviolet (UV) light applied once or 10 times on initiation by 7,12-dimethylbenz(a)anthracene (DMBA) in two-stage skin carcinogenesis was studied. Female Swiss mice, 8 wk old, were used in the experiments. Croton oil was used as promoter. The results showed that a single UV treatment increased the formation of benign tumors when given prior to initiation-promotion. The incidence of benign tumors decreased significantly when 10 doses of UV light were given after initiation. One explanation for this decrease is that DMBA might be oxidized to a noncarcinogenic derivative by UV. Another explanation is that UV light prevents tumor formation by destroying the latent tumor cells produced by the carcinogen. A few carcinomas and sarcomas occurred in mice given UV light 10 times and DMBA suggesting a summative effect of both agents, although UV light given 10 times is mildly tumorigenic by itself.

2142 CHANGES IN DNA CONTENT IN TUMOR CELL NUCLEI IN THE PROGRESSION PROCESS OF INDUCED HEPATOMA. *(Rus.)* Pozharisskii, K. M. (N. N. Petrov Res. Inst. Oncol., Leningrad. USSR), V. N. Shalumovich and Ya. D. Shaposhnikov. *Vopr Onkol* 9(3):73-80, 1973.

C3HA mouse hepatomas transplanted for 15 generations were studied cytophotometrically. Liver slices from two C3HA mice without cancer were used as controls. The average DNA content in the cells of hepatoma transplants decreased in the process of transplantation. Primary tumors had the greatest number of polyploid cells (16 n or over) and the ploidy of tumor cells decreased (to 2 n-10 n) from the second generation. In normal liver, stem-line cells were cells with 8 n, whereas the stem-line cells in primary hepatoma shifted to 16 n. The stem-line was distributed in 6 n-10 n from the second generation. Thus, there is greater homogeneity of hepatocytes in the progression of hepatomas.

See also:

* (Chem): 1852, 1856
* (Epid-Biom): 2146

2143 ASBESTOSIS AND LUNG CANCER IN OSAKA SENNAN DISTRICT. *(E.)* Sera, Y. (Natl. Kinki Central Hosp. Chest Dis., Sakai, Japan), K.-Y. Kang and K. Yokoyama. *Gann* 64(3):313-316, 1973.

A study of ten cases of lung cancer associated with asbestosis which were detected in the National Kinki Central Hospital for Chest Disease in Japan and surveys of asbestos workers in Sennan district in 1957 and 1972 are discussed. The ten cases of lung cancer were all males whose age varied from 32 to 77 years. Exposure duration of these patients was from 4 to 42 years; 8 of the 10 were cigarette smokers. Smoking appeared to increase the risk of developing lung cancer among asbestos workers compared with those who do not smoke. There were no women with asbestosis who died of associated lung cancer in this study. Histological examination of the ten cases revealed 3 adenocarcinomas and 7 squamous cell carcinomas. The first survey for the detection of asbestosis among workers in Sennan district was conducted in 1957 and identified 88 cases (10.8%). Thirty of these persons died within 15 years of the following causes: lung cancer (3), gastric cancer (3), pulmonary tuberculosis (2), other pulmonary diseases (12), cardiovascular diseases (5), and accidents and unknown causes (5). A similar survey, carried out in 1972, revealed one case of lung cancer as well as 53 cases of asbestosis (11.8%) out of 444 workers. The incidence of lung cancer associated with asbestosis on a world-wide scale is much higher than that encountered in this survey. Regarding this lower incidence and the rare cases of mesothelioma in Sennan, two potential possibilities are considered: 1) the use in these factories of chrysotile which has been described as less related to neoplasia and 2) the very heavy exposure of the workers to asbestos, owing to poor management against dust, which might mean that such patients died of asbestosis and other pulmonary diseases before a sufficient time has elapsed for the development of neoplasia.

2144 RESPIRATORY CANCER IN RELATION TO OCCUPATIONAL EXPOSURES AMONG RETIRED ASBESTOS WORKERS. *(E.)* Enterline, P. (Grad. Sch. Publ. Hlth., U. Pittsburgh, Pa.), P. de Coufle and V. Henderson. *Br J Ind Med* 30(2):162-166, 1973.

A group of 1348 men who completed their working lifetime in the asbestos industry and retired with an industry pension during the period 1941-67 was observed through 1969 for deaths. The average length of employment in the asbestos industry for these men was 25 yr and all had exposures to asbestos dust. In some instances these exposures were very high and continued for many yr. Mortality for these men after age 65 was 14.7% higher than for the entire population of U.S. white men living at the same ages and time periods. This excess was due almost entirely to cancer and repiratory disease. The cancer excess was chiefly due to respiratory cancer where mortality was 2.7 times the expected. The respiratory disease excess was entirely due to asbestosis. A time-weighted measure of asbestos dust exposure at the time of retirement was calculated for each man. This was made up of the summed products of dust levels for each job (expressed in mppcf) and yr at each level. This measure was directly related to the respiratory cancer excess at ages 65 and over, ranging from 1.7 tim expected for men with less than 125 mppcf-yr exposur to 5.6 times expected for men with 750 or more mppcf yr exposure. There appeared to be no direct relatio ship between asbestos dust exposure and respiratory cancer below 125 mppcf-yr. Important increments in respiratory cancer mortality apparently occurred som where between 100 and 200 mppcf-yr exposure. Separation of the effects of time from the effects of aver dust level on repiratory cancer mortality showed tha the contribution of each was about the same and that time-weighted measure of asbestos dust appears to be appropriate method for predicting respiratory cancer effects.

2145 EPIDEMIOLOGIC VARIABLES IN PROSTATIC CANCER. INCIDENCE RATES ON PROSTATIC CANCER RELATE TO VENEREAL DISEASE, COITAL FREQUENCY, NUMBER OF SEXUAL PARTNERS, AND USE OF CONTRACEPTIVES *(E.)* Krain, L. S. (Dept. Med., U. California, Los Angeles). *Geriatrics* 28(5):93-98, 1973.

Data from the California and Alameda County Tumor Registries on prostatic carcinoma were analyzed, and a special epidemiologic questionnaire was given to 136 patients with prostatic cancer. These were matched by age and race to 136 patients with noncancerous, nongenitourinary conditions from various Los Angeles area hospitals. There was a statistically significant ($p < 0.05$) excess of familially associated cancers, history of venereal disease, coital frequency, number of sexual partners before or after marriage, and use of contraceptive agents in the prostatic cancer cases as compared with the controls. Data on a relationship between socioeconomic class and prostatic cancer were inconsistent; data for all races showed no association; data for the nonwhite races showed a strong negative association with social class. Negroes have the highest incidence of prostatic cancer and Chinese and Japanese have the lowest. The five yr relative survival rates for prostatic cancer were 64% (1955 to 1969) for localize cases and 52% (1955 to 1969) for all stages of disease; although these rates have improved, the mortality for prostatic cancer continues to increase in California, implying an increase in incidence.

2146 GEOGRAPHICAL ASPECTS OF ORAL FOCAL EPITHELIAL HYPERPLASIA. *(E.)* Praetorius-Clausen, F. (U. Hosp., Copenhagen, Denmark). *Pathol Microbiol (Basel)* 39(4-5):204-213, 1973.

A survey of the literature indicates that oral focal epithelial hyperplasia is more common among Eskimos and American Indians than among other world population groups. The condition is a wart-like disorder of the oral mucosa which is probably contagious and may be caused by papovavirus. It is found primarily in American Indian children between the ages of 3 and 18, but is most prevalent in adult Eskimos over 30 yrs old. Electron microscopic examination reveals the presence of intranuclear papovavirus-like particles in degenerating epithelial cells. The condition is rare among Caucasians and

oes, and no cases have as yet been reported in
Far East. The prevalence of oral focal epithelial
rplasia among American Indian children is
lly less than 3.5%, whereas that among Eskimo
ts is usually greater than 10%.

DIETARY AFLATOXINS AND LIVER CANCER - A POPULATION BASED STUDY IN KENYA. *(E.)*
s, F. G. (Tropical Products Inst., London,
and) and C. A. Linsell. *Br J Cancer* 27(6):
-484, 1973.

relationship between aflatoxin ingestion by
and the incidence of liver cancer was investi-
d in the Murang'a district of Kenya. The study
designed to test the feasibility and methodology
elation to both the collection of samples and
analysis of mixed whole diets. The results show
atistically significant association between
toxin ingestion levels and liver cancer cases
he high attitude areas within Murang'a. A
ey of the dietary components included in the
tive aflatoxin diets as opposed to the negative
s revealed that maize, millet, sorghum, pigeon
, cabbage and yams appear to be included more
uently in the positive diets. The cabbage can
ably be ignored as it is normally included as a
h vegetable but the other staples are all
ary constituents in which aflatoxin contamina-
could be expected since they are often stored
r conditions which are not ideal. It is pointed
that correlation does not necessarily prove
ation. The Murang'a findings cover only a small
e of the possible association; further studies
be carried out in areas with a higher rate of
r cancer or higher dietary levels of aflatoxin
rder to test the strength and consistency of
association.

3 HISTOLOGY, SURVIVORSHIP AND RELATED FACTORS IN THE EPIDEMIOLOGY OF EYE
ERS. *(E.)* Keller, A. Z. (Dept. Med. Surg.,
Central Office, Washington, D.C.). *Am J*
demiol 97(6):386-393, 1973.

study characterizes 271 cases of eye cancers in
es by laterality, histology, race, age, survivor-
and residence. These histologically confirmed
es constitute a 7-year 100% patient sample from
veterans' hospitals in the United States. They
compared with male controls to whom they are
ched on age and hospital, and with all veterans
ing in 1960. Relative risks and incidence rates
eye cancers are determined. The following
ults are reported: 1) malignant melanomas are
more common in the left than in the right eye,
le squamous cell and intra-epithelial carcinomas
comparably frequent in both eyes; 2) the worst
vival experience is with intra-orbital mela-
as in contrast to that with conjunctival cancers
any type; 3) cumulative 5-year survival rates for
cases with localized cancers are similar to those
the male population counterparts of similar age
race; 4) the relative risk of malignant mela-
as is extremely low for Negroes; 5) incidence
es of eye cancers are significantly high above
age 59 and in regions of the south. These results emphasize that eye cancers select on factors which strongly relate to race, age, laterality and probably geography as well. They establish further that survivorship depends on the degree of spread, anatomical site and histological type of cancers within the eye.

2149 AN ANALYSIS OF SEROEPIDEMIOLOGICAL STUDIES OF HERPESVIRUS TYPE 2 AND CARCINOMA OF THE CERVIX. *(E.)* Rawls, W. E. (Baylor Coll. Med., Houston, Texas), E. Adam and J. L. Melnick. *Cancer Res* 33(6):1477-1482, 1973.

Seroepidemiological studies relating herpes simplex virus (HSV) infection and human cervical carcinoma are reviewed. Although results concerning the association of HSV infection and cervical carcinoma have varied depending on the methods used, many studies have indicated that more women with cervical carcinoma have antibodies to HSV-2 than do women without carcinoma. The results suggest that women with cervical cancer are infected earlier in life with HSV-2 than are control women. The differences in antibody activity between women with cancer and control women do not appear due to dissimilarities between groups in sexual promiscuousness. Controls with attributes of sexual and reproduction-associated factors similar to those of cancer cases still had less antibody activity to HSV-2 than did the cases. Various studies also show that increased anti-HSV-2 titers are associated with and, indeed, precede the onset of cervical dysplasia and carcinoma *in situ*. The occurrence of anti-HSV-2 antibodies among patients with other types of cancers has generally been found to be similar to that of control populations.

2150 BETEL CHEWING IN CEYLON. *(E.)* Senewiratne, B. (Dept. Med., U. Ceylon, Peradeniya) and C. G. Uragoda. *Am J Trop Med Hyg* 22(3):418-422, 1973.

Because the frequency of buccal and esophageal carcinoma may be associated with the common habit of chewing betel leaves, a survey was made of the habit in a large town in Ceylon and the results were analyzed in terms of sex, race, education, and smoking and drinking habits. A representative sample consisting of 1,127 people (aged 15 to more than 75 years) contained 29% who chewed betel. With regard to esophageal carcinoma in Ceylon, the incidence of betel chewing in 111 cases studied was 81.1%. Therefore, it is likely that betel chewing has some etiological relationship with esophageal carcinoma. There was a striking correlation between the incidence of betel chewing and the educational level with the poorly educated chewing betel more frequently and heavily. The prevalence of betel chewing among the Sinhalese, Tamils, and Moors was 26% to 30% but only 0.8% of the Burghers, with a western cultural background, chewed betel. This rarity of betel chewing among the Burghers is of special interest because bucal and esophageal carcinoma are very rare in this race. Betel chewing was nearly four times as frequent in female as in male Moors. In contrast to western countries, esophageal carcinoma is more common among females. Among all males betel chewing was more frequent among smokers and drinkers, similar in frequency to drinking but only half as frequent as smoking. If the high incidence of bucal and esophageal carcinoma in Ceylon is due to betel chewing, then it is possible that there is a high carcinogenic content in one or more ingredients of the chew.

2151 THE GROWTH RATE OF BRONCHOGENIC CARCINOMA: IS IT CONSTANT?

(E.) Weiss, W. (Hahnemann Med. Col. Sect. Pulmonary Dis. Serv., Phila. Gen. Hosp., Pa.). *Cancer* 32(1):167-171, 1973.

Six patients with bronchogenic carcinomas presenting as solitary nodules had from 7 to 9 serial chest roentgenograms which permitted measurement of tumor diameters over a long enough period of time to give an indication of the constancy of the growth rate. Growth curves were plotted on semilogarithmic and arithmetic coordinates. The findings suggested exponential growth in 3 of 6 cancers, but the curves for the other three cases did not provide evidence for constancy of growth rate. The sparse data in this series and in the literature are insufficient to permit generalization on the shape of growth curves for bronchogenic carcinoma. The limited information available suggests that some cancers have a constant growth rate during the small terminal segment of their life spans that can be observed but the proportion of cases showing such constancy is uncertain.

2152 GASTRIC CARCINOMA IN NEW ZEALAND. SOME EPIDEMIOLOGIC-PATHOLOGIC ASPECTS.

(E.) Kubo, T. (Kyushu U., Fukuoka, Japan). *Cancer* 31(6):1498-1507, 1973.

An epidemiologic-pathologic study was made of gastric carcinoma in a defined area of New Zealand with a population of 532,731. There were 1063 male and 575 female patients with gastric carcinoma in the period 1950-1970 inclusive. A further group of Maori patients was identified from throughout New Zealand; this totaled 186 male and 112 female patients in the period 1953-1969, inclusive. Despite definite differences in incidence and mortality rates, the histologic appearance of the New Zealand series showed no significant difference when compared with Japanese, Korean, and American series. This suggests that some factor is operating in different countries as a gastric carcinogen.

2153 ORAL CONTRACEPTIVES AND VENOUS THROMBOEMBOLIC DISEASE, SURGICALLY CONFIRMED GALLBLADDER DISEASE, AND BREAST TUMOURS: REPORT FROM THE BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAMME.

(E.) Greenblatt, D. J. (No affiliation), H. W. Ory and M. Levy. *Lancet* (7817):1399-1404, 1973.

Forty-three female patients with "idiopathic" venous thromboembolism, 212 women with gall-bladder disease, and 121 female patients with surgically confirmed breast tumors who were seen at 24 Boston area hospitals were evaluated in an attempt to identify associations between commonly used drugs and these diseases. Based on estimates of the local population, women aged 20-44 yr who used oral contraceptives had an 11-fold greater risk of developing venous thromboembolism than did the control population. Similar estimates indicated a two-fold greater risk for gallbladder disease among users of oral contraceptives. Oral contraceptive users, however, did not have a higher risk than controls for developing breast cancer. At each age-level studied, the risk for developing benign breast tumors was actually less than that for the control population. All patients studied had used low-dose estrogen preparations. The facts that the population studied was young (20-44 yr) and that long-term oral contraceptive users were not included precluded a categorical statement concerning the relation between breast cancer and oral contraceptives.

2154 TEMPORAL ASPECTS OF OCCUPATIONAL BLADDER CARCINOGENESIS.

(E.) Hoover, R. (Harvard Sch. Publ. Hlth., Boston, Mass.) and P. Cole. *N Engl J Med* 288(20):1040-1043, 1973.

Temporal aspects of occupationally induced bladder cancer were estimated with data from a case-control study in eastern Massachusetts. A total incidence series of men with bladder cancer were the cases, and an age-matched sample of men selected at random from the general population were the controls. The younger a man is when he begins employment in a hazardous occupation, the higher his risk of bladder cancer. Men first employed in a hazardous occupatio by the age of 25 have a risk 2.4 times that of men never so employed. Virtually no excess risk is apparent for men first employed in such occupations after the age of 25. Risk of occupational bladder cancer is greatest 41 to 50 years after beginning employment in a hazardous occupation, the "latent period." There is no change in risk with increasing duration of employment in such occupations. However, it appears that increased duration of employment in a hazardous occupation is associated with a shortene latent period.

2155 PRIMARY CARCINOMA OF THE LIVER: A STUDY OF 282 CASES IN UGANDAN AFRICANS.

(E.) Anthony, P. P. (Middlesex Hosp. Med. Sch., London, England). *J Pathol* 110(1):37-48, 1973.

Two hundred and eighty-two cases of primary carcinoma of the liver in Uganda Africans were studied, with particular attention given to the histology of liver-cell and bile-duct carcinomas, to differences in their natural history and to results of serological testing for alpha fetoprotein (AFP). Liver cell carcinoma was a tumor of younger males and was commonly associated with cirrhosis. Four main histological types were distinguished: 1) the hepatic type; 2) the pleomorphic type; 3) the adenoid type; and 4) the rare clear-cell type. In nearly half of these tumors there were no metastases present at death, which was often the result of rupture of the liver with massive intraperitoneal hemorrhage. By contrast, intrahepatic bile-duct carcinoma was a tumor of older people of both sexes and showed no association with cirrhosis. Most cases were well to moderately differentiated mucus-secreting adenocarcinomas. Widespread dissemination to many organs was common and death was usually due to carcinomatosis. AFP was detected in 53 of 72 (73.6%) liver-cell carcinomas of all types, but in none of the seven bile-duct carcinomas.

2156 AN EPIDEMIOLOGIC STUDY OF CARCINOMA *IN SITU* AND SQUAMOUS DYSPLASIA OF THE UTERINE CERVIX. *(E.)* Thomas, D. B. (Johns Hopkins U. Sch. Hyg. Public Hlth., Baltimore, Md.). *Am J Epidemiol* 98(1):10-28, 1973.

A questionnaire was administered to 324 women with conditions cytologically suggestive of cervical neoplasia and to 302 controls. The cases were 15- to 50-yr-old white residents of Washington County, Maryland, who first had an abnormal cytologic smear from 1965 through 1969. Subsequent biopsies revealed that 104 of the cases had carcinoma *in situ*, 105 had squamous dysplasia, and 25 had only chronic cervicitis; 90 cases were not biopsied. The controls were a probability sample of the population from which the cases were derived. Simultaneous adjustment of 14 variables using multiple linear regression revealed that women with carcinoma *in situ* differed from the controls by three factors: trichomoniasis, marital instability, and first child conceived before marriage. These results suggest that carcinoma *in situ* is epidemiologically similar to invasive carcinoma of the cervix as described by others, and that it may result from venereal transmission of one or more carcinogens. Only a portion of the women with dysplasia were epidemiologically similar to those with *in situ* carcinoma; they tended to have severe dysplasia. Adjustment of 14 variables showed the total group of women with dysplasia to differ from the controls only by the factors trichomoniasis and vaginal discharge. These findings suggest that much dysplasia is a nonspecific reaction of the cervical epithelium to inflammatory agents, and not caused by the same factors as carcinoma *in situ*.

2157 THE PROLIFERATION KINETICS OF L 1210 ASCITES TUMOUR. *(E.)* Dombernowsky, P. (Finsen Inst., Dept. Int. Med., Copenhagen, Denmark). *Acta Pathol Microbiol Scand* [*A*] 80(5):603-611, 1972.

2158 GROWTH PATTERN OF MENINGEOMAS PENETRATING THE SKULL BASE. *(E.)* Saksela, E. (Dept. Path., U. Helsinki, Finland), T. Hellström and B. Grahne. *Acta Otolaryngol (Stockh)* 74(5):363-370, 1972.

2159 ENHANCEMENT BY HUMAN PLACENTAL LACTOGEN OF MAMMARY HYPERPLASTIC NODULES IN OVARIECTOMIZED MICE.

(E.) Yanai, R. (Natl. Cancer Res. Inst., Tokyo, Japan) and H. Nagasawa. *Cancer Res* 33(7):1642-1644, 1973.

The effect of human placental lactogen (HPL) on the maintenance and growth of precancerous mammary hyperplastic nodules (HN) was studied in ovariectomized, multiparous C3H/He female mice. Mice which received twice daily s.c. injections of 0.5 mg HPL for 19 days after ovariectomy had a similar number of mammary HN to those in intact controls. The ovariectomized controls had significantly fewer HN than either of these groups. The average size of the HN was also significantly larger in the ovariectomized, HPL-treated mice and was smaller in the ovariectomized controls than in the intact controls. These results suggest that HPL can promote the maintenance and growth of HN in the absence of ovarian steroid hormones and imply that HPL is one of the major factors responsible for the earlier and higher incidence of spontaneous mammary tumors in multiparous mice compared to virgin mice.

2160 CHANGES IN MOUSE LIVER LIPID ANTIOXIDANT ACTIVITY DURING EXPERIMENTAL CARCINOGENESIS.

(Rus.) Burlakova, Ye. B. (Inst. Chem. Phys., Moscow, USSR) and Ye. M. Molochkina. *Biofizika* 18(2):293-298, 1973.

A study was made of the antioxidant activity of mouse liver lipids during carcinogenesis induced by o-aminoazotoluene, benzo[a]pyrene and γ-irradiation. Lymphatic leukemia was induced in C57 Bl mice by γ-irradiation (200 r x 4). Benzo[a]pyrene (3.2 mg/mouse) induced subcutaneous sarcomas in a group of hybrid mice. Hepatomas were induced in mice (breed not specified) by o-aminoazotoluene (dose not given). Changes in liver lipid antioxidant activity followed the same pattern in all these forms of carcinogenesis: a temporary increase in the antioxidant activity immediately after administration of the carcinogen, followed by a long decrease and then a sharp increase. This indicates that liver lipid antioxidant activity decreases by 40% in the first stage of carcinogenesis. This is followed by a sharp 2-3-fold increase about the time the tumor appears. Changes in liver lipid antioxidant activity were related to morphological stages of carcinogenesis (as shown in an earlier work). The effects of inhibitors of free radical processes [4-methyl-2,6-ditertbutyphenol (ionol) and 4-hydroxy-3,5-ditertbutyl-α-methylbenzylamine hydrochloride] on sarcomas induced by benzo[a]pyrene changed the course of the process at various stages of carcinogenesis. Ionol (20 mg/kg) administered to increase the liver lipid antioxidant activity somewhat accelerated sarcoma appearance. A large dose of ionol, i.e., 60 mg/kg, which decreased the liver lipid antioxidant activity, suppressed tumor development. 4-Hydroxy-3,5-ditertbutyl-α-methylbenzylamine (40 mg/kg x 4) increased the liver lipid antioxidant activity in benzo[a]pyrene-induced carcinogenesis, manifesting an inhibitory effect. These findings show that liver lipid antioxidant activity changes plays an important role in carcinogenesis.

2161 THE VALUE OF THE INTERNAL STRUCTURE OF THE NUCLEOLUS IN THE DIAGNOSIS OF MALIGNANCY.

(E.) Love, R. (Jefferson Med. Coll., Thomas Jefferson U., Philadelphia, Pa), M. Takeda, R. Z. Soriano and L. B. McCullough. *Acta Cytol (Baltimore)* 17(4):310-315, 1973.

Cells from 284 pleural and ascitic fluid preparations were studied to determine whether the internal structure of the nucleolus, as stained by a modification of the toluidine blue-molybdate method B of Love, could be of diagnostic value with respect to malignancy. Diagnoses based on the special technique were compared with those obtained by conventional techniques. Toluidine blue-molybdat staining demonstrated roughly spherical intranucleolar ribonucleoprotein structures (nucleolini) which were characteristically all the same size in nonneoplastic cells but which were strikingly dissimilar in size in neoplastic cells. A diagnosis of malignancy was made independently by nucleolar morphology and by Papinicolaou smear and cell block in 72 of 285 effusions. Malignancy was excluded by both methods in 199. Malignancy was diagnosed solely on the basis of nucleolar morphology in 11 cases (4%). Of these, eight were known to have carcinoma, five with metastatic disease. Thus internal nucleolar structure appears to be a valuabl adjunct to conventional techniques in the diagnosis of malignancy.

2162 MEMBRANE ASPECTS OF NEOPLASIA.

(E.) Wallach, D. F. H. (Tufts-New England Med. Ctr., Boston, Mass.), H. Knufermann and F. Wunderlich. *FEBS Letters* 33(3):275-280, 1973.

Several aspects of membrane involvement in neoplasia were explored at an informal workshop held in March 1973 in Germany and attended by close to 40 participants. The speakers concentrated on the plasma membrane. The possibility that information transfer between cells might be involved in malignant tissue disorganization received considerable attention. Th role of cell surface antigens as an intrinsic defens against malignant cells in a tumor-bearing host was addressed by three participants. A number of speake discussed the diverse strategems being used in attem to define the membrane anomalies of malignant cells in biochemical and/or biophysical terms. The discussion also extended to the possible application of novel methods of basic membrane research to the tumor problem. In general, the meeting emphasized the increasing need for multidisciplinary cooperatio in cancer research.

2163 A RAT MAMMARY CARCINOMA *IN VIVO* AND *IN VITRO:* ESTABLISHMENT OF CLONAL LINES OF THE TUMOR.

(E.) Kano-Sueoka, T. (Dept. Biochem., Princeton, N.J.) and P. Hsieh. *Proc Natl Acad Sci USA* 70(7):1922-1926, 1973.

The isolation of cell lines that possess a specific growth requirement *in vitro* from a rat mammary carcinoma that requires pituitary hormone for growth *in vivo* is described as well as some properties of

hese cell lines. The growth of these cell lines
n *vitro* is markedly stimulated in the presence of
rolactin. The cells grow well in a medium supple-
ented with fetal-calf serum (generation time: 9-16
r), but do not grow with calf serum. Fetal-calf serum
upports little growth when the serum is subjected to
nti-prolactin-affinity chromatography. Gel filtration
f fetal-calf serum indicates that the growth stimula-
ory activity is in a large molecular wt material
close to 100,000), suggesting that prolactin is as-
ociated with another serum component. The present
orking hypothesis is that: 1) prolactin (molecular
t 22,000-24,000) is the major hormone required by
e cells and calf serum does not contain it in suf-
icient amounts; and 2) prolactin is bound to some
arge plasma protein and this complex is biologi-
ally active or the activity of prolactin is pro-
ected by binding to the protein. A variant of the
riginal tumor, which does not require hormone for
rowth, was obtained, and clonal cell lines of this
mor were also established. These cells, in con-
rast to the hormone-dependent ones, grow well with
ither fetal-calf serum (with or without anti-prolac-
in treatment) or calf serum.

64 PARTIAL PURIFICATION AND CHARACTERIZATION
OF RNA POLYMERASE FROM NUCLEI OF EHRLICH
CITES TUMOR CELLS. *(E.)* Blair, D. G. R. (Dept.
ncer Res., U. Saskatchewan, Saskatoon, Canada) and
K. Mukherjee. *Can J Biochem* 51(5):586-596, 1973.

A-dependent RNA polymerase (nucleosidetriphos-
ate:RNA nucleotidyltransferase) was partially pur-
ied from nuclei of Ehrlich ascites tumor cells by
e techniques of sonic disruption in the presence
0.3 M ammonium sulfate, ultracentrifugation, salt
actionation, and dialysis. The purified enzyme
d an absolute requirement for DNA and was inhibited
actinomycin D and other known inhibitors of DNA-
pendent RNA polymerase, but not by rifamycin or
fampicin. The four ribonucleoside triphosphates
d Mn^{2+} ions were also required for optimal activity.
timal concentrations for Mn^{2+} and Mg^{2+} ions and
$H_4)_2SO_4$ were 2.4 mM, 6.0 mM, and 0.05 M, respec-
vely. All the native and heat-denatured DNA's
at were tested served as templates, but the rela-
ve activities with the two forms of DNA depended
on the composition of the reaction medium. Of
e natural RNA's and 11 synthetic homo- and hetero-
lyribonucleotides that were tested, the RNA's
ly(A), poly(A,U), and poly(A,C) were slightly
tive, but less than 5% as efficient as calf thymus
tive DNA, with ^3H-UTP as the labeled substrate.

65 DIFFERENCE IN THE MOBILITY OF LECTIN SITES
ON THE SURFACE MEMBRANE OF NORMAL LYMPH-
YTES AND MALIGNANT LYMPHOMA CELLS. *(E.)* Inbar, M.
ept. Genetics, Weizmann Inst. Sci., Rehovot,
rael), H. Ben-Bassat and L. Sachs. *Int J Cancer*
(1):93-99, 1973.

ssible differences in the mobility of the sites
r Concanavalin A (Con A) and the lectin from wheat
rm (WGA) and whether there is a difference in the
bility of these sites in normal lymphocytes and
lignant lymphoma cells were investigated. Normal lymphocytes were obtained from adult rat lymph nodes or mouse spleen and both sources gave the same results. Lymphoma cells were obtained from an ascites form of a Moloney virus-induced lymphoma grown in A-strain mice. It was found that binding sites for Con A can move on the surface membrane. This induction of movement resulted in the formation of caps of Con A membrane-site complexes in normal lymph-node or spleen lymphocytes, but not in the malignant lymphoma cells, indicating a difference in the mobility of Con A sites on the surface membrane of normal lymphocytes and lymphoma cells. Binding sites for WGA did not form caps either on normal lymphocytes or on lymphoma cells. Normal lymphocytes with caps induced by Con A still showed binding of WGA to the rest of the membrane. The mobility of Con A sites was, therefore, independent of WGA sites. The results suggest that binding sites for Con A are floating in a fluid membrane in a random distribution. This distribution can be changed by the interaction with Con A, so that the membrane site-Con A complexes on the surface membrane form a new distribution. Depending on the membrane fluidity, this complex will form clusters of sites on the membrane in lymphoma cells and a concentration of most of the sites on one pole of the membrane in normal lymphocytes. It is further suggested that the mobility of Con A binding sites can be used as a probe for the fluid state of this specific carbohydrate structure on the surface membrane and that malignant transformation is associated with a decrease in fluidity.

2166 ESTRADIOL RECEPTORS IN BENIGN AND MALIGNANT DISEASE OF THE BREAST. *(E.)* Leung, B. S. (U. Oregon Med. Sch., Portland), L. C. Manaugh and D. C. Wood. *Clin Chim Acta* 46(1):69-76, 1973.

Estrogen receptor in human breast carcinoma and other tissues was studied in the presence or absence of estrogen competitor by sucrose gradient centrifugation and a dextran-charcoal technique. Cytoplasmic estrogen receptor in tumor was demonstrated to be localized at approximately the 8S region of a 10-30% sucrose gradient. In the dextran-charcoal method, by employing a binding index (BI), which compared the ratio of specific estradiol receptor binding (ER) to total binding (both specific and nonspecific), tumors from 53 patients were classified into three groups: those with high BI, low BI and negative ER. The BI corresponded well with the cytoplasmic ER content in these groups and provided an excellent measurement of the relative binding capacity. Approximately two-thirds of the malignant tumors had ER. Only three of the 17 benign tumors had any measurable ER. Normal tissues from breast, ovary, adrenal, and skin did not contain receptor. There was no relationship between tumor histopathology and ER content in the tumors. The value of this simple dextran-charcoal technique, BI values and ER content for routine clinical purposes in predicting patient response to endocrine ablation is very briefly discussed. Although the dextran-charcoal method is simple, it is not without drawbacks. For example, it is not possible to be absolutely confident that a low BI does not include any negative receptor group. Such difficulty could be overcome by increasing sample size and by correlation with the clinical response of such patients to endocrine ablation. The grouping of patients by BI values in this paper is based on preliminary results. When more data is accumulated and the patients have been followed for longer intervals, it may be possible to determine precisely the exact division for positive and negative groups as related to the clinical response to endocrine ablation.

2167 THE EFFECT OF 5-FLUORODEOXYURIDINE ON THE SYNTHESIS OF DEOXYRIBONUCLEIC ACID PYRIMIDINES IN PRECANCEROUS RAT LIVER. *(E.)* Goodman, J. I. (McArdle Lab. Cancer Res., U. Wisconsin, Madison) and V. R. Potter. *Mol Pathol* 9(3): 297-303, 1973.

The *de novo* and salvage pathways for the synthesis of DNA precursors were studied in precancerous livers of male Sprague-Dawley rats fed a diet containing the hepatocarcinogen 3'-methyl-4-dimethylaminoazobenzene for three wk. Rats received repeated injections of ^{3}H-thymidine and ^{14}C-orotic acid. Ninety min after the last injection the livers were removed, homogenized, the nuclei were separated into class N-1 (hepatocyte) and class N-2 (non-hepatocyte, "stromal") and the radioactivity in pyrimidines of their DNA hydrolysates was determined following chromatographic separation. Markedly different patterns of ^{3}H and ^{14}C incorporation into DNA pyrimidines indicated substantial differences in the relative activities of the *de novo* and salvage pathways between the two cell classes. Liver cells from animals which had also received a single i.p. injection of 5-fluorodeoxyuridine (5-FUdR) showed a marked decrease in ^{14}C-orotic acid, but not ^{3}H-thymidine, incorporation into DNA pyrimidines. The difference in sensitivity of the two pathways to 5-FUdR emphasizes the advisability of monitoring both simultaneously when studying the effects of substances on nucleic acid synthesis. The activities of thymidylate synthetase, thymidine kinase and dCMP deaminase differed between hepatocyte and whole liver homogenate preparations.

2168 THE ACTIVITY OF GLUTATHIONE REDUCTASE IN DIFFERENT HUMAN AND ANIMAL TUMORS. *(Rus.)* Misheneva, V. S. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR) and T. A. Goryukhina. *Vopr Onkol* 19(3):58-63, 1973.

The activity of glutathione reductase in tumor extracts was determined spectrophotometrically by the decrease of NADP-H_2 due to oxidation. The activity of this enzyme was higher in malignant tumors from humans (uterine and mammary cancer) and in highly differentiated blastomas from animals (rhabdomyoblastomas from rats and hepatoma 22A from mice) than in normal tissues (human uterus, rabbit liver). Its activity was highest in rat rhabdomyoblastomas, being 3-4 times higher than the normal tissue. Benign human fibromyomas and fibroadenomas, rabbit Brown-Pearce tumors and mouse hepatoma 22A had slightly increased glutathione reductase activity. Only in
malignant breast tumors from women was there a dire
relation between the high level of reduced glutathi
and glutathione reductase activity. The constant
for the glutathione reductase reaction rate and the
affinity of the enzyme for the substrate were high
in all malignant tumors of humans and animals
(uterine cancer, rhabdomyoblastomas, mouse hepatoma

2169 SELECTION OF SUCCESSIVE TUMOUR LINES FOR METASTASIS. *(E.)* Fidler, I. J. (Sch. Dental Med., U. Pennsylvania, Philadelphia). *Nature* [*New Biol*] 242(118):148-149, 1973.

A system of animal transplantation combined with tissue culture has been devised to select for B_{16}
melanoma tumor lines with enhanced metastatic poten
tial. By successively injecting i.v. B_{16} cells into C57 mice, waiting 2 wk, removing pulmonary
metastatic nodules, culturing the cells as monolaye
and reinjecting the cells into new mice, a highly significant ($p < 0.001$) and stable three- to six-fold increase in metastatic potential could be attained. These differences in metastatic potential were attributed to properties intrinsic in the tumor cells.

2170 THE MINERAL CONTENT AND WATER COMPARTMENT OF LIVER AND OF MORRIS HEPATOMATA 5123tc AND 3924A AND THE CHANGES OF COMPOSITION OCCURRING DURING NECROSIS IN HEPATOMA 3924A. *(E.)* Rossum, G. D. V. van (Temple U. Sch. Med., Philadelphia, Pa.), T. Galeotti and H. P. Morris. *Cancer Res* 33(5):1078-1085, 1973.

The water, sodium, potassium, magnesium, calcium, c
ride, and inorganic phosphates contents of normal r
liver were compared with those of the viable tissue
of two Morris hepatomata of different histological
appearance. The tumors were the hepatocellular car
noma 5123tc, which is histologically intermediate b
tween well and poorly differentiated and grows with
time of 1.3 months/generation, and the poorly diffe
entiated 3924A, which grows with a time of about 1
month/generation. The water content of rat liver a
of hepatoma tissue that was free of macroscopically
visible necrosis increased in the order, liver <
5123tc < 3924A. Sodium and chloride followed the
same order. Determination of the volume of tissue
water compartments, based on the assumed extracellu
lar position of chloride, showed that the increasin
water content was shared by both intracellular and
extracellular compartments, although the latter und
went the greater change. The potassium and magnesium contents/unit dry weight increased in the order
liver < 5123tc < 3924A, but the apparent concentrations in the intracellular water varied little between the tissues. The calcium content followed th
same order but showed quantitatively greater change
than the other ions, the relative content ratio for
liver:5123tc:3924A being 1:3:7. Histological estimates of the microareas of necrosis in the apparent
ly viable regions of hepatoma 3924A suggested that much of the calcium of this tumor was probably present in necrotic cells. Comparing the apparently viable peripheral areas of hepatoma 3924A with the grossly necrotic areas in the centers of the tumors

ndicated a 25% exchange of potassium for sodium, a 5% loss of magnesium, a 50% gain of phosphate, and 500% increase of calcium during necrosis. The rossly necrotic areas contained cells that were till capable of respiring, glycolyzing and active- y transporting sodium and potassium, although the ctivities were less than in the areas free of acroscopically visible necrosis. However, the abil- ty actively to extrude calcium was apparently com- letely lost. Despite the parallel increases of alcium and phosphate in the order, liver < "viable" 924A < necrotic 3924A, the relative ratios of the wo ions suggest that the deposition of calcium hosphate alone was not the only process involved.

171 FOETAL LIVER tRNA^{phe} IN RAT HEPATOMA.

(E.) Gonano, F. (Inst. Gen. Path., U. odena, Italy), G. Pirro and S. Silvetti. *Nature lew Biol*] 242(121):236-237, 1973.

ransfer RNA (tRNA) species of adult and fetal rat iver and of Morris hepatoma 5123 C were compared reverse phase chromatography. Studies in which rified hepatoma tRNA charged with ^{14}C-phenylala- ine was co-chromatographed with either adult or etal tRNA charged with ^{3}H-phenylalanine showed at, in addition to the normal adult tRNA, hepato- cells contained fetal type tRNA. Fingerprinting partial hydrolysates of the two hepatoma tRNAs owed differing nucleotide sequences which, how- er, did not involved the anticodon region. Al- ough the presence of embryonic tRNAs in the patoma may be an epiphenomenon of the transforma- on process, it might also serve a critical role maintaining the cell in its dedifferentiated ate.

172 CYTOSOL-MODULATED TRANSPORT OF MESSENGER RNA FROM ISOLATED NUCLEI.

(E.) Schumm, E. (Ohio State U. Coll. Med., Columbus), H. P. rris and T. E. Webb. *Cancer Res* 33(8):1821-1828, 73.

e influence of cytoplasmic factors on the energy- pendent release from nuclei of messenger-like RNA ribonucleoprotein particles (*i.e.*, informosomes) s been investigated in a cell-free system that ecludes detectable nuclear lysis. Maximum release rapidly labeled RNA from the prelabeled nuclei normal, regenerating, or neoplastic rat liver s observed only when the latter were incubated in tologous cytosol. Comparative studies involving A-DNA hybridization and density gradient centrifu- tion of the presumptive messenger RNA released in tologous and heterologous systems indicated that e RNA transported is both qualitatively and quan- tatively dependent not only upon the source of the clei but also upon the source of the dialyzed cyto- l. The results suggest that the cytosol contains ndialyzable components that act posttranscription- ly to regulate the nuclear processing and/or trans- rt of messenger RNA and further that there are fferences in these factors in normal, regenerating, d neoplastic liver. Posttranscriptional controls erating within the nucleus may have particular levance to the cancer problem in view of evidence that indicates that RNA retained within the nucleus of normal cells appears in the cytoplasm of cancer cells and complementary evidence that indicates that nuclei from cancer cells behave normally when transplanted into enucleated cells containing normal cytoplasm.

2173 EVIDENCE FOR THE PRESENCE OF NONHISTONE CHROMOSOMAL PROTEINS IN THE NUCLEOPLASM OF HeLa S_3 CELLS.

(E.) Stein, G. S. (Dept. Biochem., U. Florida, Gainesville) and C. L. Thrall. *FEBS Letters* 32(1):41-45, 1973.

Nucleoplasmic and chromosomal protein fractions were purified from L-^{14}C-leucine-labeled exponentially growing HeLa S_3 cells. SDS polyacrylamide gel electrophoresis revealed nine labeled peaks present in both the nucleoplasmic and chromosomal fractions and migrating in the same region as nonhistone chromosomal proteins. Three additional nonhistone peaks were found only in the nucleoplasmic fraction, and one additional peak was present only in the chromosomal protein fraction. The finding of nonhistone proteins in both chromatin and nuclear sap is consistent with the concept of nonhistone chromosomal proteins as regulators of gene expression.

2174 NUCLEAR DOUBLE-STRANDED RIBONUCLEIC ACID OF MAMMALIAN CELLS. CHARACTERISTICS AND BIOSYNTHESIS.

(E.) Patnaik, R. (Dept. Microbiol., Indiana U., Bloomington) and M. W. Taylor. *Biochemistry* 12(10):1990-1995, 1973.

Ribonuclease-resistant RNA synthesis was examined in Sarcoma-180 cells, Ehrlich ascites tumor cells, and L cells in the presence of high or low concentrations of actinomycin D. The RNase-resistant species were characterized by sucrose gradient analysis, Sepharose 2B column chromatography, and thermal denaturation studies. These species are predominantly of nuclear origin and their synthesis is sensitive to 5 µg/ml of actinomycin D. In all the cell lines, this RNA species is polydisperse on sucrose gradient and of low molecular weight. By thermal denaturation profile, it appears to be heterogeneous and not completely base paired. Sarcoma-180 cells not only contain the low molecular weight species, but also a high molecular weight double-stranded RNA that has characteristics of a completely base-paired molecule. These observations indicate that this material is probably of viral origin. It is possible that this RNA reflects the replicative form of an unknown class of DNA virus.

2175 ABNORMAL KARYOTYPIC CLONES IN HUMAN ACUTE LEUKEMIA: THEIR NATURE AND CLINICAL SIGNIFICANCE.

(E.) Fitzgerald, P. H. (Christchurch Hosp., New Zealand), P. E. Crossen and J. W. Hamer. *Cancer* 31(5):1069-1077, 1973.

Forty-four percent of 110 unselected patients presenting with acute myeloid, lymphatic, or stem cell leukemia had a karyotypically abnormal, clonal cell line in the bone marrow, and about 60% of these patients also had apparently normal, diploid cells coexisting with the abnormal cells. The karyotypic changes were diverse and distinctive for each patient, but acute lymphatic leukemia tended to hyperdiploidy. The karyotypic changes showed low involvement of F chromosomes, and some excess involvement of C and G chromosomes. There was little evidence of karyotypic evolution. Where therapy induced full remission, abnormal cell lines tended to be replaced by diploid cells. The incidence of karyotypic abnormality at diagnosis was not related to patient age, sex, or type of leukemia. Significantly, the presence of karyotypic abnormality did not influence the chance of leukemic remission or patient survival, except that male patients with C chromosome deficiency survived on average three times longer than those with additional C chromosomes. This study and a review of five other major studies led to the conclusion that karyotypic abnormalities of acute leukemia have little or no etiological, clinical or hematological significance, and do not influence patient survival. It would appear that cell lines characterized by karyotypic abnormality rarely determine leukemic progression, but develop within limits set by a prescribed and individual cell environment.

2176 PLEIOTYPIC CONTROL BY ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE: A MODEL FOR GROWTH CONTROL IN ANIMAL CELLS. *(E.)* Kram, R. (Dept. Biochem., U. California, San Francisco), P. Mamont and G. M. Tomkins. *Proc Natl Acad Sci USA* 70(5):1432-1436, 1973.

The effects of serum deprivation on several general cellular biochemical processes ("pleiotypic response") related to the growth of normal fibroblasts can be mimicked by treatment of these cells with prostaglandin E_1 in the presence of serum. $N^6,O^{2'}$-Dibutyryl adenosine 3':5'-cyclic monophosphate and theophylline inhibit the membrane transport processes without much effect on other pleiotypic reactions such as overall protein and RNA synthesis and protein degradation. The amount of intracellular cyclic AMP increases during serum starvation and returns to the initial concentration in unstarved cells when growth is initiated again upon addition of serum. Fibroblasts transformed by simian virus 40 have a lower cyclic AMP content than their untransformed parents. Serum deprivation neither increases cyclic AMP content nor significantly affects the pleiotypic reactions in transformed cells. Cycloheximide causes a decrease in cyclic AMP content of normal fibroblasts coincidentally with the ability of this inhibitor to stimulate uridine transport and slow protein degradation in cells deprived of serum.

2177 MIXED CELL CULTURES OF NORMAL AND LEUKAEMIC CELLS. *(E.)* Pegrum, G. D. (Charing Cross Hosp. Med. Sch., London, England), I. C. Balfour, C. A. Evans and V. L. Middleton. *J Pathol* 110(1):59-66, 1973.

Peripheral blood leukocytes from 36 patients with acute or chronic (lymphatic or myeloid) leukemia were incubated in mixed culture with lymphocytes from healthy volunteers. Both "two-way" and "one-way" reactions were studied. The cells were HL-A typed, where possible, by a microcytotoxicity technique. Increased uptake of ^{3}H-thymidine was found in more than half the cultures of acute leukemic and chronic lymphatic leukemic cells, but this was not found wit chronic myeloid leukemic cells. "One-way" cultures revealed that, with two exceptions, the responding cells were the healthy lymphocytes. There was a correlation between antigenic differences detected serologically and the intensity of ^{3}H-thymidine uptake in the cultures with chronic lymphatic leukemic cells, but not with acute leukemia cells. It is suggested that antigens of the HL-A system may be implicated in this stimulation seen with chronic lymphatic leukemic cells, but not with acute leukemic cells. It is possible that in the latter case "leukemia-associated antigen" may play a significant role.

2178 COMPARATIVE STUDY OF TRANSFER RIBONUCLEIC ACIDS EXTRACTED FROM RAT LIVER AND ZAJDELA HEPATOMA. I. ACCEPTOR CAPACITY OF THE tRNAs. *(Fr.* Mercier, J. (Pasteur U., Strasbourg, France), N. Befort, J. P. Beck and J. P. Ebel. *Biochimie* 54(10):1319-1326, 1972.

tRNAs, extracted from normal rat liver or Zajdela ascites hepatoma, were reacted with 18 different amino acids in the presence of protein extracts containing homologous or heterologous aminoacyl-tRNA synthetases, Mg^{++} and ATP. Optimal quantities of enzyme extract and optimal incubation times were established separately for each amino acid. With alanine, aspartic acid, histidine and lysine, the extent of tRNA aminoacylation was about the same in homologous and heterologous systems. Regardless of the origin of the enzyme, with arginine, tryptophan and valine the extent of aminoacylation was significantly greater with tRNA from normal rat liver than with that from ascites hepatoma; the reverse was true of proline. Significant difference were also observed when the same tRNA was incubated with enzymes from normal liver or hepatoma; glycine was more reactive in the presence of hepatoma enzyme, while tyrosine was more reactive with enzyme from normal liver. Crude protein extracts contained enzymes capable of converting aspartic and glutamic acids into their respective amides and enzymes which liberated ammonia from these amides. These enzymes interfere with interpretation of the results.

2179 CELL DIVISION OF *ESCHERICHIA COLI:* CONTRO BY MEMBRANE ORGANIZATION. *(E.)* Wu, P. C. (Dept. Biochem. Sci., Princeton U., N.J.) and A. B. Pardee. *J Bacteriol* 114(2):603-611, 1973.

Cells of certain strains of *Escherichia coli*, after transfer from 37 to 45 C and incubation for 16 min, were observed to swell and subsequently divide synchronously. This swelling and the resulting stretching of the membrane are proposed to be the basis for the synchronous division. Four lines of evidence support this hypothesis. First, osmotic protection by the addition of either sodium chloride or sucrose at the time of heat shock prents both swelling and synchrony. Second, a tant neither swelled nor divided synchronously ter heat shock. Third, cells grown for several nerations with 10% sucrose in the medium swelled d divided synchronously upon transfer to medium thout sucrose. Fourth, the mutant not synronized by heat shock also swelled and underwent nchronous division after the osmotic shift. e results suggest that a special membrane conguration, which could be altered by swelling of e cell, might be critical for cell division. model of the membrane septation site is proposed suggest a mechanism for the swelling effect of ll division. The model assumes a local "ordered" ructure, called a memorcon: membrane-morphologyntrol site, at which the proteins and enzymes volved in septum formation are postulated to ve a relatively fixed orientation. Heat shock d swelling cause a shift in this orientation ading to synchronous cell division.

80 ALTERED POPULATIONS OF ACIDIC CHROMATIN PROTEINS IN BREAST CANCER CELLS. *(E.)* adohama, N. (Dept. Med., U. Wisconsin, Madison) d R. W. Turkington. *Cancer Res* 33(6):1194-1201, 73.

e acidic chromatin proteins extracted from normal d neoplastic mammary cells of C3H mice and Fischer ts have been compared by polyacrylamide gel elecophoresis, amino acid analysis, and radioactivity beling patterns of synthesis *in vitro*. Following rification of chromatin from purified nuclei and traction of histones in 2 M sodium chloride, the idic proteins associated with DNA were subjected electrophoresis on polyacrylamide gels containg sodium dodecyl sulfate, and the stained gels re compared by absorbance scanning. Striking fferences were observed in the banding patterns of oteins derived from the normal or neoplastic lls. Among a group of high-molecular-weight mponents characteristic of the lactational mamry gland, several were preserved while others re deficient or undetectable in the neoplastic lls. In addition, several components of interdiate molecular weight that were present in the oplastic cells were undetectable in the normal ttern. Differences between the protein populaons derived from normal and neoplastic cells re also demonstrated by amino acid analysis. gan cultures of carcinoma and gland explants monstrated that the normal mammary gland synthezed lower-molecular-weight species at a higher t rate, whereas the carcinomas exhibited a latively higher net rate of synthesis of highlecular-weight components. ^{32}P was incorporated as osphoserine and phosphothreonine residues in the jor portion of the proteins of all preparations, though the carcinomas exhibited a relatively wer net rate of phosphorylation of the highlecular-weight proteins. The differences been preparations derived from neoplastic and rmal tissues could not be attributed merely to e physiological state of the gland, since the tterns of the proteins from the carcinomas were so significantly different from previous patterns rived from the mammary glands of virginal or pregnant animals. The results demonstrate that the neoplastic characteristics of mammary carcinomas are associated with an altered content and pattern of synthesis of acidic chromatin proteins.

2181 STUDIES ON A CALF THYMUS RIBONUCLEASE SPECIFIC FOR RIBONUCLEIC ACID-DEOXYRIBONUCLEIC ACID HYBRIDS. *(E.)* Haberkern, R. C. (Childrens' Hosp. Med. Ctr., Boston, Mass.) and G. L. Cantoni. *Biochemistry* 12(13):2389-2395, 1973.

Ribonuclease H, an enzyme previously shown to hydrolyze specifically the RNA moiety of RNA-DNA hybrids, was purified 200-fold from calf thymus homogenates using high-speed centrifugation and ammonium and protamine sulfate precipitation followed by DEAE, hydroxylapatite and Sephadex G-200 column chromatography. The molecular wt of the enzyme, as determined by both SDS polyacrylamide gel electrophoresis and equilibrium density centrifugation, was 74,000-76,000, indicating that it is composed of a single subunit. RNase H required the presence of a divalent cation, Mn being more effective than Mg. Although poly(rAdT) was the only active substrate, poly(rA), poly(rArU), double-stranded reovirus RNA, poly(rIdC) and poly(rCdG) were effective inhibitors of RNase H activity. RNase H showed different degrees of sensitivity to four rifampicin derivatives, none of which correlated with the ability of the particular derivative to inhibit reverse transcriptase or mammalian DNA polymerase. Thin-layer electrophoretic analysis of the reaction products showed that RNase H is an endonuclease which produces mono- and oligoribonucleotides terminated by a 5'-phosphate.

2182 AMPLIFICATION AND EVOLUTION OF DEOXYRIBONUCLEIC ACID SEQUENCES EXPRESSED AS RIBONUCLEIC ACID. *(E.)* Kohne, D. E. (Dept. Terrestrial Magnetism, Carnegie Inst., Washington, D.C.) and M. J. Byers. *Biochemistry* 12(13):2373-2378, 1973.

Nonrepeated DNA sequences which are expressed by being transcribed into RNA (E-DNA) were isolated by utilizing nucleic acid hybridization techniques and RNA from various tissues of the cow. To determine the extent of gene amplification, comparisons were made of reassociation kinetics obtained when ^{3}H-prelabelled E-DNA was allowed to react with either: a) DNA from the same tissue used to obtain the RNA utilized originally to isolate the E-DNA or b) total cell nonrepeated DNA from a different tissue. The results showed no differences in the rates of reassociation of E-DNA with DNA from adult or fetal liver or adult brain, thus indicating that the majority of the E-DNA sequences are not amplified. To determine the degree of evolution of these E-DNA sequences, the thermal stabilities of homologous E-DNA total cell nonrepeated DNA duplexes and heterologous duplexes (i.e., cow E-DNA with sheep or pig total cell nonrepeated DNA) were compared. Since the thermal stability of the interspecific duplexes was much lower than that of the homologous DNA-DNA duplexes, it was concluded that the E-DNA sequences of the cow have diverged to essentially the same extent as the bulk of nonrepeated DNA since the divergence 25 million yr ago of the cow from the sheep and 55 million yr ago from the pig.

2183 HYBRIDIZATION STUDIES WITH NUCLEIC ACIDS FROM MURINE MYELOMA. COMPARISON OF PULSE-LABELED RIBONUCLEIC ACID PREPARED FROM FOUR DIFFERENT TUMORS. *(E.)* Kosky, K. (U. Washington Sch. Med., Seattle) and R. G. Krueger. *Biochemistry* 12(8):1543-1547, 1973.

Pulse-labeled RNAs were prepared from four different murine myelomas and normal mouse and spleen tissue. Quantitative and qualitative differences in the RNA populations were compared by competitive hybridization of various RNA preparations with tumor and liver DNA. Tumor cells contained RNA populations not present in normal mouse liver or spleen. Differences in the RNA populations in the different tumors were maximized if the reaction of the labeled RNA and DNA was carried out at low RNA/DNA ratios under moderately stringent reaction conditions. In the competition experiments, the effectiveness of each unlabeled RNA preparation as competitor appeared to be related to the type of immunoglobulin produced by each tumor. A comparison of nuclear and cytoplasmic tumor RNAs suggests that the major portions of the RNA populations studied were mRNA species present in both the nucleus and cytoplasm of tumor cells. These data thus indicate that the RNA transcripts coded for by a given myeloma DNA are distinguishably different from RNA transcripts from other myelomas.

2184 CERVICAL CARCINOMA IN WOMEN UNDER TWENTY-ONE YEARS OF AGE. *(E.)* Kling, T. G. (U. Iowa Hosp., Iowa City) and H. J. Buchsbaum. *Obstet Gynecol* 42(2):205-207, 1973.

Eight patients under 21 years of age (18 to 20) are reported who had invasive cervical carcinoma. Only two patients in the group were nulliparous; one was pregnant for the 4th time at age 20. Seven patients had epidermoid carcinoma, while in one, the histological pattern was that of an adenocarcinoma. The histology and clinical course parallels that of the adult type of cervical carcinoma rather than that of infancy or childhood. The rarity of invasive carcinoma in minors is supported by a review of the literature. The incidence of *in situ* carcinoma is proportionally higher in women under 21 than is invasive carcinoma.

2185 MECHANISM INVOLVED IN THE EFFECT OF SIMPLE PHENOLS ON TRANSCRIPTION. *(Rus.)* Dzhokhadze, D. I. (Inst. Plant Biochem., Acad. Sci. Georgian SSR, USSR) and R. K. Papelishvili. *Soobshcheniia Akad Nauk Gruzinsk SSR* 70(3):717-720, 1973.

To study the effect of phenols on endogenous RNA polymerase activity, highly purified nuclei isolated from rat liver were incubated with ^{14}C-labelled ATP, guanosine triphosphate, cytidine triphosphate, and uridine triphosphate in the presence of freshly sublimed pyrogallol, phloroglucinol, and hydroquinol. When nuclei containing 56 μg of DNA were incubated with 10-20 g of phloroglucinol, RNA synthesis was stimulated, but the rate of RNA synthesis began to decrease with increasing phloroglucinol concentrations. Under similar conditions pyrogallol and, to a greater extent, hydroquinol inhibited RNA synthes These findings suggest that the effect of trihydrox phenols on RNA polymerase activity depends upon the location of hydroxyl groups on the ring. UV absorption spectra of these three phenols, DNA from calf thymus or *Escherichia coli* and mixtures of phenols and DNA showed that the phenols form a complex with RNA polymerase but not with the DNA matrix. The fo mation of these complexes causes a change in RNA polymerase activity which is manifested as a change in the transcription activity of the DNA.

2186 CONTENT OF ESTROGEN RECEPTORS IN HUMAN BREAST CANCERS. *(E.)* Spaeren, U., (The Norwegian Radium Hosp., Montebello, Oslo), S. Olsne I. Brennhovd, J. Efskind and A. Pihl. *Eur J Cancer* 9(5):353-357, 1973.

The estrogen binding capacity of 251 human breast cancers has been determined. The cytosol of the tumors was incubated with labelled estradiol-17-β and subjected to sucrose-gradient-density centrifugation. The difference in the radioactivity in the 8S area between samples incubated in the absenc of an estrogen antagonist was taken as a measure of the estrogen receptor content. The receptor content of the tumors varied over a wide range. Measu able amounts were present in 184 cases (73.1%). Th content was 2-3 times higher in tumors from patient above 50 yr than in those below this age. No relationship was found between the receptor content and the histological type of the tumors with the exception that tumors of the scirrhus type containe less receptors that other types of tumor. The recep concentration was the same in different clinical st of the disease suggesting that the receptor content of human breast cancers does not tend to decline wi progression of the tumors.

2187 TUMORS OF THE MANDIBLE AND MAXILLA IN CHILDREN. II. A STUDY OF 14 PRIMARY AND SECONDARY MALIGNANT TUMORS. *(E.)* Dehner, L. P. (Barnes Hosp., St. Louis, Mo.). *Cancer* 32(1):112-120, 1973.

2188 STUDIES CONCERNING THE REGIONAL LYMPH NODE IN CANCER. VI. CORRELATION OF LYMPHOCYTE TRANSFORMATION OF REGIONAL NODE CELLS AND SOME HISTOPATHOLOGIC DISCRIMINANTS. *(E.)* Fisher, E. R. (U. Pittsburgh Sch. Med., Pa.), E. Saffer and B. Fisher. *Cancer* 32(1):104-111, 1973.

2189 DNA VALUES IN MYELOMA. *(E.)* Mundy, G. R (Dept. Med., U. Tasmania, Australia). *Cancer* 32(1):61-68, 1973.

2190 ADENYLATE CYCLASE ACTIVITY IN NORMAL AND LEUKEMIC HUMAN LEUKOCYTES AS DETERMINED BY A RADIOIMMUNOASSAY FOR CYCLIC AMP. *(E.)* Polgar, P. (Boston U. Sch. Med., Mass.), J. C. Vera, P. R. Kelley and A. M. Rutenburg. *Biochim Biophys Acta* 297(2):378-383, 1973.

2191 ADENOSINE 3',5'-MONOPHOSPHATE AND GUANOSINE 3',5'-MONOPHOSPHATE: CONCENTRATIONS IN MORRIS HEPATOMAS OF DIFFERENT GROWTH RATES. *(E.)* Thomas, E. W. (U. Virginia Sch. Med., Charlottesville), F. Murad, W. B. Looney and H. P. Morris. *Biochim Biophys Acta* 297(2):564-567, 1973.

2192 DNA POLYMERASE AND TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE ACTIVITY OF NORMAL AND BACTERIA-FREE CROWN GALL TUMOR TISSUE CULTURES OF TOBACCO. *(E.)* Sahai Srivastava, B. J. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Biochim Biophys Acta* 299(1):17-23, 1973.

2193 DIFFERENCES IN NUCLEOTIDE SEQUENCES OF RIBOSOMAL RNA BETWEEN THE LIVER AND A HEPATOMA OF C3H/HE MICE. *(E.)* Hashimoto, S. (Tokushima U. Sch. Med., Japan) and M. Muramatsu. *Eur J Biochem* 33(3):446-458, 1973.

2194 TUMOR LIPIDS: COMPARISON OF GLYCERYL ETHERS OF MOUSE AND INSECT TUMORS. *(E.)* Lambremont, E. N. (Nuclear Sci. Ctr., Louisiana St. U., Baton Rouge) and M. F. Welch. *Lipids* 8(3):160-162, 1973.

2195 CANCER DETECTED IN LIVING ANIMALS USING NUCLEAR MAGNETIC RESONANCE. *(E.)* Anonymous. *Nat Bur Stand (U.S.) Tech News Bull* 57 (2):27, 1973.

2196 REVERSIBLE AND IRREVERSIBLE EFFECTS OF TISSUE CULTURE ON ENZYME PATTERNS OF SPONTANEOUS MOUSE TUMORS AND MOUSE AND HUMAN EMBRYO TISSUES. *(E.)* Wilson, P. D. (Imperial Cancer Res. Fund, London, England). *Cancer Res* 33(2):375-382, 1973.

2197 A CONTRIBUTION TO THE CLINICAL PICTURE AND BIOCHEMISTRY OF HORMONE-PRODUCING ADRENAL CARCINOMAS. *(Ger.)* Nitschke, U. (Acad. Med. Erfurt, E. Germany) and K. Link. *Z Innere Med* 27(20):896-905, 1972.

2198 THE CALCIUM-LOSING KIDNEY AND ITS METABOLIC EFFECTS IN MULTIPLE MYELOMA. *(Ger.)* Kaiser, W. (M. Luther U. Med. Clin. Halle-Wittenberg, E. Germany), H. Krosch and G. A. Schmidt. *Z Innere Med* 27(22):995-999, 1972.

2199 THE RELATIONSHIP BETWEEN BRUCELLOSIS AND LYMPHOGRANULOMATOSIS. *(Ger.)* Abendroth, . (F. Schiller U. Med. Clin. Jena, E. Germany) and E. Ebner. *Z Innere Med* 28(4):125-128, 1973.

2200 ON MALIGNOLIPIN. XX. REASONS OF THE FAILURE IN THE ISOLATION OF MALIGNOLIPIN BY WAY OF PICRATE METHOD. *(E.)* Kosaki, T. (Sch. Med., Mie U., Tsu, Japan), S. Nakagawa, T. Saka, M. Tanaka, M. Kawase, M. Ito and H. Hibasami. *Proc Jap Acad* 48(5):330-335, 1972.

2201 ON MALIGNOLIPIN. XIX. AN ATTEMPT TO DETECT SPERMINE WITH MALACHITE POWDER. *(E.)* Kosaki, T. (Sch. Med., Mie U., Tsu, Japan) and H. Hibasami. *Proc Jap Acad* 48(5):327-329, 1972.

2202 INTRODUCTORY REMARKS: MEMBRANE-POLYSOME INTERACTIONS. *(E.)* Pitot, H. C. (McArdle Labs., Madison, Wisconsin) and T. K. Shires. *Fed Proc* 32(1):76-79, 1973.

2203 GENETIC REGULATION OF MEMBRANE FUNCTION IN MAMMALIAN CELLS IN CULTURE. *(E.)* Till, J. E. (Ontario Cancer Inst., Canada), R. M. Baker, D. M. Brunette, V. Ling, L. H. Thompson and J. A. Wright. *Fed Proc* 32(1):29-33, 1973.

2204 A SIMPLE APPARATUS FOR THE CONTINUOUS GROWTH OF EXPERIMENTAL TUMOR CELLS *IN VITRO*. *(E.)* Bloch, A. (Roswell Park Mem. Inst., Buffalo, N.Y.) and G. Dutschman. *Biotechnol Bioeng* 15(1):197-200, 1973.

2205 CHROMOSOMAL CHARACTERISTICS OF SECONDARY HUMAN BRAIN TUMOURS. *(E.)* Mark, J. (Inst. Path., U. Lund, Sweden). *Eur J Cancer* 8(4):399-407, 1972.

2206 THE RELATION BETWEEN SEBUM PRODUCTION AND PLASMA 17-OXOSTEROID LEVELS IN NORMAL WOMEN AND IN PATIENTS WITH BREAST CANCER. *(E.)* Wang, D. Y. (Imperial Cancer Res. Fund, London, England), R. D. Bulbrook, J. Guillebaud and A. Lewis. *Eur J Cancer* 8(4):381-385, 1972.

2207 IDENTIFICATION OF HISTONE MESSENGER RNA FROM HeLa CELLS. APPEARANCE OF HISTONE mRNA IN THE CYTOPLASM AND ITS TRANSLATION IN A RABBIT-RETICULOCYTE CELL-FREE SYSTEM. *(E.)* Brendel, M. (Max-Planck Inst. Cell Biol., Wilhelmshaven, West Germany) and D. Gallwitz. *Eur J Biochem* 32(2):381-391, 1973.

2208 RESCUE OF TRANSFORMING ACTIVITY FROM HUMAN OSTEOSARCOMA CELLS AFTER CO-CULTIVATION WITH HUMAN LEUKEMIC BONE MARROW CELLS. *(E.)* Georgiades, J. (U. Texas M. D. Anderson Hosp., Tumor Inst., Houston), E. S. Priori, R. L. Hales, J. M. Bowen, W. A. Newton and L. Dmochowski. *Proc Am Assoc Cancer Res* 13(March):109, 1972.

2209 THE MISSING Y CHROMOSOME AND HUMAN LEUKAEMIA. *(E.)* Vass, L. (Dept. Path., Robert Karoly Hosp., Budapest, Hungary) and M. Sellyei. *Lancet* (7802):550-551, 1973.

2210 BIOCHEMICAL, HISTOCHEMICAL AND ULTRASTRUCTURAL STUDIES OF CHANGES OCCURRING WITH GROWTH OF EHRLICH ASCITES CELLS *IN VIVO*. *(E.)* Fletcher, P. A. (Dept. Exp. Path. Cancer Res., Sch. Med., Leeds, England). *Eur J Cancer* 8(4):461-469, 1972.

2211 STIMULATED GRANULOCYTOPOIESIS IN MICE BEARING FIBROSARCOMA. *(E.)* Milas, L. (Dept. Biol., Inst. "Rudjer Boskovic", Croatia, Yugoslavia) and I. Basic. *Eur J Cancer* 8(3):309-313, 1972.

2212 MORPHOLOGY OF ASTROCYTOMAS IN TISSUE CULTURE: OPTICAL AND STEREOSCAN MICROSCOPY. *(E.)* Ambrose, E. J. (Chester Beatty Res. Inst., London, England), U. Batzdorf and D. M. Easty. *J Neuropathol Exp Neurol* 31(4):596-610, 1972.

2213 MALIGNANT GLIOMA OF THE BRAIN-STEM. A CLINICOPATHOLOGICAL ANALYSIS OF 13 CASES. *(E.)* Golden, G. S. (Montefiore Hosp. Med. Ctr., Bronx, New York), N. R. Ghatak, A. Hirano and J. H. French. *J Neurol Neurosurg Psychiatry* 35(5):732-738, 1972.

2214 STUDIES ON THE ACCUMULATION MECHANISMS OF ^{67}GA IN TUMORS: ROLE OF CALCIUM METABOLISM. *(E.)* Anghileri, L. J. (Ruhr U. Tumor Res. Section, Essen, W. Germany). *Strahlentherapie* 144 (1):29-34, 1972.

2215 ULTRASTRUCTURE OF A SIMPLE AMELOBLASTOMA. *(E.)* Lee, K. W. (Inst. Dental Surgery, U. London, England), N. G. El-Labban and I. R. H. Kramer. *J Pathol* 108(2):173-176, 1972.

2216 ENZYME ACTIVITIES IN MEMBRANES FROM THREE PHENOTYPES OF THE MURINE PLASMOCYTOMA MOPC 173, CULTIVATED *IN VITRO*. *(E.)* Lelievre, L. (Inst. Molec. Biol., Paris, France) and A. Paraf. *Biochim Biophys Acta* 291(3):671-679, 1973.

2217 BIOSTEREOMETRIC ANALYSIS OF BREAST CHANGES DURING MENSTRUAL CYCLE. *(E.)* Liebelt, R. A. (Med. Coll. Georgia, Augusta), K. Mulkey, R. Kaufman, J. Cuzzi and R. E. Herron. *Proc Am Assoc Cancer Res* 13(March):107, 1972.

2218 DEMONSTRATION OF CHOLINERGIC CELLS IN HUMAN NEUROBLASTOMA AND GANGLIONEUROMA. *(E.)* Prasad, K. N. (U. Colorado Med. Ctr., Denver), B. Mandal and S. Kumar. *J Pediatr* 82(4):677-679, 1973.

2219 INHIBITION OF LYMPHOBLASTIC RESPONSE TO CANCER CELLS BY PLASMA SIALOGLYCOPROTEINS. *(E.)* Watkins, E., Jr. (Lahey Clin. Fdn., Boston, Mass.) and L. L. Anderson. *Proc Am Assoc Cancer Res* 13(March):110, 1972.

2220 THE DIGESTION OF CELLULAR FRAGMENTS WITHIN PHAGOLYSOSOMES IN CARCINOMA CELLS. *(E.)* Kerr, J. F. R. (U. Queensland Med. Sch., Australia) and J. Searle. *J Pathol* 108(1):55-58, 1972.

2221 ULTRASTRUCTURAL STUDIES ON THE ACUTE LEUKEMIC MONOBLAST. *(E.)* Szekely, I. E. (Inst. Path., Harrisburg Hosp., Pa.), S. A. Park and H. R. Schumacher. *Blut* 25(6):376-384, 1972.

2222 A COMPARATIVE STUDY OF TRANSFER RIBONUCLEIC ACIDS EXTRACTED FROM RAT LIVER AND ZAJDELA HEPATOMA. II. COMPARING THE CHROMATOGRAPHIC FRACTIONATION PROFILES OF AMINOACYL-tRNA FROM RAT LIVER AND ZAJDELA HEPATOMA. *(Fr.)* Befort, J.-J (Dept. Pharm. U. Strasbourg, France). *Biochimie* 54 (10):1327-1333, 1972.

2223 NORMALIZATION BY NUCLEOTIDES OF IMPAIRMENTS IN DEAMINATION OF NITROGENOUS COMPOUNDS IN LIVER OF TUMOR-BEARING MICE. *(E.)* Gorkin, V. Z. (Inst. Biol. Med. Chem., Acad. Med. Sci. USSR, Moscow), E. A. Neyfakh, L. A. Romanova and M. Khuzhamberdiev. *Experientia* 29(1):22-23, 1973.

2224 URINARY CHOLESTEROL. V. ITS EXCRETION IN MEN WITH TESTICULAR AND PROSTATIC NEOPLASMS. *(E.)* Acevedo, H. F. (W. H. Singer Mem. Res. Inst., Pittsburgh, Pa.), E. A. Campbell, E. L. Saier, J. C. Frich, Jr., L. P. Merkow, D. W. Hayeslip, S. P. Bartok, R. C. Grauer and J. L. Hamilton. *Cancer* 32(1):196-205, 1973.

2225 INHIBITION BY TEREPHTHALIC ACID OF SPONTANEOUS MAMMARY TUMORIGENESIS IN MICE. *(E.)* Nagasawa, H. (Natl. Cancer Ctr. Res. Inst., Tokyo, Japan) and M. Fujimoto. *Experientia* 29(1):89-90, 1973.

2226 DOUBLE-STRANDED RNA AS AN INHIBITOR OF PROTEIN SYNTHESIS AND AS A SUBSTRATE FOR A NUCLEASE IN EXTRACTS OF KREBS II ASCITES CELLS. *(E.)* Robertson, H. D. (M.R.C. Lab. Molecular Biol., Cambridge, England) and M. B. Mathews. *Proc Natl Acad Sci USA* 70(1):225-229, 1973.

2227 REACTIVE CHANGES OCCURRING IN THE THYROID GLAND AND ADRENAL CORTEX DURING THE METASTASIZING PROCESS OF BROWN-PEARS CARCINOMA. *(Rus.)* Kolpashchikova, I. F. (S. M. Kirov Med. Ins Gor'kiy, USSR) and V. I. Pudov. *Vopr Onkol* 19(1): 108, 1973.

2228 REVERSE TRANSCRIPTASE IN TRANSFORMED CELL LINES DERIVED FROM HUMAN NEOPLASMS. *(E.)* Newton, W. A. (U. Texas M. D. Anderson Hosp., Tumor Inst., Houston), P. T. Allen, K. Maruyama and L. Dmochowski. *Proc Am Assoc Cancer Res* 13(March):108 1972.

229 SCLEROSING INFLAMMATION OF THE SUBMANDIBULAR SALIVARY GLAND (KÜTTNER TUMOUR). A
ROGRESSIVE PLASMACELLULAR DUCTITIS. *(E.)* Räsänen,
. (Dept. Path., U. Oulu, Finland), K. Jokinen and
. Dammert. *Acta Otolaryngol (Stockh)* 74(4):297-301,
972.

230 PLASMA CELL GRANULOMA (HISTIOCYTOMA) OF THE LUNG AND PLEURA: REPORT ON THREE
ASES. *(E.)* Mäkelä, V. (Central Lab. Path., U.
elinski, Finland), S. Mattila and J. Mäkinen.
cta Pathol Microbiol Scand[*a*] 80(5):634-640, 1972.

231 ELECTRON MICROSCOPIC STUDIES OF PALMAR AND PLANTAR PITS OF NEVOID BASAL CELL
PITHELIOMA. *(E.)* Hashimoto, K. (Memphis VA
osp., Tennessee), J. B. Howell, Y. Yamanishi, K.
olubar and R. Bernhard, Jr. *J Invest Dermatol*
9(5):380-393, 1972.

232 A PATHOLOGICAL STUDY ON THE JUVENILE TYPE OF CHRONIC MYELOID LEUKEMIA. *(E.)*
akanashi, R. (Dept. Path., Tokyo U., Japan).
cta Pathol Jap 22(3):489-508, 1972.

233 ARGYROPHIL CELL CARCINOID OF THE STOMACH. A LIGHT- AND ELECTRON-MICROSCOPIC OBSER-
ATION. *(E.)* Soga, J. (U. Niigata Sch. Med.,
apan), K. Tazawa, K. Wada, M. Fujimaki, K. Saito
nd K. Ikarashi. *Acta Pathol Jap* 22(3):541-553,
972.

234 VAGARIES OF RENAL CELL CARCINOMA. *(E.)* Wagle, D. G. (Roswell Park Mem. Inst.,
uffalo, N.Y.). *J Med (Basel)* 3(3):178-189, 1972.

235 MUCOPOLYSACCHARIDE SULFATION IN NORMAL AND LEUKEMIC LEUKOCYTES. *(E.)* Lau, P.
J. Pennsylvania Sch. Med., Philadelphia), A. J.
ottlieb and W. J. Williams. *Blood* 40(5):725-732,
972.

236 CARCINOMA OF THE BILHARZIAL BLADDER IN EGYPT. CLINICAL AND PATHOLOGICAL FEA-
JRES. *(E.)* El Boulkany, M. N. (Dept. Path.,
ancer Inst., Cairo, Egypt), M. A. Ghoneim and
. A. Mansour. *Br J Urol* 44(5):561-570, 1972.

237 AN AUTOPSY STUDY OF METASTATIC SITES OF BREAST CANCER. *(E.)* Viadana, E.
Roswell Park Mem. Inst., Buffalo, N.Y.), R. Cotter,
. W. Pickren and I. D. J. Bross. *Cancer Res*
3(1):179-181, 1973.

238 INDIVIDUAL *VERSUS* GROUP CAGING OF MICE WITH GRAFTED TUMORS. *(E.)* Dechambre,
-P. (Gustave-Roussy Inst., Villejuif, France)
d C. Gosse. *Cancer Res* 33(1):140-144, 1973.

2239 BONE AND CARTILAGE IN MALIGNANT TUMOURS OF THE URINARY BLADDER. *(E.)* Delides, G. S. (Dept. Path., U. Athens, Greece). *Br J Urol* 44(5):571-581, 1972.

2240 SOLITARY TESTICULAR METASTASIS FROM CARCINOMA OF THE PROSTATE. *(E.)* Jepson, P. M. (VA Hosp., Long Beach, Calif.), H. G. Labitzke and A. J. Bischoff. *Br J Urol* 44(5):594-599, 1972.

2241 IMPLANTATION OF V X 2 CARCINOMA INTO THE RABBIT STOMACH. *(E.)* Vargha, Gy. (U. Med. Sch., Debrecen, Hungary), L. Miltényl, L. Szücs, P. Endes and T. Szilágyi. *Acta Chir Acad Sci Hung* 13(1):93-103, 1972.

2242 EFFECTS OF PHOSPHOLIPIDS AND DIMETHYLSULFOXIDE ON THE ACTIVITY OF RNA POLYMERASE A FROM MOUSE MYELOMA CELLS. *(E.)* Lezius, A. (Max-Planck-Inst. Exp. Med., Göttingen, West Germany) and B. Müller-Lorsen. *Hoppe Seylers Z Physiol Chem* 353(12):1872-1876, 1972.

2243 BASAL CELL ADENOMA INVOLVING THE LIP. *(E.)* Klein, H. Z. (Mount Zion Hosp. Med. Ctr., San Francisco, Calif.) and R. L. Goldman. *Arch Path* 95(2):94-96, 1973.

2244 FAMILIAL OCCURRENCE OF THYMOMA. *(E.)* Matani, A. (Dept. Path. Anatomy, U. Athens, Greece) and C. Dritsas. *Arch Pathol* 95(2):90-91, 1973.

2245 EFFECT OF POTASSIUM IONS ON THE ELECTRICAL CHARACTERISTICS OF THE CELLS. *(E.)* Polivoda, B. I. (USSR Acad. Med. Sci., Obninsk), A. P. Nasonov and O. M. Polivoda. *Biofizika* 17(1):175-177, 1972.

2246 DEOXYRIBONUCLEIC ACID SYNTHESIS IN MAMMALIAN SYSTEMS. PERMEALYSED EHRLICH ASCITES CELLS *IN VITRO* FIRST LABEL OKAZAKI-TYPE LOW-MOLECULAR-WEIGHT DEOXYRIBONUCLEIC ACID AND THEN HIGH-MOLECULAR-WEIGHT DEOXYRIBONUCLEIC ACID. *(E.)* Burgoyne, L. A. (Sch. Biol. Sci., Flinders U. South Australia, Bedford Park). *Biochem J* 130(4):959-964, 1972.

2247 COMPARATIVE STUDIES OF SUBCUTANEOUS AND INTRADERMAL LEUKEMIC TUMORS IN GUINEA PIGS. *(E.)* Gross, L. (VA Hosp., Bronx, New York), D. G. Feldman, T. Ehrenreich, Y. Dreyfuss and L. A. Moore. *Cancer Res* 33(2):293-301, 1973.

2248 RADIOIRON INCORPORATION BY A TRANSPLANTABLE LYMPHOID TUMOR. *(E.)* Chandler, F. W., Jr. (Coll. Vet. Med., U. Georgia, Athens) and O. J. Fletcher, Jr. *Cancer Res* 33(2):342-350, 1973.

2249 INFILTRATING LOBULAR CARCINOMA OF THE BREAST. *(E.)* Ashikari, R. (Mem. Hosp., New York, N.Y.), A. G. Huvos, J. A. Urban and G. F. Robbins. *Cancer* 31(1):110-116, 1973.

2250 TUMORS OF MINOR SALIVARY ORIGIN. A CLINICOPATHOLOGIC STUDY OF 492 CASES. *(E.)* Spiro, R. H. (Mem. Hosp. Cancer Allied Dis., New York, N.Y.), L. G. Koss, S. I. Hajdu and E. W. Strong. *Cancer* 31(1):117-129, 1973.

2251 MALIGNANT SCHWANNOMA. A CLINICOPATHOLOGIC STUDY. *(E.)* Ghosh, B. C. (Memorial Sloan-Kettering Cancer Ctr., New York, N.Y.), L. Ghosh, A. G. Huvos and J. G. Fortner. *Cancer* 31(1):184-190, 1973.

2252 PLASMA CELL GRANULOMAS OF THE LUNG. *(E.)* Bahadori, M. (Dept. Pathol., U. California, San Diego, La Jolla) and A. A. Liebow. *Cancer* 31(1):191-208, 1973.

2253 ADENOCARCINOMA OF BARTHOLIN'S GLAND ASSOCIATED WITH PAGET'S DISEASE OF VULVAR AREA. *(E.)* Tchang, F. (Coll. Phys. Surgeons, Columbia U., New York, N.Y.), T. Okagaki and R. M. Richardt. *Cancer* 31(1):221-225, 1973.

2254 CARCINOMA OF THE COLON AND RECTUM: CIRCULATING MALIGNANT CELLS AND FIVE-YEAR SURVIVAL. *(E.)* Griffiths, J. D. (Royal Marden Hosp., England), J. A. McKinna, H. D. Rowbotham, P. Tsolakidis and A. J. Salsbury. *Cancer* 31(1):226-236, 1973.

2255 MYXOMA OF THE JAW BONES. *(E.)* Ghosh, B. C. (Memorial Sloan-Kettering Cancer Ctr., New York, N.Y.), A. G. Huvos, F. P. Gerold and T. R. Miller. *Cancer* 31(1):237-240, 1973.

2256 EXTRATHORACIC ANGIOFOLLICULAR LYMPHOID HYPERPLASIA WITH COINCIDENTAL MYASTHENIA GRAVIS. *(E.)* Emson, H. E. (St. Paul's Hosp., Saskatoon, Saskatchewan, Canada). *Cancer* 31(1): 241-245, 1973.

2257 REJECTION OF ASCITES TUMOR ALLOGRAFTS. II. A PATHWAY FOR CELL-MEDIATED TUMOR DESTRUCTION *IN VITRO* BY PERITONEAL EXUDATE LYMPHOID CELLS. *(E.)* Berke, G. (Duke U. Med. Ctr., Durham, North Carolina), K. A. Sullivan and B. Amos. *J Exp Med* 136(6):1594-1604, 1972.

2258 DETERMINATION OF PRIMARY SITE BY EXAMINATION OF CANCER CELLS IN BODY FLUIDS. *(E.)* Murphy, W. M. (Inst. Path., Case Western Reserve U., Cleveland, Ohio) and A. B. P. Ng. *Am J Clin Pathol* 58(5):479-488, 1972.

2259 OXYPHILIC ADENOMA OF THE HUMAN THYROID. A MORPHOLOGICAL AND BIOCHEMICAL STUDY. *(E.)* Heimann, P. (Dept. Anatomy, U. Göteborg, Sweden), J.-G. Ljunggren, T. Löwhagen and B. Hjern. *Cancer* 31(1):246-254, 1973.

2260 HEMANGIOPERICYTOMA. AN ULTRASTRUCTURAL STUDY. *(E.)* Hahn, M. J. (U. Missouri Sch. Med., Columbia, Mo.), R. Dawson, J. A. Esterly and D. J. Joseph. *Cancer* 31(1):255-261, 1973.

2261 CALCIUM BINDING TO PHOSPHOLIPIDS FROM EXPERIMENTAL TUMORS. *(E.)* Anghileri, L. J. (Ruhr U. Tumor Res. Clin., Essen, West Germany). *Z Krebsforsch* 78:337-344, 1972.

2262 SELECTIVE SECRETION OF LYSOSOMAL ENZYMES OF THE YOSHIDA HEPATOMA AH 130 IN THE PERITONEAL FLUID. *(E.)* Hegner, D. (J Liebig-U., Inst. Pharmacol. Toxicol., Giessen, West Germany) and D. Platt. *Z Krebsforsch* 78:310-316, 1972.

2263 MAINTENANCE OF NORMAL, METAPLASTIC, AND DYSPLASTIC STATES OF ADULT HUMAN BRONCHIAL MUCOSA IN ORGAN CULTURE. *(E.)* O'Donnell, T. V. (Sch. Med., U. California, San Francisco), T. T. Crocker and L. L. Nunes. *Cancer Res* 33(1): 78-87, 1973.

2264 IDENTIFICATION OF THE ENZYMATIC PATHWAYS OF NUCLEOTIDE METABOLISM IN HUMAN LYMPHOCYTES AND LEUKEMIA CELLS. *(E.)* Scholar, E. M. (Roger Williams Gen. Hosp., Providence, Rhode Island) and P. Calabresi. *Cancer Res* 33(1): 94-103, 1973.

2265 A NEW INBRED SUBLINE OF MICE (129/terSv) WITH A HIGH INCIDENCE OF SPONTANEOUS CONGENITAL TESTICULAR TERATOMAS. *(E.)* Stevens, L. C. (Jackson Lab., Bar Harbor, Maine). *J Natl Cancer Inst* 50(1):235-242, 1973.

2266 PREPARATION OF PURE CULTURES OF TUMOR MACROPHAGES. *(E.)* Evans, R. (Chester Beatty Res. Inst., Sutton, England). *J Natl Cancer Inst* 50(1):271-273, 1973.

2267 THYMIDINE TRANSPORT BY CULTURED NOVIKOFF HEPATOMA CELLS AND UPTAKE BY SIMPLE DIFFUSION AND RELATIONSHIP TO INCORPORATION INTO DEOXYRIBONUCLEIC ACID. *(E.)* Plagemann, P. G. W. (Med. Sch., U. Minnesota, Minneapolis) and J. Erbe. *J Cell Biol* 55(1):161-178, 1972.

2268 FRACTIONATION AND PARTIAL CHARACTERIZATIO OF PROTEINS OF THE BILEAFLET NUCLEAR MEMBRANE FROM RAT LIVER. *(E.)* Bornens, M. (McArdle Lab. Cancer Res., U. Wisconsin, Madison) and C. B. Kasper. *J Biol Chem* 248(2):571-579, 1973.

269 MODES OF SPREAD OF HODGKIN'S DISEASE. *(E.)* Lillicrap, S. C. (Royal Marsden osp., Sutton, England). *Br J Radiol* 46(541):18-3, 1973.

270 BIOSYNTHESIS OF N-ACETYLNEURAMINIC ACID IN MORRIS HEPATOMAS. *(E.)* Harms, E. Biochem. Inst., U. Freiburg, Breisgau, West ermany), W. Kreisel, H. P. Morris and W. Reutter. *ur J Biochem* 32(2):254-262, 1973.

271 ESTROGEN RECEPTORS IN HUMAN BREAST CANCER. *(E.)* McGuire, W. L. (U. Texas Med. Sch., an Antonio). *J Clin Invest* 52(1):73-77, 1973.

272 CALCIUM TRANSPORT IN INTACT EHRLICH ASCITES TUMOR CELLS. *(E.)* Cittadini, A. U. Pennsylvania, Sch. Med., Philadelphia), . Scarpa and B. Chance. *Biochim Biophys Acta* 91(1):246-259, 1973.

273 GALACTOSYLTRANSFER IN MOUSE MASTOCYTOMA: PURIFICATION AND PROPERTIES OF *N*-CETYLLACTOSAMINE SYNTHETASE. *(E.)* Helting, T. Inst. Med. Chem., U. Uppsala, Sweden) and . Erbing. *Biochim Biophys Acta* 293(1):94-104, 973.

274 PHENYLALANINE HYDROXYLASE IN CULTURED HEPATOCYTES. HORMONAL CONTROL OF ENZYME EVELS. *(E.)* Haggerty, D. F. (UCLA Sch. Med., os Angeles, Calif.), P. L. Young, G. Popják and . H. Carnes. *J Biol Chem* 248(1):223-232, 1973.

275 SYNTHETIC TEMPLATE SPECIFICITY OF A DEOXYRIBONUCLEIC ACID POLYMERASE FROM EGENERATING RAT LIVER. *(E.)* De Recondo, A.-M. Inst. Sci. Res. Cancer, Villejuif, France), .-A. Lepesant, O. Fichot, L. Grasset, J.-M. ossignol and M. Cazillis. *J Biol Chem* 248(1): 31-137, 1973.

276 STOMACH CANCER AND LACTATE DEHYDROGENASE IN GASTRIC JUICE. *(E.)* Simon, L. Jaszbereny Hosp., Hungary) and I. A. Figus. *ancet* (7796):204, 1973.

277 FAMILIAL OCCURRENCE OF METASTASIZING CARCINOID TUMORS. *(E.)* Moertel, C. G. Mayo Clin. Fdn., Rochester, Minn.) and M. B. ockerty. *Ann Intern Med* 78(3):389-390, 1973.

278 EVIDENCE THAT THE BONE RESORPTION-STIMULATING FACTOR PRODUCED BY MOUSE FIBROSARCOMA ELLS IS PROSTAGLANDIN E_2. A NEW MODEL FOR THE YPERCALCEMIA OF CANCER. *(E.)* Tashjian, A. H., Jr. Harvard Sch. Dental Med., Boston, Mass.), E. F. oelkel, L. Levine and P. Goldhaber. *J Exp Med* 36(6):1329-1343, 1972.

2279 VARIABILITY OF HORMONE CONCENTRATIONS AND RATIOS IN COMMERCIAL SERA USED FOR TISSUE CULTURE. *(E.)* Esber, H. J. (Mason Res. Inst., Worcester, Mass.), I. J. Payne and A. E. Bogden. *J Natl Cancer Inst* 50(2):559-562, 1973.

2280 MALIGNANT MESENCHYMOMA OF THE LUNG. *(E.)* Kalus, M. (Baylor Coll. Med., Houston, Tex.) F. Rahman, D. E. Jenkins and A. C. Beall, Jr. *Arch Patol* 95(3):199-202, 1973.

2281 YOLK SAC CARCINOMA GROWN FROM MOUSE EGG CYLINDER. *(E.)* Damjanov, I. (Dept. Path., U. Zagreb, Yugoslavia) and D. Solter. *Arch Pathol* 95(3):182-184, 1973.

2282 HYALINE GLOBULES IN HUMAN NEOPLASMS. A REPORT OF THREE AUTOPSY CASES. *(E.)* Dekker, A. (Dept. Path., Presbyterian-U. Hosp., Pittsburgh, Pa.) and J. R. Krause. *Arch Pathol* 95(3):178-181, 1973.

2283 MYOEPITHELIOMA OF THE PALATE. HISTOCHEMICAL AND ULTRASTRUCTURAL OBSERVATIONS. *(E.)* Kahn, L. B. (U. Cape Town Med. Sch., South Africa) and L. Schoub. *Arch Pathol* 95(3):209-212, 1973.

2284 MALIGNANT GIANT CELL TUMOR OF THE TENDON SHEATH. ULTRASTRUCTURAL STUDY AND REVIEW OF THE LITERATURE. *(E.)* Kahn, L. B. (U. Cape Town Med. Sch., South Africa). *Arch Pathol* 95(3):203-208, 1973.

2285 ACTIVITIES AND ISOENZYME PATTERNS OF SOME ENZYMES OF GLUCOSE METABOLISM IN HUMAN PRIMARY MALIGNANT HEPATOMA. *(E.)* Balinsky, D. (South African Inst. Med. Res., Johannesburg), E. Cayanis, E. W. Geddes and I. Bersohn. *Cancer Res* 33(2):249-255, 1973.

2286 ALVEOLAR CELL CARCINOMA. AN *IN VITRO* STUDY. *(E.)* Coalson, R. E. (U Oklahoma Hlth. Sci. Ctr., Oklahoma City), R. E. Nordquist, J. J. Coalson, J. A. Mohr and E. R. Rhoades. *Lab Invest* 28(1):38-47, 1973.

2287 NATURALLY OCCURRING BONE TUMORS IN C57BL/Icrf MICE. *(E.)* Franks, L. M. (Imperial Cancer Res. Fund, London, England), C. Rowlatt and F. C. Chesterman. *J Natl Cancer Inst* 50(2):431-438, 1973.

2288 SKIN TUMORS IN DOMESTIC ANIMALS. DATA FROM 12 UNITED STATES AND CANADIAN COLLEGES OF VETERINARY MEDICINE. *(E.)* Priester, W. A. (Natl. Cancer Inst., Bethesda, Md.). *J Natl Cancer Inst* 50(2):457-466, 1973.

2289 TUMORS OF THE MANDIBLE AND MAXILLA IN CHILDREN. I. CLINICOPATHOLOGIC STUDY OF 46 HISTOLOGICALLY BENIGN LESIONS. *(E.)* Dehner, L. P. (Washington U. Sch. Med., St. Louis., Mo.). *Cancer* 31(2):364-384, 1973.

2290 MIXED (ADENOSQUAMOUS) CARCINOMA OF THE ENDOMETRIUM: ELECTRON MICROSCOPIC OBSERVATIONS. *(E.)* Aikawa, M. (Inst. Path., Case Western Reserve U., Cleveland, Ohio) and B. P. Alan. *Cancer* 31(2):385-397, 1973.

2291 RETROPERITONEAL XANTHOGRANULOMA AND XANTHOSARCOMA (MALIGNANT FIBROUS XANTHOMA). *(E.)* Kahn, L. B. (U. Cape Town Med. Sch., South Africa). *Cancer* 31(2):411-422, 1973.

2292 GRANULOCYTIC SARCOMA (CHLOROMA), TWO YEARS PRECEDING MYELOGENOUS LEUKEMIA. *(E.)* Mason, T. E. (Dept. Path., Fitzsimons Gen. Hosp., Denver, Colo.) R. S. Demaree, Jr. and C. I. Margolis. *Cancer* 31(2):423-432, 1973.

2293 MEDULLARY CARCINOMA OF THYROID GLAND. SUBCELLULAR DISTRIBUTION OF CALCITONIN AND RELATIONSHIP BETWEEN GRANULES AND AMYLOID. *(E.)* Meyer, J. S. (Washington U. Sch. Med., St. Louis, Mo.) W. E. Hutton and A. D. Kenny. *Cancer* 31(2):433-441, 1973.

2294 EMBRYONAL RHABDOMYOSARCOMA OF THE PROSTATE. AN ULTRASTRUCTURAL STUDY. *(E.)* Sarkar, K. (Dept. Path., U. Ottawa, Ontario, Canada), G. Tolnai and D. E. McKay. *Cancer* 31(2):442-448, 1973.

2295 INTRAHEPATIC BILE DUCT CARCINOMA AND *C. SINENSIS* INFECTION IN HONG KONG. *(E.)* Belamaric, J. (Wayne St. U. Sch. Med., Detroit, Mich.). *Cancer* 31(2):468-473, 1973.

2296 SPONGIOBLASTOMAS, ASTROCYTOMAS AND ROSENTHAL FIBERS. ULTRASTRUCTURAL, TISSUE CULTURE AND ENZYME HISTOCHEMICAL INVESTIGATIONS. *(E.)* Gullotta, F. (Inst. Neuropathol., U. Bonn, Germany) and E. Fliedner. *Acta Neuropathol (Berl)* 22:68-78, 1972.

2297 CEREBRAL MEDULLOEPITHELIOMA. *(E.)* Jellinger, K. (Neurological Inst. U. Vienna, Austria). *Acta Neuropathol (Berl)* 22:95-101, 1972.

2298 THE FINE STRUCTURE OF BLOOD VESSELS IN CHROMOPHOBE ADENOMA. *(E.)* Hirano, A. (Montefiore Hosp. Med. Ctr., Bronx, N.Y.), U. Tomiyasu and H. M. Zimmerman. *Acta Neuropathol (Berl)* 22:200-207, 1972.

2299 RNase AND ALKALI SENSITIVITY OF CLOSED CIRCULAR MITOCHONDRIAL DNA OF RAT ASCITES HEPATOMA CELLS. *(E.)* Miyaki, M. (Japanese Fdn. Cancer Res., Tokyo), K. Koide and T. Ono. *Biochem Biophys Res Comm* 50(2):252-258, 1973.

2300 INTERACTION OF CALCIUM WITH MITOCHONDRIA ISOLATED FROM EHRLICH ASCITES TUMOUR CELLS *(E.)* Thorne, R. F. W. (Dept. Biochem., Australian National U., Canberra) and F. L. Bygrave. *Biochem Biophys Res Commun* 50(2):294-299, 1973.

2301 POLYADENYLIC ACID SEQUENCES IN ASCITES CELLS NUCLEAR PARTICLES AND MEMBRANE-BOUND MESSENGER RNA. *(E.)* Cornudella, L. (New York Blood Ctr., New York), I. Faiferman and A. O. Pogo. *Biochem Biophys Acta* 294(3):541-546, 1973.

2302 METABOLIC REGULATION OF STEROIDOGENESIS IN ADRENOCORTICAL CARCINOMA CELLS OF RAT. EFFECT OF ADRENOCORTICOTROPIN AND ADENOSINE CYCLIC 3':5'-MONOPHOSPHATE ON CORTICOSTEROIDOGENESIS. *(E.)* Sharma, R. K. (Dept. Biochem., U. Tennessee Med. Units, Memphis). *Eur J Biochem* 32(3):506-512, 1973.

2303 α-AMANITIN INHIBITS TUMOR INDUCTION BY *AGROBACTERIUM TUMEFACIENS.* *(E.)* Beiderbeck, R. (Inst. Botony, U. Heidelberg, West Germany). *Z Naturforsch [B]* 27(11):1393-1394, 1972.

2304 THYMIDINE KINASE IN RAT TISSUES DURING GROWTH AND DIFFERENTIATION. *(Ger.)* Machovich, R. (Harvard Med. Sch., Boston, Mass.) and O. Greengard. *Biochim Biophys Acta* 286(2):375-381, 1972.

2305 ISOLATION OF POLY(ADENYLIC ACID)-RICH RIBONUCLEIC ACID FROM MOUSE MYELOMA AND SYNTHESIS OF COMPLEMENTARY DEOXYRIBONUCLEIC ACID. *(E.)* Faust, C. H., Jr. (Dept. Path., U. Geneva, Switzerland), H. Diggelmann and B. Mach. *Biochemistry* 12(5):925-931, 1973.

2306 COMPARATIVE KINETIC STUDY OF HUMAN PYRUVATE KINASES ISOLATED FROM ADULT AND FETAL LIVERS AND FROM HEPATOMA. *(E.)* Balinsky, D. (South African Inst. Med. Res., Johannesburg), E. Cayanis and I. Bersohn. *Biochemistry* 12(5):863-870, 1973.

2307 TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE IN A CASE OF CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA. *(E.)* McCaffrey, R. (Dept. Biol., Massachusetts Inst. Tech., Boston), D. F. Smoler and D. Baltimore. *Proc Natl Acad Sci USA* 70(2):521-525, 1973.

2308 THE UPTAKE OF PHOSPHOLIPIDS BY EXPERIMENTAL TUMORS. *(E.)* Anghileri, L. J. (Ruhr J. Tumor Res. Clin., Essen, West Germany). *Z Krebsforsch* 78:326-336, 1972.

2309 APPLICABILITY OF THE TNM-SYSTEM ON LUNG CARCINOMA, FIELD TRIAL. PROPOSAL OF ADVANCED TNM-CLASSIFICATION INCLUDING A CATEGORY FOR DIAGNOSTIC CERTAINTY DEGREE. *(E.)* Dold, U. (Ruhr U. Tumor Res. Clin., Essen, West Germany), V. Schneider and F. Krause. *Z Krebsforsch* 78:317-325, 1972.

2310 OCCULT METASTASES TO THE SCALENE LYMPH NODES IN PATIENTS WITH CLINICALLY OPERABLE CARCINOMA OF THE CERVIX. *(E.)* Ketcham, A. S. (Natl. Cancer Inst., Bethesda, Md.), P. B. Chretien, R. C. Hoye, J. D. Harrah, P. J. Deckers, E. V. Sugarbaker, P. T. Taylor and A. S. Rabson. *Cancer* 31(1):180-183, 1973.

2311 PERIANAL AND ANORECTAL COMPLICATIONS IN LEUKEMIA. *(E.)* Sehdev, M. K. (Memorial Hosp. Cancer Allied Dis., New York, N.Y.), M. D. Dowling, Jr., S. H. Seal and M. W. Stearns, Jr. *Cancer* 31(1):149-152, 1973.

2312 LOBULAR CARCINOMA *IN SITU*: INCIDENCE AND TREATMENT. *(E.)* Giordano, J. M. (No affiliation) and C. T. Klopp. *Cancer* 31(1):105-109, 1973.

2313 ABNORMAL EXCRETION OF CORTICOSTEROID SULPHATES IN PATIENTS WITH BREAST CANCER. *(E.)* Ghosh, P. C. (Jessop Hosp. Women, Sheffield, England), E. Lockwood, G. W. Pennington. *Br Med J* (5849):328-330, 1973.

2314 RHEOLOGY OF LEUKOCYTES, LEUKOCYTE SUSPENSIONS, AND BLOOD IN LEUKEMIA. POSSIBLE RELATIONSHIP TO CLINICAL MANIFESTATIONS. *(E.)* Lichtman, M. A. (U. Rochester Sch. Med., N.Y.), A. Gregory and E. Kearney. *J Clin Invest* 52(2):350-358, 1973.

2315 THECA GRANULOSA CELL TUMOR ARISING IN ADRENAL. *(E.)* Orselli, R. C. (Centinela Valley Community Hosp., Inglewood, Calif.) T. J. Bassler. *Cancer* 31(2):474-477, 1973.

2316 MALIGNANT LYMPHOMA OF THE SMALL INTESTINE IN IRAQ (MIDDLE EAST LYMPHOMA). *(E.)* Al-Saleem, T. (Med. Coll., U. Baghdad, Iraq) and Z. Al-Bahrani. *Cancer* 31(2):291-294, 1973.

2317 SERUM COPPER IN HODGKIN'S DISEASE IN CHILDREN. *(E.)* Tessmer, C. F. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.) M. Hrgovcic and J. Wilbur. *Cancer* 31(2):303-315, 1973.

2318 THE SCOPE AND LIMITATIONS OF PLASMA FREE MEDIUM (P.F.M.) IN STUDYING HUMAN LYMPHOCYTE TRANSFORMATION. *(E.)* Stewart, T. H. M. (Ottawa Gen. Hosp., Ontario, Canada), J. E. Harris, R. Bagai, K. Afghan and P. Sattar. *Proc Am Assoc Cancer Res* 13(March):125, 1972.

2319 RNA LABELING: DIAGNOSTIC AND PROGNOSTIC AID IN HODGKIN'S DISEASE. *(E.)* Hyman, G. (Coll. Physicians Surgeons, Columbia U., New York, N.Y.) and B. Fingerhut. *Proc Am Assoc Cancer Res* 13(March):125, 1972.

2320 ACUTE LYMPHOCYTIC LEUKEMIA OF THE CENTRAL NERVOUS SYSTEM: THE ARACHNOID. *(E.)* Price, R. A. (St. Jude Children's Res. Hosp., Memphis, Tenn.) and W. W. Johnson. *Proc Am Assoc Cancer Res* 13(March):124, 1972.

2321 THE ULTRASTRUCTURE OF LABELED AND NON-LABELED MOUSE MAMMARY CARCINOMA CELLS FOLLOWING PROLONGED PERFUSION WITH TRITIATED THYMIDINE. *(E.)* Wylie, C. V. (U. Colorado, Denver), G. B. Pierce and P. K. Nakane. *Proc Am Assoc Cancer Res* 13(March):123, 1972.

2322 A MAMMALIAN DNA REPAIR ENDONUCLEASE. *(E.)* Van Lancker, J. L. (UCLA Med. Sch., Los Angeles, Calif.) and T. Tomura. *Proc Am Assoc Cancer Res* 13(March):122, 1972.

2323 STEROID METABOLISM BY AN ESTROGEN-DEPENDENT INTERSTITIAL-CELL TUMOR OF THE TESTIS IN HOODED RATS. *(E.)* Jull, J. W. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada), S. Coupey and D. McClellan. *Proc Am Assoc Cancer Res* 13(March):122, 1972.

2324 AN ESTROGEN DEPENDENT TESTICULAR CARCINOMA OF THE RAT CAUSING UNUSUAL ANDROGEN ACTIVITY. *(E.)* Noble, R. L. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada). *Proc Am Assoc Cancer Res* 13(March):121, 1972.

2325 CYTOTOXICITY OF A FACTOR ISOLATED FROM HUMAN SPLEEN. *(E.)* Lozzio, C. B. (U. Tennessee Mem. Res. Ctr. Hosp., Knoxville) and B. B. Lossio. *J Natl Cancer Inst* 50(2):535-538, 1973.

2326 SPECIFIC VASCULAR ALTERATIONS IN LYMPHOGRANULOMATOSIS. *(Rus.)* Yarosheva, A. A. (Minsk, USSR). *Vopr Onkol* 18(2):80-81, 1972. 1972.

2327 X-RAY FINDINGS IN HEART TUMORS AND CYSTS. *(Ger.)* Dimitrov, M. (3rd City Hosp. Sofia, Bulgaria) and G. Jovev. *Z Innere Med* 27(21):919-924, 1972.

2328 ULTRASTRUCTURAL FEATURES OF HEPATOCELLULAR TUMORS IN HUMANS. *(Rus.)* Filippova, N. A. (Inst. Exp. Clin. Oncol., Moscow, USSR). *Vopr Oncol* 18(1):55-61, 1972.

2329 ALDOLASE ACTIVITY IN HUMAN AND ANIMAL TUMORS. *(Rus.)* Voronova, L. A. (N. N. Petrov Inst. Oncol., Leningrad, USSR) and S. B. Lebedeva. *Vopr Onkol* 18(11):62-65, 1972.

2330 BIOSYNTHESIS OF PROTEINS TYPICAL FOR LIVER REGENERATION AND HEPATOMA. *(Ukr.)* Danko, I. M. (Inst. Biochem Ukr SSR Acad. Sci., Kiev, USSR) and V. P. Korotkoruchko. *Ukr Biokhim Zh* 44(5):554-558, 1972.

2331 ADENOID SQUAMOUS CELL CARCINOMA OF THE LIP: REPORT OF CASES. *(E.)* Tomich, C. E. (No affiliation) and C. E. Hutton. *J Oral Surg* 30(8):592-598, 1972.

2332 VARIOUS ASPECTS OF GLUTAMIC ACID METABOLISM IN EHRLICH ASCITES TUMOR CELLS. A BIOCHEMICAL AND HISTOCHEMICAL STUDY. *(Sp.)* Toledano, A. (Cajal Inst. Histochem. Lab. Madrid, Spain) and R. Martinez. *Acta Oncol* 9(1):28-39, 1970.

2333 NUCLEAR ARYLSULPHATASE ACTIVITY IN LEUKAEMIA AND SOME OTHER HAEMATOLOGICAL DISORDERS. *(E.)* Petersen, H. S. (Dept. Hematol. U. Aarhus, Denmark). *Scan J Haematol* 10(1):35-43, 1973.

2334 EOSINOPHIL LEUKEMIA, AN IMMATURE-CELL MYELOID LEUKEMIA WITH CHLOROACETATE-POSITIVE EOSINOPHILIA. A MORPHOLOGICAL AND CYTOCHEMICAL STUDY OF MONOPHYLETIC MYELOID LEUKEMIA. *(Ger.)* Schaefer, H.-E. (Köln U. Med. Clin., W. Germany), K.-P. Hellriegel, H.-H. Henneheuser, G. Hübner, J. Zach, R. Fischer and R. Gross. *Blut* 26(1):7-19, 1973

2335 LACTATE DEHYDROGENASE ISOENZYMES OCCURRING IN GENITAL ORGANS OF THE MALE RAT CARRYING TESTICULAR INTERSTITIAL TUMOR GRAFTS. *(Fr.)* Callard, R. (Brest Med. Sch. France). *Experientia* 28(12):1474-1475, 1972.

2336 TUMOR DEVELOPMENT AROUND A TUMOR CELL-CONTAINING DIFFUSION CHAMBER IMPLANTED INTRAPERITONEALLY IN RATS. *(Rus.)* Manoylov, S. Ye. (Central Sci. Res. Inst. Radiol. Leningrad, USSR), A. K. Ryabukha and L. A. Nesterova. *Vopr Onkol* 19(2):98, 1973.

2337 CYTOGENETIC AND ULTRASTRUCTURAL EVIDENCE OF ALTERED DNA METABOLISM IN LEUKEMIC CELLS. *(E.)* Ahearn, M. J. (U. Texas M. D. Anderson Hosp., Tumor Inst., Houston) and J. M. Trujillo. *Proc Am Assoc Cancer Res* 13(March):108, 1972.

2338 THE CAUSES OF ARTERIAL HYPOXIA IN CANCER PATIENTS. *(Rus.)* Pevzner, Yu. D. (Rostov Sci. Res. Inst. Oncol., USSR). *Vopr Onkol* 19(1):49-52, 1973.

2339 A STUDY OF PIGMENT FRACTIONS EXTRACTED FROM A GARDING-PASSY MELANOMA. *(Rus.)* Malama, A. A. (Exp. Enzyme Prep. Production Plant, Vilnyus, USSR). *Vopr Onkol* 19(1):75-77, 1973.

2340 CHROMOSOMAL FEATURES OF A TUMOR WITH ALTERED DRUG RESISTANCE. (AN EXPERIMENT). *(Rus.)* Voytovitskiy, V. K. (Kazakh Sci. Res. Inst. Oncol. Radiol., Alma-Ata, USSR), A. K. Yedygenova and O. K. Kabiyev. *Vopr Onkol* 19(1):77-82, 1973. 1973.

2341 GROWTH CHARACTERISTICS AND METASTASES OF CANCER OF THE UPPER LEFT LOBE OF THE LUNG AND THEIR IMPORTANCE IN DETERMINING THE EXTENT OF SURGICAL INTERVENTION. *(Rus.)* Kabulov, M. K. (N. N. Petrov Sci. Res. Oncol. Leningrad, USSR). *Vopr Onkol* 18(11);16-20, 1972.

2342 THE FUNCTIONAL CONDITION OF THE PITUITARY GLAND-ADRENAL CORTEX SYSTEM IN PATIENTS WITH GASTRIC CANCER. *(Rus.)* Pinskiy, S. B. (Irkuts Med. Inst., USSR) and A. M. Bratslavskiy. *Vopr Onkol* 18(11):21-24, 1972.

2343 THE DNA CONTENT OF SEX CHROMATIN POLYPLOID CANCERS OF THE MAMMARY GLAND. (A CYTOPHOTOMETRIC STUDY). *(Rus.)* Zus', B. A. (Med. Inst. Leningrad, USSR) and N. A. Shlyakhter. *Vopr Onkol* 18(11):50-52, 1972.

2344 THE DIFFERENTIAL ACTIVITY OF ERYTHROCYTIC CATALASE IN PATIENTS WITH LUNG CANCER. *(Rus.)* Komov, V. P. (Inst. Pharmac. Chem., Leningrad, USSR), T. F. Rakhmanina and E. Ya. Daukin. *Vopr Onkol* 18(11):6-8, 1972.

2345 MAMMALIAN DEOXYRIBONUCLEIC ACID LIGASE. ISOLATION OF AN ACTIVE ENZYME-ADENYLATE COMPLEX. *(E.)* Söderhäll, S. (Dept. Chem., Karolinska Inst., Stockholm, Sweden) and T. Lindahl. *J Biol Chem* 248(2):672-675, 1973.

2346 BIOLOGICAL AND BIOCHEMICAL ALTERATIONS IN CULTURED MAMMALIAN CELLS INDUCED BY SUGARS RELATED TO CELL MEMBRANE. *(E.)* Baba, T. (Aichi Cancer Center Res. Inst., Japan), K. Aoki, M. Kaku, N. Kimura, S. Fujii and K. Kojima. *Gann* 63(6):785-794, 1972.

2347 INDUCTION OF CYTOLYSIS OF CULTURED LYMPHOMA CELLS BY ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE AND THE ISOLATION OF RESISTANT VARIANTS. *(E.)* Daniel, V. (Dept. Biochem., U. California, San Francisco), G. Litwack and G. M. Tomkins. *Proc Natl Acad Sci USA* 70(1):76-79, 1973.

3 CYTOGENETIC OBSERVATIONS ON DIFFERENT CELL
LINES IN HODGKIN'S DISEASE. *(E.)* Olinici,
). (Inst. Oncology, Cluj, Romania). *Acta Haematol*
el) 48:283-287, 1972.

9 HETEROMORPHIC PAIR OF METACENTRIC CHROMO-
SOMES WITH FUSED ARMS AND THE PHILADELPHIA
OMOSOME IN A CASE OF ACUTE MYELOID LEUKEMIA. *(E.)*
, M. H. (Goethe U. Dept. Hematol. Frankfort/Main,
ermany). *Acta Haematol (Basel)* 48:312-319, 1972.

) MALIGNANT TUMOURS OF THE THYROID. *(E.)*
Stewart, D. J. (Royal Infirm. Med. Sch.,
deen, Scotland), A. J. Carr, C. A. S. Pegg and
ichie. *J Roy Coll Surg Edinb* 17(6):335-348,
.

INTESTINAL MALAKOPLAKIA: REPORT OF A CASE.
(E.) Wilkey, I. S. (Port Moresby Gen.
., Papua, New Guinea) and L. R. Rubel. *Pathology*
:311-314, 1972.

2 MALIGNANT MENINGIOMA WITH EXTRACRANIAL
METASTASES AND SEEDING OF THE SUBARACHNOID
E AND THE VENTRICLES. *(E.)* Miller, A. A. (Group
., Royal Infirm. Harris Coll., Preston, England)
F. Ramsden. *Pathol Eur* 7(2):167-175, 1972.

3 MEDULLARY CANCER OF THE THYROID GLAND AND
ITS POSSIBLE RELATIONS TO CARCINOIDS. *(E.)*
ath, E. (Dept. Path., St. Michael's Hosp.,
nto, Canada), K. Kovacs and R. C. Ross. *Virchows*
[Pathol Anat] 356(4):281-292, 1972.

4 EPIDERMOID CARCINOMA IN A CUTIS GRAFT AFTER
REPAIR OF AN INCISIONAL HERNIA. *(E.)* Gomez,
(Henry Ford Hosp., Detroit, Mich.), J. H. Wylie
J. L. Ponka. *Rev Surg* 29(6):381-387, 1972.

5 BROMODEOXYURIDINE DEPENDENCE-A NEW MUTA-
TION IN MAMMALIAN CELLS. *(E.)* Davidson,
. (Children's Hosp. Med. Ctr., Boston, Mass.)
M. D. Bick. *Proc Natl Acad Sci USA* 70(1):
-142, 1973.

6 AUTONOMOUS ERYTHROPOIESIS DURING ERYTHRO-
BLASTIC CRISIS OF CHRONIC MYELOCYTIC
KEMIA. *(E.)* Srodes, C. H. (U. Pittsburgh
. Med., Pa.), E. H. Hyde and D. R. Boggs.
lin Invest 52(2):512-515, 1973.

7 EXPRESSION OF DIFFERENTIATED FUNCTIONS IN
HEPATOMA CELL HYBRIDS: ALANINE AMINOTRANS-
ASE. *(E.)* Sparkes, R. S. (Molecular Genetic
., Gif-sur Yvette, France) and M. C. Weiss.
c Natl Acad Sci USA 70(2):377-381, 1973.

2358 SERUM MYELOPEROXIDASE IN LEUKAEMIA AND POLYCYTHAEMIA VERA. *(E.)* Malmquist, J. (Dept. Med., Malmö Gen. Hosp., Sweden). *Scand J Haematol* 9(4):311-317, 1972.

2359 SERUM LACTOFERRIN IN LEUKAEMIA AND POLYCYTHAEMIA VERA. *(E.)* Malmquist, J. (Dept. Med., Malmö Gen. Hosp., Sweden). *Scand J Haematol* 9(4):305-310, 1972.

2360 SERUM MURAMIDASE LEVELS IN ACUTE LEUKEMIA. *(E.)* Seligman, B. R. (Long Island Jewish Med. Ctr., Jamaica, N.Y.), F. Rosner, F. Parise and S. L. Lee. *Am J Med Sci* 264(1):69-82, 1972.

2361 EPITHELIOMA ARISING IN POROKERATOSIS OF MIBELLI. *(E.)* Cort, D. F. (Queen Elizabeth Hosp., Birmingham, England) and A.-H. M. Abdel-Aziz. *Br J Plast Surg* 25(3):318-328, 1972.

2362 GUANYL CYCLASE ACTIVITY IN MORRIS HEPATOMAS 7787, 7795, 7800 AND 9618A-2. *(E.)* Northup, S. J. (Cancer Res. Ctr., Columbia, Mo.), J. S. Barthel, H. D. Brown, S. K. Chattopadhyay and H. P. Morris. *Mo Med* 69(12):934-937, 1972.

2363 CHRONICITY OF MAMMARY CARCINOMA. *(E.)* Herrmann, J. B. (New York Med. Coll., New York). *NY State J Med* 72(18):2310-2314, 1972.

2364 INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION IN OAT-CELL CARCINOMA OF BRONCHUS. AGGRAVATION OF HYPONATRAEMIA BY INTRAVENOUS CYCLOPHOSPHAMIDE. *(E.)* Munro, A. H. G. (Northern Gen. Hosp., Edinburgh, Scotland) and G. K. Crompton. *Thorax* 27(5):640-642, 1972.

2365 PYRIMIDINE METABOLISM IN NORMAL AND TUMOR TISSUES. *(E.)* Imura, H. (Sch. Med., Tokushima U., Japan), H. Okuda, K. Takēda and S. Fujii. *Gann* 63(6):685-692, 1972.

2366 ON THE QUESTION OF CROWN-GALL TUMOR INITIATION BY DNA OF BACTERIOPHAGE PS8. *(E.)* Beiderbeck, R. (Dept. Biol. Sci., Northwestern U., Evanston, Ill.), G. T. Heberlein and J. A. Lippincott. *J Virol* 11(2):345-350, 1973.

2367 FEATURES OF THE X-RAY DIAGNOSTICS OF LUNG HAMARTOMAS. *(Rus.)* Vintergal'ter, S. F. (N.N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Vopr Onkol* 19(1):102-105, 1973.

2368 GLOMIC TUMORS OF THE STOMACH. (TWO CASES). *(Rus.)* Cherkes, V. L. (Inst. Exp. Clin. Oncol., USSR Acad. Med. Sci.) and T. A. Belous. *Vopr Onkol* 19(1):105-107, 1973.

2369 TUMORS OF THE PNEUMOGASTRIC NERVE: REPORTING TWO CASES. *(Fr.)* Gignoux, B. (Red Cross Hosp. Lyon, France), L. M. Patricot, C. Vieville and V. Tran.Minh. *J Fr Otorhinolaryngol* 21(6):504-508, 1972.

2370 A JUXTAGLOMERULAR CELL TUMOUR: LIGHT AND ELECTRON MICROSCOPIC STUDIES OF A RENIN-SECRETING KIDNEY TUMOUR CONTAINING BOTH JUXTAGLOMERULAR CELLS AND MAST CELLS. *(E.)* Phillips, G. (Inst. Med. Vet. Sci., Adelaide, South Australia) and T. M. Mukherjee. *Pathology* 4(3):193-204, 1972.

2371 INCORPORATION OF 2-DEOXY-D-GLUCOSE INTO GLYCOLIPIDS OF NORMAL AND SV40-TRANSFORMED HAMSTER CELLS. *(E.)* Steiner, S. (Baylor Coll. Med., Houston, Tex.) and M. R. Steiner. *Biochim Biophys Acta* 296(2):403-410, 1973.

2372 RELATIONS OF CALCITONIN AND GASTRIN IN THE ZOLLINGER-ELLISON SYNDROME AND MEDULLARY CARCINOMA OF THE THYROID. *(E.)* Sizemore, G. W. (Mayo Clin. Fdn., Rochester, Minn.), V. L. W. Go, E. L. Kaplan, L. J. Sanzenbacher, K. H. Holtermuller and C. D. Arnaud. *N Engl J Med* 288(13):641-644, 1973.

2373 THE RARITY OF SMALL-BOWEL TUMOURS: AN ALTERNATIVE HYPOTHESIS. *(E.)* Bone, G. (Dept. Surgery, U. Newcastle upon Tyne, England) and N. A. Wright. *Lancet* 1(7803):618, 1973.

2374 SOME PROPERTIES OF LYSINE-RICH HISTONES OF NORMAL AND NEOPLASTIC TISSUES. *(E.)* Vescia, A. (Inst. Tumor Studies Ther., Naples, Italy) and L. Cecco. *Ital J Biochem* 21(3):114-121, 1972.

2375 SELECTIVE ARTERIAL SCANNING IN LUNG CANCER DIAGNOSTICS. *(Rus.)* Rabkin, I. Kh. (Sci. Res. Inst. Exp. Clin. Surg. Moscow, USSR), A. L. Matevosov and E. A. Lebedeva. *Vopr Onkol* 19(1): 18-25, 1973.

2376 LUNG SCANNING IN LYMPHOGRANULOMATOSIS. *(Rus.)* Fadeyev, N. P. (N. N. Petrov Sci. Res. Inst. Oncol. Leningrad, USSR), M. L. Gershanovich and S. I. Yushkantseva. *Vopr Onkol* 19(1):25-30, 1973.

2377 DIAGNOSTICS OF INTRAABDOMINAL ESOPHAGEAL CANCER METASTASES BY MEANS OF LAPAROSCOPY. *(Rus.)* Sotnikoy, V. N. (N. I. Pirogov Med. Inst.), A. S. Yermolov, V. I. Litvinov, S. S. Yemel'yanov and V. S. Samsonov. *Vopr Onkol* 19(1):35-38, 1973.

2378 THE IMPORTANCE OF THE ELECTROGASTROGRAM IN THE EVALUATION OF THE GASTRIC MOTOR FUNCTION IN CANCER. *(Rus.)* Androsov, N. S. (Inst. Med. Radiol., Obninsk, USSR), P. P. Firsova and P. I. Tolstykh. *Vopr Onkol* 19(1):39-43, 1973.

2379 CLEAR-CELL ACANTHOMA. *(Jap.)* Ishikawa, K. (Dept. Dermatol., Kawasaki City Hosp., Japa *Jap J Clin Derm* 26(12):1107-1115, 1972.

2380 ACUTE LEUKEMIA WITH C-G CHROMOSOME TRANSLOCATION. *(E.)* Hinkes, E. (U. California Sch. Med., Los Angeles), B. F. Crandall, F. Weber and C. G. Craddock. *Blood* 41(2):259-263, 1973.

2381 THE ISOLATION AND CHEMICAL COMPOSITION OF PREMELANOSOMES AND MELANOSOMES: HUMAN AND MOUSE MELANOMAS. *(E.)* Siakotos, A. N. (Indiana U. Med. Ctr., Indianapolis), V. Patel and A. Cantabon *Biochem Med* 7(1):14-24, 1973.

2382 AUTORADIOGRAPHIC DETECTION OF DNA POLYMERAS CONTAINING NUCLEI IN SARCOMA 180 ASCITES CELLS. *(E.)* Nelson, J. S. R. (Allegheny Gen. Hosp. Pittsburgh, Pa.) and L. M. Schiffer. *Cell Tissue Kinet* 6(1):45-54, 1973.

2383 MORPHOCYTOCHEMICAL STUDIES OF NUCLEOPROTEIN AND GLYCOGEN IN SOME HUMAN SARCOMAS AND ADENOCARCINOMAS. *(E.)* El-Fiky, S. M. (Faculty Sci., U. Lybia, Tripoli), A. M. Kolkaila, D. S. Dawd and R. M. A. Wahab. *Acta Histochem (Jena)* 44(1): 152-162, 1972.

2384 CYSTEINYL DOPA IN HUMAN MALIGNANT MELANOMA. *(E.)* Björklund, A. (Dept. Plastic Surg., U. Lund, Sweden), B. Falck, S. Jacobsson, H. Rorsman, A.-M. Rosengren and E. Rosengren. *Acta Derm Venereol (Stockh)* 52(5):357-360, 1972.

2385 A NUCLEAR MAGNETIC RESONANCE STUDY OF WATER IN TWO MORRIS HEPATOMAS. *(E.)* Hollis, D. P. (Johns Hopkins U. Sch. Med., Baltimore, Md.), L. A. Saryan and H. P. Morris. *Johns Hopkins Med J* 131(6):441-444, 1972.

2386 STUDIES OF THE PROCOAGULANT AND FIBRINOLYTIC ACTIVITY OF PROMYELOCYTES IN ACUTE PROMYELOCYTIC LEUKAEMIA. *(E.)* Gralnick, H. R. (Natl. Inst. Hlth., Bethesda, Md.) and E. Abrell. *Br J Haematol* 24(1):89-99, 1973.

2387 UTILIZATION OF L-GLUTAMINE IN INTERCELLULAR ADHESION: ASCITES TUMOR AND EMBRYONIC CELLS *(E.)* Oppenheimer, S. B. (Dept. Biol., California St U., Northridge). *Exp Cell Res* 77(1-2):175-182, 1973.

2388 KINETICS OF REDOX CHANGES OF NICOTINAMIDE-ADENINE DINUCLEOTIDES IN EHRLICH ASCITES TUMOUR CELLS. *(E.)* Dionisi, O. (Inst. Gen. Path. Rome, Italy), T. Galeotti, T. Terranova and A. Azzi. *FEBS Letters* 30(1):84-88, 1973.

389 DECREASED LEUKOCYTE ALKALINE PHOSPHATASE IN MONOCYTIC LEUKEMIA. *(E.)* Garg, S. K. ew York U. Med. Ctr., New York) and R. Silber. *J Clin Pathol* 58(6):668-674, 1972.

390 PRIMARY MALIGNANT MELANOMA OF THE ESOPHAGUS: AN ELECTRON MICROSCOPIC STUDY. *(E.)* Frable, J. (Med. Coll. Virginia, Richmond), S. Kay and P. hatzki. *Am J Clin Pathol* 58(6):659-667, 1972.

391 TYROSINE HYDROXYLASE ACTIVITY IN NEUROBLASTOMA AND HUMAN ADRENAL GLAND. *(E.)* ashuku, S. (Kyoto Prefectural U. Med., Japan), Takada, T. Sawada and T. Nakamura. *J Lab Clin d* 80(2):190-199, 1972.

392 THE MICROTUBULES OF LEUKAEMIC RIEDER CELLS: AN ULTRASTRUCTURAL STUDY. *(E.)* Cawley, C. (Dept. Med., U. Cambridge, England). *Scand Haematol* 9(5):417-423, 1972.

393 DYSFIBRINOGENAEMIA ASSOCIATED WITH PRIMARY HEPATOMA. *(E.)* Verhaeghe, R. (Dept. nternal Med., U. Leuven, Belgium), B. Van Damme, . Molla and J. Vermylen. *Scand J Haematol* 9(5): 51-458, 1972.

394 FIBROSIS AND CARCINOMA OF THE LUNG IN A FAMILY WITH HAEMOGLOBIN MALMÖ: ANATOMIC INDINGS. *(E.)* Berglund, S. (Dept. Internal Med., almö Gen. Hosp., Sweden) and F. Linell. *Scand J aematol* 9(5):424-432, 1972.

395 DISTURBED MYELOCYTE PROLIFERATION IN ACUTE LEUKAEMIA. *(E.)* Wickramasinghe, S. N. St. Mary's Hosp. Med. Sch., London, England) and . Moffatt. *Scand J Haematol* 9(5):477-479, 1972.

396 ARGINASE IN HUMAN UROGENITAL TUMORS. *(E.)* Yamanaka, H. (Sch. Med., Gunma U., apan), T. Mayuzumi, M. Matsuoka, J. Shimazaki and . Shida. *Gann* 63(6):693-700, 1972.

2397 MICROANGIOPATHIC HAEMOLYTIC ANAEMIA ASSOCIATED WITH HYPERCALCAEMIA IN AN EXPERIMENTAL RAT TUMOUR. *(E.)* Hilgard, P. (Royal Postgrad. Med. Sch., London, England), R. Hohage, W. Schmitt and W. Köhle. *Br J Haematol* 24(2):245-254, 1973.

2398 HEMANGIOPERICYTOMA OF THE THYROID GLAND. *(E.)* Deshmukh, N. (No affiliation) and J. Beck. *Guthrie Clin Bull* 42(2):32-34, 1972.

2399 CARCINOMA OF THE LARYNX: ANALYSIS OF 311 CASES TREATED SURGICALLY. *(E.)* Putney, F. J. (Med. U. South Carolina, Charleston) and C. E. Chapman. *Ann Otol Rhinol Laryngol* 81(4):455-464, 1972.

2400 TUMOR OF THE CILIARY PIGMENT EPITHELIUM. *(E.)* Streeten, B. W. (St. U. New York Upstate Med. Ctr., Syracuse) and J. L. McGraw. *Am J Ophthalmol* 74(3):420-429, 1972.

LIBRARY U. OF I. URBANA

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

AARONSON, S.A.
1948, 1968, 2043*
ABDEL-AZIZ, A.-H.M.
2361*
ABDEL-WAHAB, K.S.
1974
ABE, M.
2121*
ABENDROTH, K.
2199*
ABLASHI, D.V.
1943, 1947, 2064
ABRELL, E.
2386*
ACEVEDO, H.F.
2224*
ADAM, E.
2149
ADAMS, R.A.
2074
ADELOYE, A.
2007*
ADELSTEIN, S.J.
1945
AFGHAN, K.
2318*
AGEYENKO, A.I.
2002
AHEARN, M.J.
2337*
AIKAWA, M.
2290*
AISENBERG, A.C.
2087
AL-BAHRANI, Z.
2316*
AL-SALEEM, T.
2316*
ALAN, B.P.
2290*
ALBACH, R.A.
1974
ALBERT, E.
2128*
ALBRECHT, R.
1890
ALEKSANDROV, V.A.
1893
ALLEN, P.T.
1950, 2228*
ALONI, Y.
2005
ALTANER, C.
2021*
ALTHOFF, J.
1836
ALVAREZ, J.
1987
ALVAREZ, Y.
1987
AMBROSE, C.T.
2102
AMBROSE, E.J.
2212*
AMOS, B.
2257*
AMOS, D.B.
2077
ANDERSON, L.L.
2219*
ANDERSON, W.B.
1976
ANDROSOV, N.S.
2378*
ANGHILERI, L.J.
2214*, 2261*, 2308*
ANIGSTEIN, D.M.
2097
ANIGSTEIN, L.
2097
ANISIMOV, W.N.
1858
ANTHONY, D.D.
1970
ANTHONY, P.P.
2155
AOKI, K.
2346*
AOKI, T.
1982
ARAI, M.
1884
ARMSTRONG, G.R.
1943
ARNAUD, C.D.
2372*
ARON, G.M.
2028*
ASHIKARI, R.
2249*
AUGUST, J.T.
1940
AURELIAN, L.
1949, 2003
AYRE, J.E.
2068
AZZI, A.
2388*
BABA, T.
1993, 2346*
BABKOVA, O.V.
2105
BACHVAROFF, R.
2114*
BAGAI, R.
2318*
BAHADORI, M.
2252*
BAIN, R.W.
1904*
BAKER, R.M.
2203*
BALDI, A.
1880
BALDWIN, R.W.
2070
BALFOUR, I.C.
2177
BALINSKY, D.
2285*, 2306*
BALTIMORE, D.
2307*
BANERJEE, M.R.
1856
BANNIKOV, G.A.
1831
BANSAL, S.C.
2053
BARAHONA, H.H.
2036*
BARANDUN, S.
2081
BARAUD, J.
1867
BARTHEL, J.S.
2362*
BARTLEY, W.
1887
BARTOK, W.P.
2224*
BASIC, I.
2211*
BASILICO, C.
1967
BASSIN, R.H.
1929
BASSLER, T.J.
2315*
BATES, R.R.
1837
BATZDORF, U.
2212*
BAUGHMAN, F.F.
1877
BAUM, J.W.
1912
BAXTER-GABBARD, K.L.
1958
BEALL, A.C.
2280*
BECK, J.
2398*
BECK, J.P.
2178
BECKER, H.
2136
BEFORT, J.-J.
2222*
BEFORT, N.
2178
BEIDERBECK, R.
2303*, 2366*
BELAMARIC, J.
2295*
BELHANI, M.
2106
BELOUS, T.A.
2368*
BEN-BASSAT, H.
2165
BEN-HUR, E.
1915
BENABADJI, M.
2106
BENDICH, A.
2086
BENDIXEN, G.
2089
BENYESH-MELNICK, M.
2027*, 2028*
BERGLUND, S.
2394*
BERKE, G.
2077, 2257*

BERLIN, R.D.
1962
BERNHARD, R., JR.
2231*
BERNSTEIN, E.H.
1936
BERSOHN, I.
2285*, 2306*
BERTRAMS, J.
2128*
BICHLER, K.H.
2096
BICK, M.D.
2355*
BIGNER, D.D.
1986
BILLARDON, C.
2095
BIRG, F.
2107
BISCHOFF, A.J.
2240*
BISHUN, N.P.
1846
BISWAL, N.
2028*
BJOERKLUND, A.
2384*
BJURSELL, G.
1971
BLACK, L.M.
2022*
BLACK, P.H.
2079
BLACKMAN, E.A.
1947
BLAIR, D.G.R.
2164
BLIZNAKOV, E.G.
1996
BLOCH, A.
2204*
BLUNCK, J.M.
1852
BLUNDELL, G.P.
1813*
BOBROW, M.
1923*
BOEHME, U.
2128*
BOGDANOV, G.N.
2132
BOGEL, E.
1889
BOGGS, D.R.
2356*
BOGOVSKII, S.P.
1851
BOILEAU, G.
2074
BONDURANT, M.C.
1925
BONE, G.
2373*
BOODEN, T.
1974
BORENFREUND, E.
2086
BORNENS, M.
2268*
BOSCH, D.A.
1869
BOSE, H.R.
1961
BOWEN, J.M.
1950, 2208*
BOYCE, E.A.
2059
BRAND, I.
1981
BRAND, K.G.
1981
BRATSLAVSKIY, A.M.
2342*
BRENDEL, M.
2207*
BRENNHOVD, I.
2186
BRILL, E.
1855
BRITTINGER, G.
2099
BROMLEY, P.A.
1926
BRONK, B.V.
1843
BROSS, I.D.J.
2237*
BROWN, H.D.
2362*
BROWN, J.M.
2138
BROWN, M.
1977
BRUNETTE, D.M.
2203*
BRYAN, G.T.
1891
BRYAN, R.J.
1835
BUCHSBAUM, H.J.
2184
BUKRINSKAYA, A.G.
1997
BULBROOK, R.D.
2206*
BUDEN, L.C.
1981
BURCH, P.R.J.
1810*
BURGOYNE, L.A.
2246*
BURLAKOVA, YE.B.
2160
BURNS, C.P.
2054
BURTIN, P.
2071
BYERS, M.J.
2182
BYGRAVE, F.L.
2300*
BYKOVSKY, A.F.
1952
CADWALLADER, J.
2036*
CAJONE, F.
1847
CALABRESI, P.
2110, 2264*
CALARCO, P.G.
1965
CALLARD, R.
2335*
CALVO, F.C.
2036*
CAMPBELL, C.E.
1900*
CAMPBELL, E.A.
2224*
CANTABONI, A.
2381*
CANTONI, G.L.
2181
CANTRELL, E.
1829
CAPUTO, A.
1933
CARBONE, G.
1832
CARCHMAN, R.A.
1932
CARDESA, A.
1836
CARIGLIA, N.
1904*
CARNAUD, C
2052
CARNES, W.H.
2274*
CARR, A.J.
2350*
CARR, D.H.
2047
CARRARA, G.
2034*
CASEY, A.C.
1996
CASPARY, E.A.
2058
CASTELLI, L.
1933
CASTLEDEN, C.M.
1842
CASTRO, A.E.
1937
CASTRO, G.A.M.
1898*
CASTRO, S.
1904*
CAWLEY, J.C.
2392*
CAYANIS, E.
2285*, 2306*
CAZILLIS, M.
2275*
CECCO, L.
2374*
CHADWICK, K.H.
1910
CHAN, J.C.
1950
CHANCE, B.
1975, 2272*

CHANDLER, F.W., JR.
2248*
CHAPMAN, C.E.
2399*
CHAPMAN, W.H.
1853
CHATTOPADHYAY, S.K.
2362*
CHEEVERS, W.P.
2001
CHEN, J.H.
2125*
CHERKES, V.L.
2368*
CHESTERMAN, F.C.
2287*
CHEW, B.K.
2122*
CHIPYSHEVA, T.A.
1831
CHIU, J.-F.
1845
CHRETIEN, P.B.
2135, 2310*
CHU, E.H.Y.
1918*
CHURCH, A.
1898*
CITTADINI, A.
2272*
CLARK, H.F.
1953
CLEVELAND, P.
1937
CLIFFORD, P.
2006*
CLINE, M.J.
2134
COALSON, J.J.
2286*
COALSON, R.E.
2286*
CODINGTON, J.E.
2084
COFFIN, J.M.
1926
COHEN, I.R.
2052
COHEN, S.M.
1891
COHNEN, G.
2099
COLE, P.
2154
COLE, P.V.
1842
COLE, T.J.
1899*
COLLER, J.A.
2083
COLNAGHI, M.I.
2088
COLONNA, P.
2106
COLUMBO, J.P.
2023*
CONG, N.V.
2095
COOPER, N.R.
2101
CORNALBA, G.
2088
CORNUDELLA, L.
2301*
CORT, D.F.
2361*
COTTER, R.
2237*
COTTER, S.
1938
COULSON, D.B.
1824
COUPEY, S.
2323*
COURTNEY, R.J.
2027*
COWAN, K.
1970
CRADDOCK, C.
1845
CRADDOCK, C.G.
2380*
CRADDOCK, V.M.
1822
CRANDALL, B.F.
2380*
CRICHLOW, R.W.
2083
CROCE, C.M.
2026*
CROCKER, T.T.
2263*
CROMPTON, G.K.
2364*
CROSSEN, P.E.
2175
CRUMPACKER, C.S.
1956, 1973
CUMMINGS, F.J.
2110
CUMMINGS, K.B.
2062
CUZZI, J.
2217*
DALTON, A.J.
1947
DAMJANOV, I.
2281*
DAMMERT, K.
2229*
DANIEL, M.D.
2036*
DANIEL, V.
2347*
DANKO, I.M.
2330*
DARAI, G.
1959
DAUKIN, E.YA.
2344*
DAVIDSON, R.L.
1945, 2355*
DAVIS, H.J.
2003
DAVIS, K.J.
1864
DAWD, D.S.
2383*
DAWSON, R.
2260*
DE COUFLE, P.
2144
DE MICCO-PAGIS, C.
2120*
DE MICCO, PH.
2120*
DE PETRIS, S.
2094
DE RECONDO, A.-M.
2275*
DECHAMBRE, R.-P.
2238*
DECKERS, P.J.
2310*
DEHNER, L.P.
2187*, 2289*
DEINHARDT, F.
2064
DEKKER, A.
2282*
DELIDES, G.S.
2239*
DEMAREE, R.S., JR.
2292*
DEMOISE, C.
1835
DESAI, S.M.
2009*
DESHMUKH, N.
2398*
DIGGELMANN, H.
2305*
DIMITROV, M.
2327*
DIONISI, O.
2388*
DIXON, R.E.
2014*
DMOCHOWSKI, L.
1950, 1978, 2208*
DOCKERTY, M.B.
2277*
DODGE, W.H.
2035*
DOLD, U.
2309*
DOLPHIN, G.W.
1920*
DOMBERNOWSKY, P.
2157*
DONIACH, I.
2111*
DORE, J.F.
2095
DOUGLAS, S.D.
2099
DOWLING, M.D., JR.
2311*
DREYFUSS, Y.
2247*
DRITSAS, C.
2244*
DRUCKREY, H.
1892

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

DUGLE, D.L.
1900*
DUTSCHMAN, G.
2204*
DZHOKHADZE, D.I.
2185
EASON, R.
2033*
EAST, J.L.
1950
EASTY, D.M.
2212*
EATON, B.T.
2042*
EBEL, J.P.
2178
EBELS, E.J.
1869
EBNER, E.
2199*
ECKEL, H.
1878
EDELSTEIN, L.M.
1904*
EFSKIND, J.
2186
EHRENREICH, T.
2247*
EISENMAN, R.
2031*
EL BOULKANY, M.N.
2236*
EL-FIKY, S.M.
2383*
EL-LABBAN, N.G.
2215*
ELGJO, K.
1848
ELIZAN, T.S.
2038*
ELKIND, M.M.
1915
ELKINS, W.L.
2100
ELLIOTT, A.Y.
1937
EMANUEL, N.M.
2132
EMBLETON, M.J.
2070
EMMELOT, P.
1804, 1816
EMSON, H.E.
2256*
ENDES, P.
2241*
ENTERLINE, P.
2144
EPSTEIN, S.M.
1844
ERBE, J.
2267*
ERBING, B.
2273*
ERICKSON, R.J.
1963
ERTUERK, E.
1891
ESBER, H.J.
2279*
ESSEX, M.
1938
ESTERLY, J.A.
2260*
EVANS, C.A.
2177
EVANS, J.T.
1868
EVANS, R.
2266*
FADEYEV, N.P.
2376*
FAHRIG, R.
1889
FAIFERMAN, I.
2301*
FALCK, B.
2384*
FALCOFF, E.
2034*
FALK, L.
2064
FASSKE, E.
2004
FAULKNER, P.
2042*
FAUST, C.H., JR.
2305*
FELDMAN, D.G.
2247*
FELDMAN, S.P.
1972
FELDMANN, M.
2094
FEORINC, P.M.
2127*
FERON, V.J.
1816
FERRER, J.F.
1913
FERRERI, A.M.
1894
FERRONE, S.
2101
FETTING, R.
2004
FICHOT, O.
2275*
FIDLER, I.J.
2169
FIELD, E.J.
2058
FIGUS, I.A.
2276*
FILIPPOVA, N.A.
2328*
FINGERHUT, B.
2319*
FIORE-DONATI, L.
2115*
FIRSOVA, P.P.
2378*
FISCHER, R.
2334*
FISCHINGER, P.J.
1929
FISHER, B.
2188*
FISHER, E.R.
2188*
FITZGERALD, P.H.
2175
FLAD, H.-D.
2113*
FLANNERY, G.R.
2078
FLETCHER, O.J., JR.
2248*
FLETCHER, P.A.
2210*
FLICKINGER, R.A.
1944
FLIEDNER, E.
2296*
FLIPPEN, J.L.
1916
FONG, C.K.Y.
1989, 2000
FORROSTER, P.I.
1850
FORTNER, J.G.
2251*
FOX, B.W.
1914
FOX, M.
1914
FRABLE, W.J.
2390*
FRALEY, E.E.
1937
FRANKLIN, E.
1951
FRANKS, L.M.
2287*
FREDRICKSON, T.N.
2049
FREEMAN, A.E.
1835
FRENCH, J.H.
2213*
FRIBERG, S.
2103
FRIIS, R.R.
1957, 1958, 1994
FRITZ, W.
1820
FUJII, S.
2346*, 2365*
FUJIMAKI, M.
2233*
FUJIMOTO, M.
2225*
FUKUSHIMA,S.
1886
FURST, A.
1875
GABRIELIDES, C.G.
1840
GAL, F.
1879
GALEOTTI, T.
2170, 2388*
GALLWITZ, D.
2207*

GALZIGNA, L.
1812*
GANTT, R.
2025*
GARCIA, H.
2141
GARDNER, H.L.
2014*
GARG, S.K.
2389*
GARRETT, C.T.
1834
GARVICAN, L.
1847
GAZDAR, A.F.
2061
GEDDES, E.W.
2285*
GEL'FAND, V.I.
2076
GEL'SHTEYN, V.I.
1831
GELB, L.D.
2043*
GELFAND, I.M.
1966
GENDRON, M.C.
2071
GEORGIADES, J.
2208*
GEORGII, A.
2030*
GEROLD, F.P.
2255*
GERSHANOVICH, M.L.
2376*
GERY, I.
2092
GETZ, S.
1845
GHATAK, N.R.
2213*
GHOBRIAL, H.K.G.
1981
GHONEIM, M.A.
2236*
GHOSH, B.C.
2251*, 2255*
GHOSH, L.
2251*
GHOSH, P.C.
2313*
GIGNOUX, B.
2369*
GILDEN, R.
1935, 1984
GILDEN, R.V.
2069
GILEAD, Z.
1930
GILLESPIE, C.J.
1900*
GIMMY, J.
1892
GIORDANO, J.M.
2312*
GIRARDI, A.
2069
GLAZER, R.I.
1841
GLEW, R.H.
2046
GO, V.L.W.
2372*
GOBDEN, A.E.
2279*
GOGATE, S.A.
1906*
GOLDBLUM, N.
2124*
GOLDE, D.W.
2134
GOLDEN, G.S.
2213*
GOLDHABER, P.
2278*
GOLDMAN, R.L.
2243*
GOLDSTEIN, A.I.
1860
GOMARD, E.
2056
GOMEZ, J.
2354*
GONANO, F.
2171
GONZALEZ-MUJICA, F.
1833
GOODMAN, J.I.
2167
GORDON, R.J.
1835
GORKIN, V.Z.
2223*
GORYUKHINA, T.A.
2168
GOSSE, C.
2238*
GOTOHDA, E.
2066
GOTTLIEB, A.J.
2235*
GRAHAM, S.L.
1864
GRAHNE, B.
2158*
GRALNICK, H.R.
2386*
GRANOFF, A.
2016*
GRANTHAM, P.H.
1865
GRASSET, L.
2275*
GRAUER, R.C.
2224*
GREEN, M.
1964
GREENAWAY, P.J.
2013*
GREENBERG, J.
1905*
GREENBLATT, D.J.
2153
GREENGARD, O.
2304*
GREGORY, A.
2314*
GRIFFITHS, J.D.
2254*
GRILLI, S.
1894
GROSS, L.
2247*
GROSS, P.A.
1989, 2000
GROSS, R.
2334*
GROVER, P.
1801
GROVER, P.L.
1859
GRUBBS, C.J.
1825
GUELSTEIN, V.I.
1966
GUILLEBAUD, J.
2206*
GULLOTTA, F.
2296*
HABERKERN, R.C.
2181
HACKETT, A.J.
1925
HADJIOLOV, D.
1863
HAGGERTY, D.F.
2274*
HAGLID, K.G.
1871
HAHN, B.S.
1916
HAHN, M.J.
2260*
HAJDU, S.I.
2250*
HAKALA, T.
1937
HALES, R.L.
2208*
HALLERMAYER, E.
1866
HALPERN, M.S.
1958
HAMADA, K.
2037*
HAMANAKA, N.
1907*
HAMER, J.W.
2175
HAMILTON, J.L.
2224*
HAMLADJI, R.M.
2106
HAMMARSTROEM, S.
1971
HAMPAR, B.
2069
HANCOCK, R.L.
1850
HANSEN, W.H.
1864
HARA, M.
2048

HARDY, W.D., JR.
1938, 2065
HAREL, E.
1930
HARMS, E.
2270*
HAROZ, R.K.
1926
HARRAH, J.D.
2310*
HARRIS, C.C.
1870
HARRIS, J.E.
2318*
HASHIMOTO, K.
2231*
HASHIMOTO, S.
2193*
HATANAKA, M.
1935, 1984
HATFIELD, J.M.N.
1927
HAYAKAWA, K.
1907*
HAYESLIP, P.W.
2224*
HEADKA, M.
2021*
HEBERLEIN, G.T.
2366*
HEGNER, D.
2262*
HEIMANN, F.
2259*
HEINE, U.
1947
HELLERSTEIN, E.E.
2074
HELLRIEGEL, K.-P.
2334*
HELLSTROEM, T.
2158*
HELTING, T.
2273*
HENDERSON, V.
2144
HENNEHEUSER, H.-H.
2334*
HENRY, M.C.
1837
HENRY, P.H.
1956, 1973
HEPPNER, G.H.
2110
HERRMANN, J.B.
2363*
HERRON, R.E.
2217*
HERZOG, L.
2030*
HESS, F.W.
1938
HEWER, A.
1859
HIAI, H.
2045
HIBASAMI, H.
2200*, 2201*
HILFRICH, J.
1878
HILGARD, P.
2397*
HINKES, E.
2380*
HINZE, H.C.
2017*
HIRAGUN, A.
1817
HIRANO, A.
2213*, 2298*
HIRSCH, M.S.
2079
HIRT, B.
1805
HJERN, B.
2259*
HNILICA, L.S.
1845
HO, W.
1875
HOAK, J.C.
2054
HOEPPING, W.
2128*
HOFFMAN, G.C.
1877
HOHAGE, R.
2397*
HOLLIS, D.P.
2385*
HOLTERMANN, O.A.
2126*
HOLTERMULLER, K.H.
2372*
HOLUBAR, K.
2231*
HOOVER, R.
2154
HORIE, M.
2137
HORN, L.
1818
HORNEERGER, E.
2069
HORTON, B.J.
2130
HORVATH, E.
2353*
HOULDITCH, G.S.
1928
HOWELL, J.B.
2231*
HOYE, R.C.
2310*
HRGOVCIC, M.
2317*
HSIA, J.C.
2108
HSIEH, D.P.H.
1854
HSIEH, P.
2163
HSIUNG, G.D.
1989, 2000
HSOSKAWA, M.
2066
HSU, K.C.
2069
HUEBNER, G.
2334*
HUEBNER, R.J.
1835
HUEBNER, V.
1984
HUGHES, D.
2058
HUNG, P.
2012*
HUNG, P.P.
1931
HUNTER, E.
1957
HURCH, A.
1904*
HUSEBY, R.A.
1880
HUTTON, C.E.
2331*
HUTTON, W.E.
2293*
HUVOS, A.G.
2249*, 2251*, 2255*
HYDE, E.H.
2356*
HYMAN, G.
2319*
IKARASHI, K.
2233*
IKEDA, H.
2059
ILFELD, D
2052
ILYIN, K.V.
1952
IMAMURA, N.
2008*
IMASHUKU, S.
2391*
IMURA, H.
2365*
INBAR, M.
1941, 2165
INUI, N.
1901*
IOKI, Y.
1885
IPEMA, A.
1869
IRUNBERRY, J.
2106
ISHIDA, R.
1993
ISHIKAWA, K.
2379*
ISHIMARU, T.
1917*
ISHITANI, K.
1881
ISLIKER, H.
2117*
ITO, M.
2200*
ITO, N.
1884

ITO, Y. 2044
IVANOV-GOLITSYN, M.N. 1858
IVANOVA, O.YU. 1966
IVANYI, L. 2051
JACKMAN, D.A. 2036*
JACKSON, F. 1870
JACOBS, S. 2059
JACOBSSON, S. 2384*
JAKOWSKI, R.M. 2049
JANIK, B. 1963
JEANLOZ, R.W. 2084
JELLINGER, K. 2297*
JENKINS, D.E. 2280*
JENKINS, R. 1951
JEPSON, P.N. 2240*
JHUNG, J.W. 1904*
JOHNSON, B.F. 1905*
JOHNSON, G.S. 1932
JOHNSON, K.H. 1981
JOHNSON, W.W. 2320*
JOKINEN, K. 2229*
JONEJA, M.G. 1824
JONES, D.B. 2080
JONES, J.S.P. 2070
JONES, S.E. 2091
JONSSON, N. 2072
JOSEPH, D.J. 2260*
JOSEY, W.E. 1951
JOUPPILA, F. 1815
JOVEV, G. 2327*
JULIAN, B. 2025*
JULIAN, C.G. 2003
JULL, F.W. 2323*
KABIYEV, O.K. 2340*
KABULOV, M.K. 2341*
KADOHAMA, N. 2180
KAERKI, N.T. 1815
KAHN, L.B. 2283*, 2284*, 2291*
KAISER, W. 2198*
KAKU, M. 2346*
KALCHERT, S.L. 1877
KALUS, M. 2280*
KAMMAMOTO, Y. 1884
KANG, C.-Y. 1942
KANG, K.-Y. 2143
KANO-SUEOKA, T. 2163
KANO, T. 1882
KAPLAN, A.S. 1809
KAPLAN, E.L. 2372*
KAPLAN, H.S. 1913
KARASAKI, S. 2131
KARBY, S. 1930
KARLE, I L. 1916
KARNOVSKY, M.J. 1962
KASPER, C.B. 2268*
KATO, K. 1975
KATZ, C. 1834
KAUFMAN, D.G. 1837, 1870
KAUFMAN, J.J. 2062
KAUFMAN, R. 2217*
KAUFMAN, R.H. 2014*
KAWASE, M. 2200*
KAY, S. 2390*
KAYMAN, S.C. 2046
KEARNEY, E. 2314*
KEEFER, L. 1895
KEKO-PINTO, C. 1890
KELLEN, J.A. 2067
KELLER, A.Z. 2148
KELLERMANN, G. 1829
KELLEY, P.R. 2190*
KELLOFF, G. 2069
KELLY, F. 2010*
KENNY, A.D. 2293*
KENYON, D.H. 1807
KERR, J.F.R. 2220*
KERSEY, J.H. 1948
KETCHAM, A.S. 2135, 2310*
KEYDAR, J. 1930
KHAN, M.H. 2349*
KHUZHAMBERDIEV, M. 2223*
KIMURA, I. 2022*
KIMURA, N. 2346*
KINDER, D.L. 1856
KING, N.W. 2036*
KIRCHHEIM, D. 1853
KIRCHNER, C. 2096
KIRKPATRICK, D. 2092
KITAGAWA, M. 2048
KITAGO, M. 1881
KLEIN, E. 2126*
KLEIN, G. 2064, 2103
KLEIN, H.A. 2243*
KLEIN, R. 1935
KLIEM, U. 2117*
KLIETMAN, W. 1975
KLING, T.G. 2184
KLOPP, C.T. 2312*
KLOSTERHALFEN, H. 2136
KMOCHOWSKI, L. 2228*
KNESEK, J.E. 1950
KNIPPERS, R. 2041*

KNUFERMANN, H.
2162
KOBAYASHI, H.
2066, 2090
KODAMA, M.
1885
KODAMA, T.
2066, 2090, 2104
KOEHLE, W.
2397*
KOENIG, E.
2099
KOGAN, I.YA.
2002
KOHNE, D.E.
2182
KOIDE, K.
2299*
KOJIMA, K.
2346*
KOLESNICHENKO, T.S.
1838, 1908*
KOLKAILA, A.M.
2383*
KOLPASHCHIKOVA, I.F.
2227*
KOMOV, V.P.
2344*
KONDRAT'YEVA, A.F.
1823
KONDRATICK, J.M.
1947
KOPROWSKI, H.
2026*
KOROSTELEVA, T.A.
1823
KOROTKORUCHKO, V.F.
2330*
KOSAKI, T.
2200*, 2201*
KOSKY, K.
2183
KOSS, L.G.
2250*
KOTHARI, R.M.
2012*
KOTCH, K.
1880
KOVACS, K.
2353*
KOWALEWSKI, K.
1897*
KOYAMA, H.
1999
KRAIN, L.S.
2145
KRAM, R.
2176
KRAMER, I.R.H.
2215*
KRAUSE, E.
2041*
KRAUSE, F.
2309*
KRAUSE, J.R.
2282*
KREISEL, W.
2270*
KROSCH, H.
2198*
KRSMANOVIC, V.
1995
KRUEGER, R.G.
2183
KRYUKOVA, I.N.
2105
KUBO, T.
2152
KUHLENSCHMIDT, M.S.
2046
KUMAR, S.
2057, 2218*
KUNII, A.
2044
KUZUMAKI, N.
2066, 2090, 2104
LA CARBIERE, M.
1946
LABITZKE, H.G.
2240*
LAFLEUR, L.
2093
LAISHES, B.A.
1876
LAMBREMONT, E.N.
2194*
LANDSHUETZ, C.
1892
LANE, I.
1934
LANG, W.
2030*
LANGMAN, M.J.S.
2070
LARSEN, T.E.
1848
LASZLO, J.
2139
LAU, P.
2235*
LAUSCH, R.N.
1939
LAWSON, T.A.
1818
LE BORGNE DE KAOUEL, C.
2095
LE GUERRIER, J.M.
2068
LE VINE, D.
2013*
LEAVITT, P.
1934
LEBEDEVA, E.A.
2375*
LEBEDEVA, S.B.
2329*
LEE, J.
1874
LEE, K.W.
2215*
LEE, S.L.
2360*
LEENHOUTS, H.P.
1910
LEES, B.J.
1852
LEHMAN, J.
1992
LEHNER, T.
2051
LELIEVRE, L.
2216*
LEMBO, R.
1832
LEPESANT, J.-A.
2275*
LEUCHTENBERGER, C.
2117*
LEUNG, B.S.
2166
LEVIN, M.J.
1954, 1956
LEVINE, A.J.
1808
LEVINE, A.S.
1954, 1956, 1958, 1973
LEVINE, L.
2278*
LEVY, J.P.
2056
LEVY, M.
2153
LEWIS, A.
2206*
LEWIS, A.M., JR.
1954, 1956
LEWIS, A.M., JR
1973
LEZIUS, A.
2242*
LIAO, S.K.
2047
LICHTMAN, M.A.
2314*
LIEBACH, J.
1909*
LIEBELT, R.A.
2217*
LIEBERMAN, M.
1913
LIEBOW, A.A.
2252*
LIJINSKY, W.
1895
LIKHACHEV, A.IA.
1858
LILLICRAP, S.C.
2269*
LILLY, F.
2059
LINDAHL, T.
2345*
LINELL, F.
2394*
LING, V.
2203*
LINIAL, M.
2018*
LINK, K.
2197*
LINSELL, C.A.
2147
LIPPINCOTT, J.A.
2366*

LISAFELD, E.A.
2126*
LITTLE, J.R.
2108
LITVINOV, V.I.
2377*
LITWACK, G.
2347*
LIU, H.-Y.
2022*
LIU, P.I.
1917*
LJUNGGREN, J.-G.
2259*
LLOYD, D.C.
1920*
LLOYD, N.
1846
LO, A.C.-H.
2067
LO GERFO, J.J.
2068
LOCKER, H.
2005
LOCKWOOD, E.
2313*
LOEB, L.A.
1983
LOEB, W.F.
1943
LOEWHAGEN, T.
2259*
LOG, T.
2061
LONG, C.
1984
LONG, J.C.
2087
LOO, J.
1895
LOONEY, W.B.
2191*
LOSSIO, B.E.
2325*
LOVE, R.
2161
LOVELACE, E.
1976
LOWENTHAL, A.
2023*
LOWER, G.M., JR.
1891
LOWY, R.
1890
LOZZIO, C.B.
2325*
LUGINBUHL, R.E.
2049
LUPATIN-TRIDENTE, E.
2115*
LUTMAN, G.
1868
MAC DONALD, J.
2129*
MAC PHERSON, I.
1955
MACA, R.D.
2054
MACH, B.
2305*
MACHOVICH, R.
2304*
MAEKELAE, V.
2230*
MAEKINEN, J.
2230*
MAENOSONO, J.
1882
MAGEE, F.N.
1888
MAGNIER, M.
1827
MAKIURA, S.
1884
MALAMA, A.A.
2339*
MALDONADO, R.L.
1961
MALMQUIST, J.
2358*, 2359*
MAMONT, P.
2176
MANAUGH, L.C.
2166
MANCHON, PH.
1890
MANDAL, B.
2218*
MANOYLOV, S.YE.
2336*
MANSOUR, M.A.
2236*
MARCANTE, M.L.
1933
MARGALITH, E.
2124*
MARGALITH, M.
2124*
MARGOLIS, C.I.
2292*
MARGOLIS, L.B.
1966
MARHOLEV, L.
2095
MARK, J.
2205*
MARKOE, A.M.
1990
MARKOV, D.
1863
MARTIN, W.J.
2129*
MARTINEZ, R.
2332*
MARTINK M.A.
2043*
MARTUZZI, M.
2140
MARUYAMA, K.
2228*
MASON, T.E.
2292*
MATANI, A.
2244*
MATAYOSHI, K.
1884
MATEVOSOV, A.L.
2375*
MATHEWS, M.B.
2226*
MATHIAS, A.P.
1833
MATSUDAIRA, Y.
2045
MATSUOKA, M.
2396*
MATSUOKA, Y.
2048
MATTILA, S.
2230*
MAURICE, A.
1867
MAYER, V.W.
1883
MAYEVSKIY, A.A.
1839
MAYOR, H.D.
1928
MAYUZUMI, T.
2396*
MC CAFFREY, R.
2307*
MC CARTER, J.A.
2020*
MC CLELLAN, D
2323*
MC CULLOUGH, L.B.
2161
MC GRAW, J.L.
2400*
MC GREGOR, D.H.
1917*
MC GUIRE, W.L.
2271*
MC KAY, D.E.
2294*
MC KINNA, J.A.
2254*
MC ROBERTS, J.W.
1853
MECHALI, M.
2034*
MEEKER, B.E.
1900*
MELENDEZ, L.M.
2036*
MELLORS, R.C.
2065
MELNICK, J.L.
2149
MENARD, S.
2088
MENNE, H.-D.
2113*
MERCIER, J.
2178
MERKOW, L.P.
1844, 2224*
METCALF, D.
1803
MEYER, G.
2107, 2120*
MEYER, J.S.
2293*

MICHALSKI, F.
1953
MICHIE, W.
2350*
MIDDLETON, V.L.
2177
MILAS, L.
2211*
MILLER, A.A.
2352*
MILLER, R.G.
2093
MILLER, T.R.
2255*
MILLMAN, I.
1983
MILLS, J.
1846
MILSTEIN, J.B.
2043*
MILTENYL, L.
2241*
MIRSON, I.M.
1839
MISHENEVA, V.S.
2168
MITCHELL, M.S.
2092
MITTELMAN, A.
1868
MIWA, M.
1901*
MIYAKI, M.
2299*
MODICA, M.M.
2068
MOEHLER, K.
1866
MOERTEL, C.G.
2277*
MOFFATT, B.
2395*
MOHR, J.A.
2286*
MOHR, U.
1836, 1878
MOK, G.
2115*
MOKYR, M.B.
2092
MOLLA, A.
2393*
MOLNAR, J.
2103
MOLOCHKINA, YE.M.
2160
MONACO, A.P.
2079
MONTAGNIER, L.
1985
MONTESANO, R.
1802
MOON, R.C.
1825
MOORE, L.A.
2247*
MOORE, M.
2080
MOORE, R.E.
1834
MORAN, S.
1934
MORELL, A.
2081
MORRIS, H.P.
2170, 2172, 2191*, 2270*,
2362*, 2385*
MORRIS-JONES, P.
2057
MORTON, J.I.
1977
MOSCOVICI, C.
2035*
MOTOMURA, S.
2137
MOULE, Y.
1819
MOUTET, J.-P.
1827
MUELLER-LORSEN, B.
2242*
MUKHERJEE, A.K.
2164
MUKHERJEE, T.M.
2370*
MULKEY, K.
2217*
MULLER, H.K.
2078
MUMFORD, D.M.
1928
MUNDY, G.R.
2189*
MUNK, K.
1959
MUNRO, A.H.G.
2364*
MURAD, F.
2191*
MURAD, T.M.
1909*
MURAMATSU, M.
2193*
MURPHY, W.M.
2258*
MUSHINSKI, E.B.
2098
MYERS, D.E.
1980
MYHR, B.C.
1857
NAGASAWA, H.
2159, 2225*
NAGATA, C.
1885
NAHMIAS, A.J.
1951
NAIB, Z.M.
1951
NAKAGAWA, S.
2200*
NAKAJIMA, K.
2037*
NAKAMURA, M.
2121*
NAKAMURA, T.
2391*
NAKANE, P.K.
2321*
NANAVATI, A.N.D.
2009*
NAPIAS, C.
1867
NARVAEZ, R.
2068
NASONOV, A.P.
2245*
NATHANS, D.
2019*
NATORI, H.
1881
NAZERIAN, K.
1806, 2125*
NELSON, J.S.R.
2382*
NESTEROVA, L.A.
2336*
NEWTON, W.A.
2208*, 2228*
NEYFAKH, E.A.
2223*
NG, A.B.P.
2258*
NICOLSON, G.L.
1946
NIITSU, Y.
1881
NISHIMOTO, Y.
1817
NISHIZUKA, Y.
2045
NISONOFF, A.
2098
NISSEN, E.D.
1860
NITSCHKE, U.
2197*
NOBLE, R.L.
2324*
NOGAMI, T.
1911
NOMURA, T.
1830
NOON, M.C.
2055
NORDQUIST, R.E.
2085, 2286*
NORTHUP, S.J.
2362*
NORVELL, J.
1984
NUNES, L.L.
2263*
O'CONNELL, A.
1983
O'DONNELL, T.V.
2263*
O'TOOLE, C.
2073
OBE, G.
1889
OBOSHI, S.
1885
OBUKH, I.B.

2105
ODEKU, E.L.
2007*
OGI, K.
2137
OKAGAKI, T.
2253*
OKANO, T.
1882
OKUDA, H.
2365*
OKUDAIRA, Y.
1907*
OKUNEWICK, J.P.
1990
OLD, L.J.
1938, 2059
OLINICI, C.D.
2348*
OLSNES, S.
2186
ONG, Y.W.
2122*
ONO, T.
2299*
ONODA, I.
1882
OPPENHEIMER, S.B.
2387*
OROSZLAN, S.
2069
ORSELLI, R.C.
2315*
ORY, H.W.
2153
OSATO, T.
2060
OSIPOV, N.YE.
2133
OSUNTOKUN, B.O.
2007*
OTH, D.
1821
OTSU, H.
1902*
OXMAN, M.N.
1945, 1954
PALMER, E.L.
2127*
PALMER, P.D.
2084
PAPAGEORGE, A.G.
1929
PAPELISHVILI, R.K.
2185
PARAF, A.
2216*
PARDEE, A.B.
2179
PARDO, M.
1844
PARISE, F.
2360*
PARK, S.A.
2221*
PARKE, D.V.
1846
PARKIN, R.
1888
PARKS, W.P.
1969, 2055
PARMIANI, G.
1832
PARSONS, J.T.
1926
PARTENSKY, C.
1802
PASTAN, I.
1932, 1976
PATEL, S.A.
2006*
PATEL, V.
2381*
PATNAIK, R.
2174
PATRICOT, L.M.
2369*
PAUL, D.
2011*
PAWLAK, L.L.
2098
PAYNE, I.J.
2279*
PEARSON, G.
2064
PEARSON, G.R.
1988
PEARSON, J.W.
1988
PECHET, L.
1898*
PEEBLES, P.T.
1929
PEERS, F.G.
2147
PEGG, C.A.S.
2350*
PEGRUM, G.D.
2177
PELKONEN, O.
1815
PELLEGRINO, M.A.
2101
PENNINGTON, G.W.
2313*
PEREZ-BERCOFF, R.
2034*
PERLMANN, P.
2073
PERRIN, J.
2111*
PERRY, C.H.
1864
PETER, J.B.
2062
PETERSEN, H.S.
2333*
PEVNITSKY, L.A.
2123*
PEVZNER, YU.D.
2338*
PHILLIPS, B.
2116*
PHILLIPS, G.
2370*
PHILLIPS, R.A.
2093
PICARD, J.Y.
2095
PICKREN, J.W.
2237*
PIERCE, G.B.
2321*
PIHL, A.
2186
PINSKIY, S.B.
2342*
PINTO, R.E.
1887
PIRRO, G.
2171
PITOT, H.C.
1834, 2202*
PLAGEMANN, P.G.W.
2267*
PLATA, F.
2056
PLATT, D.
2262*
POGO, A.O.
2301*
POLGAR, P.
2190*
POLIVODA, B.I.
2245*
POLIVODA, O.M.
2245*
POLYZONIS, M.
1840
PONKA, J.L.
2354*
PONOMAR'KOV, V.I.
2133
POPJAK, G.
2274*
POPP, F.A.
1872
PORT, C.D.
1837
PORZSOLT, F.
2096
POTHIER, L.
2074
POTTER, M.
2098
POTTER, V.R.
2167
POUND, A.W.
1818
POUR, P.
1836
POZHARISSKII, K.M.
2142
PRAETORIUS-CLAUSEN, F.
2146
PRASAD, K.N.
2218*
PRICE, R.A.
2320*
PRIESTER, W.A.
2288*
PRIORI, E.S.
2208*
PRODI, G.

1894
PRYSE, K.
2108
PUDOV, V.I.
2227*
PURROTT, R.J.
1920*
PUTNEY, F.J.
2399*
RABIN, H.
2064
RABKIN, I.KH.
2375*
RABSON, A.
2064
RABSON, A.S.
2310*
RADCMSKI, J.L.
1855
RAESAENEN, O.
2229*
RAFF, M.C.
2094
RAHMAN, F.
2280*
RAKHMANINA, T.F.
2344*
RAMSDEN, F.
2352*
RAO, K.V.N.
1849
RAPAPORT, F.T.
2114*
RAPP, F.
1998
RAVEN, R.W.
1846
RAWLA, W.E.
2149
RAWLS, W.E.
2014*
REBOURCET, R.
2095
REDMON, L.W.
1988
REES, K.R.
1847
REGAN, F.D.
1843
REIN, A.
1932
REISFELD, R.A.
2101
RENGER, H.C.
1967
RELTTER, W.
2270*
REY, A.A.
1855
REZNIK, G.
1878
REZNIK-SCHUELLER, H.
1878
RHIM, J.S.
1835
RHOADES, E.R.
2286*
RICHARDT, R.M.
2253*
RIMAN, J.
2032*
ROBBINS, G.F.
2249*
ROBERT, F.
1821
ROBERTSON, H.D.
2226*
ROCCHI, P.
1894
ROCHE, F.M.
1944
RODRIGUEZ, L.M.
1944
ROED-PETERSEN, J.
2089
ROED-PETERSON, B.
2089
ROGERS, S.
2023*
ROMAN, C.S.
1923*
ROMANOVA, L.A.
2223*
ROMARY, F.
1827
RORSMAN, H.
2384*
ROSENBERG, J.
1939
ROSENBLITH, J.Z.
1962
ROSENGREN, A.-M.
2384*
ROSENGREN, E.
2384*
ROSNER, F.
2360*
ROSS, A.E.
1895
ROSS, R.C.
2353*
ROSSIGNOL, J.-M.
2275*
ROWBOTHAM, H.D.
2254*
ROWE, W.P.
2059
ROWLATT, C.
2287*
RUBEL, L.R.
2351*
RUCKER, E.
1958
RUEHLANC, D.
2004
RUSS, L.J.
2020*
RUTENBURG, A.M.
2190*
RYABUKHA, A.K.
2336*
SABINE, J.R.
2130
SABOLOVIC, D.
1821
SACHS, L.
1941, 2165
SACHS, R.
1984
SACK, G.H., JR.
2019*
SAFFER, E.
2188*
SAFFIOTTI, U.
1870
SAHAI SRIVASTAVA, B.J.
2192*
SAIER, E.L.
2224*
SAINT-FRANCCIS, B.
1814
SAITO, K.
2233*
SAKA, T.
2200*
SAKSELA, E.
2119*, 2158*
SALSBURY, A.J.
2254*
SALZMAN, N P.
1960
SAMAHA, R.J.
1956
SAMBROOK, J.
2010*
SAMSONOV, V.S.
2377*
SAMUELS, L.T.
1880
SAMUELSSON, B.
1971
SANFORD, B.H.
2084
SANZENBACHER, L.J.
2372*
SARASIN, A.
1819
SARKAR, K.
2294*
SARMA, P.S.
2061
SARYAN, L.A.
2385*
SASAKI, T.
1911
SATO, H.
1886, 2059
SATO, N.
1975
SATO, Y.
1907*
SATTAR, P.
2318*
SAWADA, T.
2391*
SAWAI, Y.
2121*
SCARPA, A.
2272*
SCHAEFER, H.-E.
2334*
SCHAFFER, F.L.
1925
SCHAFFER, P.A.

2028*
SCHATZKI, F.
2390*
SCHENGRUND, C.L.
1939
SCHIFFER, L.M.
2382*
SCHILDBERG, P.
2128*
SCHLECHTE, H.
2021*
SCHLOM, J.
1972, 1991
SCHMIDT, G.A.
2198*
SCHMITT, W.
2397*
SCHNEIDER, V.
2309*
SCHNIPPER, L.E.
1973
SCHOLAR, E.M.
2264*
SCHOUB, L.
2283*
SCHUMACHER, H.R.
2221*
SCHUMM, D.E.
2172
SCHWARTZ, J.
2038*
SCOLNICK, E.M.
1969, 2055
SEAH, C.S.
2122*
SEAL, S.H.
2311*
SEHDEV, M.K.
2311*
SEHGAL, P.E.
2009*
SEIDEL, H.J.
2029*
SELF, D.J.
1986
SELIGMAN, B.R.
2360*
SELLYEI, M.
2209*
SEMAN, G.
1978
SEN, S.
1861
SENDO, F.
2066, 2090
SENEWIRATNE, B.
2150
SENIK, A.
2056
SERA, Y.
2143
SERALE, J.
2220*
SEVER, J.L.
2015*
SHALUMOVICH, V.N.
2142
SHAMBERGER, R.J.
1877
SHANMUGAN, G.
1964
SHAPOSHNIKOV, YA.D.
2142
SHARMA, R.K.
2302*
SHAW, C.R.
1829
SHCHERBOKOV, V.YA.
2075
SHELLABARGER, C.J.
1826
SHENDRIKOVA, I.A.
1858, 1893
SHENK, T.E.
2040*
SHIBATA, T.
2044
SHIDA, K.
2396*
SHILLITOE, E.J.
2051
SHIMAZAKI, J.
2396*
SHIRES, T.K.
2202*
SHIRODKAR, M.V.
2009*
SHISA, H.
2045
SHLYAKHTER, N.A.
2343*
SHMONINA, V.M.
2132
SHUBIK, P.
2141
SIAKOTOS, A.N.
2381*
SIEGEL, B.V.
1977
SIEGISMUND, G.
2030*
SIEGLER, R.
1934
SILBER, R.
2389*
SILVETTI, S.
2171
SIMON, L.
2276*
SIMS, P.
1859
SINGH, S.
2006*
SIZEMORE, G.W.
2372*
SJOGREN, H.O.
2053
SKACHKOV, A.P.
1823
SKVARIL, F.
2081
SLIFKIN, M.
1844
SMAHA, R.J.
1973
SMITH, A.E.
1807
SMITH, G.H.
2025*
SMITH, J.M.
1870
SMITH, R.E.
1936
SMOLER, D.F.
2307*
SNYDER, L.M.
1898*, 1904*
SOEDERHAELL, S.
2345*
SOGA, J.
2233*
SOLTER, D.
2281*
SOMFAI, S.
1879
SOMMER, R.G.
1963
SOO, V.A.
1826
SORIANO, R.Z.
2161
SOROKINA, YU.D.
1851
SOTNIKOY, V.N.
2377*
SPAEREN, U.
2186
SPARKES, R.S.
2357*
SPIEGELMAN, S.
1972, 1991
SPIRO, R.H.
2250*
SPORN, M.B.
1870
SRODES, C.H.
2356*
ST. GEME, J.W., JR.
2039*
ST. JEOR, S.
1998
STAVROU, D.
1871
STEARNS, M.W., JR.
2311*
STEER, A.
1917*
STEIN, G.S.
2173
STEIN, N.
1937
STEINER, M.R.
2371*
STEINER, S.
2371*
STEINER, S.M.
2027*
STENBACK, F.
2141
STEPHENSON, J.R.
1968
STEVENS, L.C.
2265*
STEWARD, J.K.

2057
STEWART, D.J.
2350*
STEWART, T.H.M.
2318*
STICH, H.F.
1876
STOCKERT, E.
2059
STOLBACH, L.
2110
STOLLAR, V.
2040*
STONEHILL, E.H.
2086
STONINGTON, O.G.
1992
STRAETLING, W.
2041*
STRAND, M.
1940
STREETEN, E.W.
2400*
STRONG, E.W.
2250*
STUPFEL, M.
1827
SUGARBAKER, E.V.
2310*
SUGAWARA, K.
2060
SUGIHARA, S.
1884
SUGIMURA, T.
1901*
SUKHORUKOV, B.I.
1839
SULLIVAN, K.A.
2257*
SUTNICK, A.I.
1983
SUZUKI, I.
1993
SYLVESTRE, D.
2095
SZEKELY, I.E.
2221*
SZENTIRMAY, Z.
1879
SZILAGYI, T.
2241*
SZOLLOSI, D.
1965
SZUECS, P.
2241*
TABARES, E.
1987
TAGASHIRA, Y.
1885
TAKADA, H.
2391*
TAKAHASHI, M.
1886
TAKANASHI, R.
2232*
TAKATSU, K.
2048
TAKAYAMA, S.
1901*
TAKEDA, K.
2365*
TAKEDA, M.
2161
TAKEICHI, N.
2066, 2090, 2104
TAN, L.
2122*
TANAKA, M.
2044, 2200*
TASHJIAN, A.H., JR.
2278*
TATRA, G.
2050
TAYLOR, G.
2057
TAYLOR, M.
2012*
TAYLOR, M.W.
2174
TAYLOR, P.T.
2310*
TAZAWA, K.
2233*
TCHANG, F.
2253*
TEGTMEYER, P.
1970
TEMIN, H.M.
1942
TEPPO, L.
1811*
TERAO, K.
1902*
TERAYAMA, H.
1828
TERHEGGEN, H.G.
2023*
TERRANOVA, T.
2388*
TESSMER, C.F.
2317*
THEMANN, H.
2004
THOMAS, D.B.
2116*, 2156
THOMAS, E.W.
2191*
THOMPSON, L.H.
2203*
THOREN, M.M.
1960
THORINGTON, R.W.
2036*
THORNE, R.F.W.
2300*
THRALL, C.L.
2173
THUNOLD, S.
2109
TILL, J.E.
2203*
TODARO, G.J.
1948, 1982
TOKUMA, Y.
1828
TOLEDANO, A.
2332*
TOLNAI, G.
2294*
TOLSTYKH, P.I.
2378*
TOMATIS, L.
1802
TOMICH, C.E.
2331*
TOMIYASU, U.
2298*
TOMKINS, G.M.
2176, 2347*
TOMURA, T.
2322*
TONDER, O.
2109
TONI, R.
1935
TORPIER, G.
1985
TRAININ, N.
2052
TRAN, M.-H.
1827
TRAN MINH, V.
2369*
TRAVNICEK, M.
2032*
TRIDENTE, G.
2115*
TROSKI, J.E.
1918*
TROUPP, H.
2119*
TRUJILLO, J.M.
2337*
TSOCHIDA, N.
1964
TSOLAKIDIS, P.
2254*
TURKINGTON, R.W.
2180
TWEEDELL, K.S.
1953
UEDA, G.
1907*
UFREZAL, J.
2095
UKENA, T.E.
1962
ULLAND, B.M.
1862
ULLRICH, V.
1896*
UNSGAARD, B.
2073
UNTERHARNSCHEIDT, F.J.
2097
URAGODA, C.G.
2150
URBAN, J.A.
2249*
URUSHIZAKI, I.
1881
VAAGE, J.
2063
VALERIO, M.G.

1943
VALLADARES, Y.
1987
VAN DAMME, B.
2393*
VAN LANCKER, J.L.
2322*
VAN ROSSUM, G.D.V.
2170
VARGHA, GY.
2241*
VASILIEV, JU.M.
1966
VASS, L.
2209*
VECCHIO, G.
1964
VELOURS, J.
1867
VERA, J.C.
2190*
VERHAEGHE, R.
2393*
VERMYLEN, J.
2393*
VESCIA, A.
2374*
VESSELINOVITCH, S.D.
1849
VIADANA, E.
2237*
VIEVILLE, C.
2369*
VILENCHIK, M.M.
1839
VINTERGAL'TER, S.F.
2367*
VLODAVSKY, I.
1941
VOELKEL, E.F.
2278*
VOGT, V.M.
2031*
VOIGT, K.D.
2136
VON HAAM, E.
1906*, 1909*
VORONOVA, L.A.
2329*
VOYTOVITSKIY, V.K.
2340*
WADA, K.
2233*
WADE, E.
1958
WAGHE, M.A.
2057
WAGLE, D.G.
1874, 2234*
WAGNER, J.E.
1856
WAHAB, R.N.A.
2383*
WAHLSTROEM, T.
2119*
WALINDER, G.
1921*, 1922*
WALKER, J.
1935
WALKER, F.M.B.
1927
WALLACH, D.F.H.
2162
WANG, D.Y.
2206*
WANG, S.
2012*
WANG, S.Y.
1916
WARD, J.F.
1919*
WASON, W.M.
2082
WATKINS, E., JR.
2219*
WATKINS, S.M.
2112*
WATSON, C.J.
2055
WAYNFORTH, H.B.
1888
WEBB, T.E.
2172
WEBBER, M.M.
1992
WEBER, F.
2380*
WEBER, M.J.
1979
WECHSLER, W.
1986
WEGNER, D.L.
2017*
WEIL, D.
2095
WEISBURGER, E.K.
1862, 1865
WEISBURGER, J.H.
1862, 1865
WEISS, M.C.
2357*
WEISS, W.
1873, 2151
WEISSMAN, S.M.
1995
WEISSMANN, C.
1926
WELCH, M.F.
2194*
WEXLER, H.
2135
WHITE, M.
2033*
WICKRAMASINGHE, S.N.
2395*
WIGZELL, H.
2073
WILBIN, C.N.
1955
WILBUR, J.
2317*
WILFONG, R.F.
1986
WILKEY, I.S.
2351*
WILKINS, R.J.
1843
WILLIAMS, D.C.
1846
WILLIAMS, R.C.
1983
WILLIAMS, W.J.
2235*
WILLIS, C.E.
1877
WILSON, D.A.
1974
WILSON, P.D.
2196*
WILTON, J.M.A.
2051
WITSCHI, H.P.
1814
WOLF, E.
2050
WOLFE, L.
2064
WOLFORD, R.G.
1835
WONG, L.T.L.
2108
WONG, P.K.Y.
2020*
WOOD, D.C.
2166
WOOD, M.L.
2079
WRIGHT, J.A.
2203*
WRIGHT, N.A.
2373*
WU, P.C.
2179
WUNDERLICH, F.
2162
WUNDERLICH, J.R.
2129*
WYKE, J.A.
2018*, 2024*
WYLIE, C.V.
2321*
WYLIE, J.H.
2354*
YABE, Y.
1999
YAMAGUCHI, N.
1993
YAMAMOTO, R.S.
1862
YAMAMOTO, T.
1917*
YAMANAKA, H.
2396*
YAMANISHI, Y.
2231*
YANAI, R.
2159
YAROSHEVA, A.A.
2326*
YEDYGENOVA, A.K.
2340*
YEMEL'UANOV, S.S.
2377*
YERMOLOV, A.S.

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

2377*
YIN, H.H.
1962
YIN, L.K.
2083
YOHN, D.
1953
YOKOTA, M.
1881
YOKOYAMA, K.
2143
YOSHIKI, T.
2065
YOSHIKURA, H.
2118*
YOSHINARE, S.
1907*
YOSHIOKA, Y.
1884
YOUNG, P.L.
2274*
YOUNG, R.L.
2014*
YUNG, L.
2115*
YUNIS, E.J.
1948
YUSHKANTSEVA, S.I.
2376*
ZACH, J.
2334*
ZAMECNIK, M.V.
2087
ZAMECNIK, P.C.
2087
ZAPOROZHCHENKO, I.G.
1924*
ZEIGEL, R.F.
1953
ZETTERLUND, C.G.
2073
ZEYA, H.I.
2139
ZHDANOV, V.M.
1952
ZIMMERMAN, H.M.
2298*
ZOBL, H.
2030*
ZUS', B.A.
2343*

- ACANTHOMA
  - CLEAR CELL
    - CASE REPORT, 2379*

- N-ACETYLLACTOSAMINE SYNTHETASE
  - MASTOCYTOMA
    - MOUSE, 2273*

- N-ACETYLNEURAMINIC ACID
  - SYNTHESIS IN MORRIS HEPATOMA
    - RAT, 2270*

- ACTINOMYCIN D
  - POLYOMA VIRUS-INDUCED SURFACE ANTIGEN
    - HAMSTER CELLS, 2107
  - VIRUS, SV40
    - ANTIGEN INDUCTION, 2124*

- ADENOCARCINOMA
  - BARTHOLIN'S GLAND
    - CASE REPORT, 2253*
  - CILIARY PIGMENT EPITHELIUM TUMOR
    - ULTRASTRUCTURE, CASE REPORT, 2400*
  - ENDOMETRIUM
    - ULTRASTRUCTURE, HUMAN, 2290*
  - GASTRIC
    - 2,7-DIACETYLAMINOFLUORENE, PEPTIC ULCER, RAT, 1897*
  - MAMMARY GLAND
    - STEROID ADMINISTRATION, RAT, 1826
  - NUCLEOPROTEINS, GLYCOGEN
    - MORPHOCYTOCHEMISTRY, 2383*
  - SALIVARY GLAND
    - ONCOGENICITY, BITTNER VIRUS, MOUSE, 1933

- ADENOID CYSTIC CARCINOMA
  - SALIVARY GLAND
    - CLINICAL STUDY, 2250*

- ADENOID SQUAMOUS CELL CARCINOMA
  - LIP
    - CASE REPORTS, 2331*

- ADENOMA
  - CHROMOPHOBE
    - BLOOD VESSEL ULTRASTRUCTURE, 2298*
  - PROSTATE
    - 3H-5ALPHA-DIHYDROTESTOSTERONE METABOLISM,HUMAN, 2136
    - 3H-TESTOSTERONE METABOLISM, HUMAN, 2136

- ADENOMA, BASAL CELL
  - SALIVARY GLAND
    - CASE REPORT, 2243*

- ADENOMAS, OXYPHILIC
  - PARATHYROID GLAND
    - CLINICAL, BIOCHEMICAL STUDY, 2259*
  - SALIVARY GLAND
    - CLINICAL, BIOCHEMICAL STUDY, 2259*
  - THYROID GLAND
    - CLINICAL, BIOCHEMICAL STUDY, 2259*

- ADENOMATA
  - IMMUNOGENICITY
    - LUNG, MOUSE, 2088
  - URETHANE
    - LUNG, MOUSE, 2088

- ADENYLATE CYCLASE
  - ACTIVITY
    - ROUS SARCOMA VIRUS-TRANSFORMED CHICK CELLS, 1976
  - ACTIVITY, LEUKOCYTE
    - LEUKEMIA, RADIOIMMUNOASSAY, 2190*

- ADRENAL GLAND
  - 7,12-DIMETHYLBENA(A)ANTHRACENE
    - ULTRASTRUCTURE, RAT, 1909*
  - HORMONE-PRODUCING CARCINOMA
    - CASE REPORT, 2197*
  - THECA GRANULOSA CELL TUMOR
    - CASE REPORT, 2315*
  - TYROSINE HYDROXYLASE
    - BIOCHEMISTRY, 2391*

- ADRENOCORTICAL CARCINOMA
  - HORMONAL REGULATION
    - CORTICOSTEROIDOGENESIS, RAT, 2302*

- AFLATOXIN
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - LIVER CANCER INCIDENCE
    - KENYA, 2147

- AFLATOXIN B1
  - BIOSYNTHESIS INHIBITION
    - DIMETHYL 2,2-DICHLOROVINYL PHOSPHATE, 1854
  - HEPATOCARCINOGENESIS
    - RAT, 1861
  - PROTEIN SYNTHESIS
    - LIVER, RAT, 1819
  - PROTEIN SYNTHESIS INHIBITION
    - MONKEY KIDNEY CELL CULTURES, 1847
  - RENAL CARCINOMA
    - ULTRASTRUCTURE, RAT, 1844

- AGGLUTINATION
  - CONCANAVALIN A
    - MEMBRANE CHANGES, ATP CONTENT, TRANSFORMED HAMSTER CELLS, 1941

- AGING
  - PEROXIDATION
    - CHROMOSOME BREAKAGE, 1877

- ALANINE AMINOTRANSFERASE
  - ACTIVITY
    - HEPATOMA HYBRIDS, 2357*

- ALKYLATING AGENT
  - DNA DAMAGE
    - THERMAL ENHANCEMENT, HUMAN, 1843

- ALVEOLAR CELL CARCINOMA
  - MYCOPLASMA
    - NUCLEAR INCLUSIONS, 2286*

- AMARANTH
  - ENZYME ACTIVITY CHANGES
    - RATS, 1890

- AMELOBLASTOMA
  - TOOTH

    - ULTRASTRUCTURE, HUMAN, 2215*

- AMINO ACID
  - COMPOSITION
    - SV40 VIRUS, 2013*

- AMINOACETONITRILE
  - EFFECT ON ULTRASTRUCTURE, CYTOCHEMISTRY
    - LIVER CELLS, RAT, 1863

- O-AMINOAZOTOLUENE
  - HEPATOMA
    - GROWTH KINETICS, MOUSE, 2132

- 4-AMINOBIPHENYL
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - METABOLIC BY-PRODUCT
    - URINE, DOG, 1855
  - URINARY BLADDER TUMOR
    - DOG, 1855

- AMYLOIDOSIS
  - LYMPHOMA, MALIGNANT
    - GRAFT-VS-HOST REACTION, MOUSE, 2044

- ANDROGEN
  - ACTIVITY
    - ESTROGEN DEPENDENT TESTICULAR CARCINOMA, RAT, 2324*

- ANEMIA
  - MICROANGIOPATHIC HEMOLYTIC
    - HYPERCALCEMIA, RAT TUMOR, 2397*
  - VIRUS, ABELSON
    - RAT, 1934
  - VIRUS, MURINE SARCOMA-LEUKEMIA
    - RAT, 1934

- ANTHRACENE
  - FORMATION DURING HEATING OF FOODS
    - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820

- ANTIBODY
  - ANTIBODY-FORMING CELLS
    - STATISTICAL TREATMENT, MOUSE, 2123*
  - ANTIGEN-SPECIFIC
    - IMMUNE RESPONSE REGULATION, MOUSE, 2082
  - ANTI-P-AZOPHENYLARSONATE
    - IG CH LOCUS LINKAGE, MOUSE, 2098
  - CYTOTOXIC LYMPHOCYTE
    - LEUKEMIA, MOUSE, 2077
  - HISTOCOMPATIBILITY
    - LYMPHOID CELLS, COMPLEMENT, HUMAN, 2101
  - IMMUNOSUPPRESSION
    - MAREK'S DISEASE, 2049
  - MAMMARY TUMOR
    - IMMUNOFLUORESCENCE, RAT, 2067
  - MAREK'S DISEASE VIRUS ANTIGEN
    - CHICKEN, RABBIT, 2125*
  - RD-114 VIRAL POLYMERASE
    - HYPERIMMUNE GUINEA PIG ANTISERUM, 1984
  - SECONDARY RESPONSE
    - ANTIBODY INHIBITORY MATERIAL, LYMPH NODE, RABBIT, 2102
  - VIRUS, CYTOMEGALO-
    - SERUM, HUMAN SOLID TUMOR DONOR, 1947
  - VIRUS, EPSTEIN-BARR
    - SERUM, HUMAN SOLID TUMOR DONOR, 1947
  - VIRUS, HERPES HOMINIS
    - CANCER PATIENTS, HUMAN, 2051
  - VIRUS, HERPES SAIMIRI
    - PRIMATE, 2064
  - VIRUS, HERPES SIMPLEX 2
    - CERVICAL CARCINOMA PATIENTS, 2127*
  - VIRUS, MAREK'S DISEASE
    - IMMUNOSUPPRESSION, 2049

- ANTIGEN
  - ADENOVIRUS 2-SV40 HYBRID
    - CHARACTERIZATION, 1956
  - ALLOGENEIC
    - CELL-MEDIATED IMMUNITY, HUMAN, 2062
  - AUSTRALIA
    - DNA POLYMERASE ACTIVITY, HUMAN SERUM, 1983
  - CANCER-LIKE
    - EMBRYONIC TISSUE, NORMAL, HUMAN, 2058
  - CARCINOEMBRYONIC
    - FECES, NORMAL ADULTS, 2048
    - RADIOIMMUNOELECTROPHORETIC BINDING ASSAY, 2083
    - REVIEW, 1813*
  - CELL-SURFACE
    - FRIEND VIRUS-INDUCED TUMOR, ULTRASTRUCTURE, RAT, 2104
    - LYMPHOSARCOMA, CAT, 2065
    - MURINE LEUKEMIA VIRUS-INDUCED, RAT, 2090
    - VIRUS, FELINE C-TYPE RNA LEUKEMIA, 2065
    - VIRUS, MURINE C-TYPE RNA LEUKEMIA, 2065
  - COUPLED TUMOR PROTEIN
    - INDUCED REGRESSION, CERVICAL CARCINOMA PATIENTS, 2068
  - EMBRYONIC TISSUE, HUMAN
    - CULTURE EFFECTS, 2058
  - FRIEND VIRUS-INDUCED TUMOR
    - IMMUNO-ELECTRON MICROSCOPIC STUDIES, RAT, 2104
  - GROUP- AND SPECIES-SPECIFIC
    - SITES, HUMAN LYMPHOCYTE, 2114*
  - GROUP-SPECIFIC
    - C-TYPE VIRUS, INTRACELLULAR LOCALIZATION, 2069
  - H-2
    - LOCALIZATION, CELL MEMBRANE, MOUSE, 2103
  - HISTOCOMPATIBILITY
    - FRIEND VIRUS-INDUCED TUMOR, ULTRASTRUCTURE, RAT, 2104
    - LOCALIZATION, CELL MEMBRANE, MOUSE, 2103
    - TROPHOBLASTIC TUMORS, 2050
  - HL-A SYSTEM
    - HUMAN FIBROBLAST CELLS, 1948

  - LEUKEMIC NORMAL LYMPHOCYTE MEMBRANE DYNAMICS, 2113*
  - LYMPHOID CELLS, COMPLEMENT, HUMAN, 2101
  - MURINE SARCOMA VIRUS-TRANSFORMED CELLS, HUMAN, 1948
  - RETINOBLASTOMA, CLINICAL STUDY, 2128*
  - SV40-TRANSFORMED CELLS, HUMAN, 1948
- HODGKIN'S DISEASE
  - CHARACTERIZATION, 2087
- IMMUNE RESPONSE
  - ANTIBODY ADMINISTRATION, MOUSE, 2082
  - RENAL CELL CARCINOMA, HUMAN, 2062
- MEMBRANE
  - HUMAN B LYMPHOBLAST-SPECIFIC, 2116*
- MICROSOMAL
  - DISTRIBUTION, THYROID TUMOR, 2111*
- MYELOMA
  - DEFICIENCY, HUMAN, 2081
- NORMAL GLIAL
  - IMMUNITY, INFILTRATING GLIAL TUMOR, 2119*
- SYNGENEIC
  - IMMUNE RESPONSE, MOUSE SPLEEN CELLS, 2052
- TRANSPLANTATION
  - IMMUNE RESPONSE, MAMMARY TUMOR, MOUSE, 2084
  - IMMUNE RESPONSE, MURINE SARCOMA TUMOR CELLS, 2080
  - IMMUNE RESPONSE, MURINE TUMORS, RAT, 2066
  - ROUS SARCOMA, RAT, 2072
  - TUMOR CELL, MOUSE, 2103
- TUMOR
  - HODGKIN'S DISEASE PATIENTS, 2087
  - IMMUNE RESPONSE, CANCER PATIENTS, 2110*
- TUMOR-ASSOCIATED
  - NEPHROBLASTOMA, HUMAN, 2071
- TUMOR-SPECIFIC
  - AVEOLAR CELL CARCINOMA, HUMAN, 2085
  - CERVICAL CARCINOMA, HUMAN, 2003
  - HYPERNEPHROID CARCINOMA, HUMAN, 2096
  - RENAL CELL CARCINOMA, HUMAN, 2062
  - ROUS SARCOMA, RAT, 2072
- VIRUS, C-TYPE
  - TRANSFORMED MOUSE EMBRYO CELLS, 1982
- VIRUS, C-TYPE RNA
  - COMMON TO MURINE AND FELINE VIRUSES, 2065
- VIRUS, GAZDAR MURINE SARCOMA
  - HAMSTER TUMOR CELL, 2061
- VIRUS, HERPES 2
  - CERVICAL CARCINOMA CELLS, HUMAN CELLS, 1949
- VIRUS, HERPES SAIMIRI
  - ANTIBODY RESPONSE, PRIMATE, 2064
- VIRUS, MURINE LEUKEMIA
  - CHROMOSOME, MOUSE, 2059
  - TUMOR TRANSPLANT, VIRUS-TOLERANT RAT, 2066
- VIRUS-RELATED, ONCOGENIC
  - HODGKIN'S DISEASE PATIENTS, 2087
- VIRUS, ROUS SARCOMA
  - TEMPERATURE-SENSITIVE MUTANT, CHICK CELLS, 1957
- VIRUS, SV40
  - ANTINOMYCIN D, 2124*

ANTIGEN, EPIDERMAL
- KERATOACANTHOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RABBIT, RAT, 2078
- PAPILLOMA, SQUAMOUS
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RABBIT, RAT, 2078
- SQUAMOUS CELL CARCINOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RABBIT, RAT, 2078

ANTIGEN, INTERSPECIES
- VIRUS, C-TYPE RNA
  - PURIFICATION AND CHARACTERIZATION, 1940
- VIRUS, RAUSCHER LEUKEMIA
  - PURIFICATION AND CHARACTERIZATION, 1940

ANTIGEN, SPECIES-SPECIFIC
- VIRUS, C-TYPE
  - PRIMATE, 2055

ANTIGEN, SURFACE
- POLYOMA VIRUS-INDUCED
  - EFFECT OF INHIBITORS, HAMSTER CELLS, 2107

ANTIGEN, T
- ULTRASTRUCTURAL LOCALIZATION
  - KIDNEY CELLS, SV40-TRANSFORMED, MONKEY, 1993

ANTIGEN, THETA
- N-NITROSOBUTYLUREA LEUKEMOGENESIS
  - MOUSE, 2045

ANTISERUM
- IMMUNE RESPONSE
  - RADIATION-INDUCED LYMPHOMAS, 1913
- MYELOMA
  - IMMUNOGLOBULIN, HUMAN, 2081
- THYMUS, HUMAN
  - EFFECT ON TUMOR TRANSPLANTS, MOUSE, 2097
- VIRUS, RADIATION LEUKEMIA
  - LYMPHOMA INHIBITION, MOUSE, 1913

ARGINASE
- UROGENITAL TUMOR
  - BIOCHEMISTRY, 2396*

ARGYROPHYL CELL CARCINOID
- STOMACH
  - CASE REPORT, 2233*

ARYL HYDROCARBON HYDROXYLASE
- INDUCTION
  - 3-METHYLCHOLANTHRENE, HUMAN LYMPHOCYTES, 1829

- ARYLSULPHATASE
  - ACTIVITY
    - LEUKEMIA, CLINICAL STUDY, 2333*

- ASBESTOS
  - LUNG CANCER
    - CLINICAL STUDY, 2143
  - OCCUPATIONAL HAZARD
    - RESPIRATORY CANCER, CLINICAL STUDY, 2144

- ASCITES CELLS
  - DOUBLE STRANDED RNA
    - INHIBITOR OF PROTEIN SYNTHESIS SUBSTRATE NUCLEASE, 2226*

- ASCITES HEPATOMA
  - CLOSED CIRCULAR DNA
    - RNASE AND ALKALI SENSISTIVITY, RAT, 2299*

- ASCITES TUMOR
  - SEE ALSO EHRLICH
  - DNA POLYMERASE ACTIVITY
    - MOUSE, 2362*
  - EFFECT, CROWDING OR ISOLATION
    - MOUSE, 2238*
  - L-GLUTAMIN IN INTERCELLULAR ADHESION
    - MOUSE, CHICKEN, 2387*
  - MEMBRANE-BOUND MESSENGER RNA
    - MOUSE, 2301*
  - METABOLIC IMPAIRMENT, NORMALIZATION
    - MOUSE LIVER, 2223*
  - POLYADENYLIC ACID SEQUENCES
    - MOUSE, 2301*
  - PROLIFERATION KINETICS
    - MOUSE, 2157*

- ASTROCYTOMA
  - ROSENTHAL FIBERS, SPONGIOBLASTOMA
    - ULTRASTRUCTURE, HISTOLOGY, 2296*

- AURAMINE
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802

- AUTOIMMUNE DISORDERS
  - LYMPHOMA, MALIGNANT
    - HUMAN, 2091

- AVEOLAR CELL CARCINOMA
  - ANTIGEN, TUMOR-SPECIFIC
    - HUMAN, 2085

- ANTI-P-AZOPHENYLARSONATE
  - ANTIBODY
    - IG CH LOCUS LINKAGE, MOUSE, 2098

- BACILLUS CALMETTE-GUERIN
  - IMMUNE RESPONSE
    - POLYOMA TUMOR, RAT, 2053
  - LEUKEMIA
    - MOUSE, 2092

- BARTHOLIN'S GLAND
  - ADENOCARCINOMA
    - CASE REPORT, 2253*

- BASAL CELL ADENOMA
  - SALIVARY GLAND
    - CASE REPORT, 2243*

- BASAL CELL CARCINOMA
  - PHAGOLYSOSOME DIGESTION OF CELLULAR FRAGMENTS
    - ULTRASTRUCTURE, 2220*

- BENZ(A)ANTHRACENE
  - FORMATION DURING HEATING OF FOODS
    - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820

- BENZ(A)ANTHRACENE 5,6-OXIDE
  - EPOXIDES, K-REGION
    - METABOLIC FORMATION, HUMAN LUNG PREPARATIONS, 1859

- BENZIDINE
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802

- BENZO(A)PYRENE
  - CARCINOGENESIS, MODEL
    - ELECTRONIC STRUCTURE, RESONANCE, 1872
  - EFFECT ON LIPIDS
    - PROTOPLASMIC MEMBRANES, MITOCHONDRIA, YEAST, 1867
  - FENTON'S REAGENT
    - EFFECT ON CARCINOGENICITY, MICE, 1885
  - FERRIC CHLORIDE
    - EFFECT ON CARCINOGENICITY, MICE, 1885
  - FERROUS CHLORIDE
    - EFFECT ON CARCINOGENICITY, MICE, 1885
  - FORMATION DURING HEATING OF FOODS
    - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - HYDROGEN PEROXIDE
    - EFFECT ON CARCINOGENICITY, MICE, 1885
  - INTERACTION WITH DNA
    - SPECTROPHOTOMETRIC STUDIES, 1839
  - INTRATRACHEAL INSTILLATION
    - LUNGS, MOUSE, 1875
  - METABOLISM
    - MATERNAL TOBACCO SMOKING, HUMAN FETAL LIVER, PLACENTA, 1815
  - RESPIRATORY SYSTEM TUMORS
    - HAMSTER, 1816
    - INDUCTION, HAMSTERS, 1837

- BENZO(E)PYRENE
  - INTERACTION WITH DNA
    - SPECTROPHOTOMETRIC STUDIES, 1839

- BENZPYRENE HYDROXYLASE
  - ACTIVITY
    - CANNABIS ADMINISTRATION, LIVER, LUNG, RAT, 1814

- BLOOD
  - MALIGNOLIPIN
    - MALACHITE POWDER METHOD, 2201*

- BONE
  - MYXOMA OF JAW
    - CASE STUDY, 2255*
  - TUMORS, INCIDENCE AND HISTOLOGY
    - MOUSE, 2287*
- BRAIN TUMOR
  - ROUS SARCOMA VIRUS-INDUCED
    - CLASSIFICATION, RAT, 1986
- BREAST
  - SEE ALSO MAMMARY
  - CHANGES DURING MENSTRUAL CYCLE
    - BIOSTEREOMETRIC ANALYSIS, HUMAN, 2217*
- 5'-BROMODEOXYURIDINE
  - MUTAGENESIS
    - MURINE SARCOMA VIRUS-TRANSFORMED CELL LINE, 1935
- 5-BROMODEOXYURIDINE
  - POLYOMA VIRUS-INDUCED SURFACE ANTIGEN
    - HAMSTER CELLS, 2107
  - RESISTANCE
    - AVIAN SARCOMA VIRUS TRANSFORMED CELL, HAMSTER, 2021*
- BRONCHIAL CARCINOMA
  - HYPONATREMIA
    - CASE REPORT, 2364*
- BRONCHUS
  - CARCINOMA
    - GROWTH, CLINICAL STUDY, 2151
- BRUCELLOSIS
  - HODGKIN'S DISEASE
    - CASE REPORT, 2199*
- BURKITT'S LYMPHOMA
  - NERVOUS SYSTEM
    - CLINICAL STUDY, 2007*
    - CLINICAL STUDY, KENYA, 2006*
  - RNA TRANSCRIPTION
    - HUMAN CELLS, 1944
- BUTTER YELLOW
  - ENZYME ACTIVITY CHANGES
    - RATS, 1890
- N-BUTYL-N-(4-HYDROXYBUTYL)NITROSAMINE
  - URINARY BLADDER TUMOR
    - METABOLITE CONCENTRATION, URINE, 1884
    - RAT, AGE FACTOR, 1884
    - RAT, GUINEA PIG, HAMSTER, 1884
- BUTYLATED HYDROXYTOLUENE
  - INHIBITION OF TUMOR FORMATION
    - RAT, 1862
- BUTYLATED HYROXYTOLUENE
  - N-2-FLUORENYLACETAMIDE METABOLISM
    - RAT, 1865
  - N-HYDROXY-N-2-FLUORENYLACETAMIDE METABOLISM
    - RAT, 1865
- CAFFEINE
  - DNA REPAIR INHIBITION, MUTATION INDUCTION
    - HAMSTER, 1918*
- CALCIUM
  - BINDING TO PHOSPHOLIPIDS IN TUMOR CELLS
    - BIOCHEMICAL STUDY, 2261*
  - METABOLISM
    - EXPERIMENTAL TUMORS, 2214*
  - MITOCHONDRIA
    - EHRLICH ASCITES TUMOR, RAT, 2300*
  - TRANSPORT, EHRLICH ASCITES TUMOR CELLS
    - MOUSE, 2272*
- CANCER
  - DETECTION
    - NUCLEAR MAGNETIC RESONANCE, MOUSE, 2195*
  - HYPOXIA, ARTERIAL
    - CLINICAL STUDY, 2338*
  - WARBURG THEORY
    - REVIEW, 1807
- CANNABIS
  - BENZPYRENE HYDROXYLASE ACTIVITY
    - LIVER, LUNG, RAT, 1814
- CARBON TETRACHLORIDE
  - HEPATOCARCINOGENESIS
    - DIMETHYLNITROSAMINE, RAT, 1818
  - KIDNEY TUMOR INDUCTION
    - DIMETHYLNITROSAMINE, RAT, 1818
  - NUCLEIC ACID
    - BINDING, RAT, MOUSE, LIVER, 1894
- CARBOXYHEMOGLOBIN
  - LEVELS
    - PIPE AND CIGAR SMOKERS, 1842
- CARCINOGEN, ATMOSPHERIC
  - TRANSFORMATION
    - EMBRYO CELLS, MOUSE, RAT, 1835
- CARCINOGEN, CHEMICAL
  - HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
- CARCINOGENESIS, MODEL
  - ELECTRONIC STRUCTURE, RESONANCE
    - BENZO(A)PYRENE, BENZO(E)PYRENE, 1872
- CARCINOMA
  - CELL ALTERATION BY MEMBRANE SUGARS
    - RAT, 2346*
- CELL
  - COMPLEMENT FIXATION STUDIES
    - SUSPENSION CULTURES, HUMAN SOLID TUMORS, 1947
  - CONTACT INHIBITION OF MOVEMENT
    - MOLONEY SARCOMA VIRUS-TRANSFORMED MOUSE CELL, 1966
  - INTRACELLULAR MEMBRANE
    - POLYSOME INTERACTION, 2202*
  - SCHIZOGENIC
    - INTRACELL FORMATION, MOUSE EMBRYO

  - CELLS, 1987
- SURFACE ALTERATION
  - BIOCHEMICAL PROPERTIES, REVIEW, 1804
- SURVIVAL
  - RADIATION, MOLECULAR THEORY, 1910
- ULTRASTRUCTURE
  - SUSPENSION CULTURES, HUMAN SOLID TUMORS, 1947

CELL DIVISION
- CONTROL BY CELL MEMBRANE
  - E. COLI, 2179

CELL MEMBRANE
- ALTERATIONS
  - AGGLUTINATION BY CON A, TRANSFORMED HAMSTER CELLS, 1941
- ATP CONTENT
  - AGGLUTINATION BY CON A, TRANSFORMED HAMSTER CELLS, 1941
- CONTROL OF CELL DIVISION
  - E. COLI, 2179
- LYMPHOMA, MALIGNANT
  - BINDING SITE, CONCANAVALIN A, 2165

CELL PROLIFERATION
- VIRUS, POLYOMA
  - INHIBITION, NONTRANSFORMED CELLS, RAT, 2030*

CELL, TUMOR
- CONCANAVALIN A BINDING
  - HEMADSCYSTION METHOD, 2076
- CYTOTOXICITY
  - LYMPHOCYTES, AUTOSENSITIZED, MOUSE, 2052
  - LYMPHOCYTES, HUMAN, 2057
- DIFFERENTIAL TOXICITY OF ACIDIFIED SERUM, 2011*

CELLS, PERMISSIVE
- CONVERSION FROM NONPERMISSIVE
  - 5-IODO-2'-DEOXYRUIDINE, HUMAN, 1998

CERVICAL CARCINOMA
- ANTIGEN, TUMOR-SPECIFIC
  - HERPESVIRUS 2-INDUCED, HUMAN, 2003
- BEHAVIOR PATTERNS AND CYTOIMMUNOLOGY
  - INDUCED REGRESSION, IMMUNOACTIVE COUPLED TUMOR PROTEIN ANTIGEN, 2068
- HERPES SIMPLEX VIRUS 2
  - ANTIBODY, 2127*
- HERPESVIRUS TYPE 2-INDUCED
  - MONKEY, 2015*
- METASTASES, LYMPH NODE
  - CLINICAL STUDY, 2310*
- SQUAMOUS DYSPLASIA
  - EPIDEMIOLOGY, 2156
- VIRUS, HERPES 2
  - HUMAN, 1949
  - SEROEPIDEMIOLOGICAL STUDIES, 2149
- VIRUS, HERPES SIMPLEX
  - ETIOLOGY, HUMAN, 1951
- WOMEN UNDER 21 YEARS
  - CASE REPORTS, 2184

CHARCOAL
- CONTAMINATION DURING HEATING OF FOODS
  - POLYCYCLIC HYDROCARBON FORMATION, 1820

CHOLESTEROL
- LEVELS
  - PIPE AND CIGAR SMOKERS, 1842
- URINARY EXCRETION
  - TESTICULAR, PROSTATIC NEOPLASM, HUMAN, 2224*

CHOLESTEROL SYNTHESIS
- N-2-FLUORENYLACETAMIDE
  - PRECANCEROUS LIVER, RAT, 2130

CHROMATIN
- PHOSPHOKINASE ACTIVITY
  - LIVER TUMOR, RAT, 1845

CHROMOSOME
- CENTRAL NERVOUS SYSTEM NEOPLASMS
  - CLINICAL STUDY, 2205*
- KARYOTYPIC ABNORMALITY
  - ACUTE LEUKEMIA, HUMAN, 2175
- 6-MERCAPTOPURINE-RESISTANT TUMOR
  - RAT, 2340*
- MISSING Y
  - LEUKEMIC MALES, 2209*
- MURINE LEUKEMIA VIRUS-ASSOCIATED ANTIGENS
  - MOUSE, 2059
- NONHISTONE PROTEIN
  - HELA CELL NUCLEOPLASM, 2173
- RESCUE OF SV40 GENOME
  - MONKEY, 2026*
- SITES, RADIATION INDUCED BREAKAGE
  - HUMAN LYMPHOCYTE, 1923*

CHROMOSOME ABBERATIONS
- RADIATION, X-RAY
  - DOSIMETRY, CELL CULTURE, 1920*

CHROMOSOME ABERRATIONS
- BLOOD LEUKOCYTE CULTURE
  - ANTIOXIDANT PROTECTION, 1877
- CHEMICAL EFFECTS
  - HUMAN, REVIEW, 1846
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - ANTIOXIDANT PROTECTION, 1877
- LEUKEMIA, MYELOID
  - CYTOGENETIC STUDY, CASE REPORT, 2349*
- 4-NITROQUINOLINE-1-OXIDE
  - CULTURED HUMAN LYMPHOCYTES, 1817
- SODIUM CYCLAMATE
  - ANTIOXIDANT PROTECTION, 1877
- TRANSLOCATION, ACUTE LYMPHOCYTIC LEUKEMIA
  - FAMILIAL CASE REPORT, 2380*

CHROMOSOME BREAKAGE
- PEROXIDATION
  - AGING, 1877

CIGARETTE SMOKE
- TOBACCO
  - CARCINOGENIC PROPERTIES, 1873

- OAL TAR
  - EPIDERMIS
    - GROWTH KINETICS, MOUSE, 1848
- OLON
  - LARGE BOWEL NEOPLASMS
    - 1,2-DIMETHYLHYDRAZINE, MOUSE, 1868
- OLON CARCINOMA
  - CIRCULATING MALIGNANT CELLS
    - LONG-TERM CLINICAL STUDY, 2254*
- ONCANAVALIN A
  - AGGLUTINATION
    - MEMBRANE CHANGES, ATP CONTENT, TRANSFORMED HAMSTER CELLS, 1941
  - BINDING
    - MITOCHONDRIA, RAT LIVER, 2046
    - MOUSE CELL, 1962
    - NORMAL CELLS, TUMOR CELLS, 2076
    - PROTEASE TREATED CELL, MOUSE, 1962
    - SV40-TRANSFORMED CELL, MOUSE, 1962
  - RECEPTOR SITE DISTRIBUTION
    - ULTRASTRUCTURE, TRANSFORMED HAMSTER CELLS, 1985
  - TRANSPLANTATION
    - TRANSFORMED CELLS, INDUCED CHANGES, 2120*
- ONTACT INHIBITION
  - CELL ALTERATION BY MEMBRANE SUGARS
    - RAT, 2346*
- ORTICOSTEROIDOGENESIS
  - HORMONAL REGULATION
    - ADRENOCORTICAL CARCINOMA, RAT, 2302*
- YCLIC AMP
  - GROWTH REGULATION
    - FIBROBLAST, 2176
  - VIRUS, SV40
    - MOUSE CELLS, 1932
- YCLIC NUCLEOTIDE SYNTHETASE
  - MORRIS HEPATOMA
    - RAT, 2362*
- YCLOHEXIMIDE
  - DNA SYNTHESIS
    - POLYOMA VIRUS-INFECTED MOUSE EMBRYO CELLS, 2001
  - PROTEIN SYNTHESIS
    - POLYOMA VIRUS-INFECTED MOUSE EMBRYO CELLS, 2001
- YTOCHROME
  - VIRUS, SV40
    - TRANSFORMED AND NORMAL CELLS, COMPARATIVE STUDY, 1975
- YTOSINE ARABINOSIDE
  - POLYOMA VIRUS-INDUCED SURFACE ANTIGEN
    - HAMSTER CELLS, 2107
- YTOSONE
  - RADIOLYSIS
    - 1-CARBAMYLIMIDAZOLIDONE-4,5-DIOLS, 1916
- CYTOTOXICITY
  - CELL-MEDIATED
    - LYMPHOCYTES, MOUSE, 2129*
  - CELL, TUMOR
    - LYMPHOCYTES, AUTOSENSITIZED, MOUSE, 2052
    - LYMPHOCYTES, HUMAN, 2057
  - ETHYLNITROSOUREA
    - INHIBITION BY CYCLOHEXIMIDE, RAT, 1869
  - LEUKOCYTE
    - KIDNEY SARCOMA, RAT, 2126*
  - LYMPHOCYTE
    - CARCINOMA, HUMAN, 2070
  - METHYLNITROSOUREA
    - INHIBITION BY CYCLOHEXIMIDE, RAT, 1869
  - PESTICIDES
    - PROTEIN AND RNA SYNTHESIS INHIBITION, HELA CELLS, 1857
- DEMETHYL SULFOXIDE
  - RNA POLYMERASE IN MYELOMA CELLS
    - MOUSE, 2242*
- 2-DEOXY-D-GLUCOSE
  - VIRUS, HERPES SIMPLEX
    - REPLICATION, 2027*
- 2,7-DIACETYLAMINOFLUORENE
  - GASTRIC ADENOCARCINOMA
    - PEPTIC ULCER, VAGOTOMY, RAT, 1897*
- DIBENZ(A,C)ANTHRACENE
  - EFFECT ON LIPIDS
    - PROTOPLASMIC MEMBRANES, MITOCHONDRIA, YEAST, 1867
- DIBENZ(A,H)ANTHRACENE
  - EFFECT ON LIPIDS
    - PROTOPLASMIC MEMBRANES, MITOCHONDRIA, YEAST, 1867
- N-DIBUTYLNITROSAMINE
  - RESPIRATORY SYSTEM TUMORS
    - X-RAY DETERMINATION, HAMSTER, 1878
- DIBUTYRYL CYCLIC AMP
  - LYMPHOMA
    - CYTOLYSIS AND RESISTANT STRAIN, MOUSE, 2347*
- DIETHYLNITROSAMINE
  - DEALKYLATION ACTIVITY
    - HEPATOCARCINOGENESIS, MOUSE LIVER, 1849
  - GENETIC DAMAGE POTENTIAL
    - EVALUATION, MITOTIC CROSSING OVER TECHNIQUE, 1883
  - GLUTATHIONE PEROXIDASE
    - HEPATOMA, RAT, 1887
  - GLUTATHIONE REDUCTASE
    - HEPATOMA, RAT, 1887
  - HEPATOCARCINOGENESIS
    - COMPETITIVE RNA-DNA HYBRIDIZATION, RAT, 1834
    - EFFECT OF AMINOACETONITRILE, ULTRASTRUCTURE, LIVER CELLS, RAT, 1863

- TRANSPLACENTAL CARCINOGENESIS
  - EMBRYONIC LUNG TISSUES, HAMSTER, 1838

N-DIETHYLNITROSAMINE
- RESPIRATORY SYSTEM TUMORS
  - X-RAY DETERMINATION, HAMSTER, 1878

DIETHYLSTILBESTROL
- DNA SYNTHESIS
  - TESTES, MOUSE, 1880
- MATERNAL ADMINISTRATION
  - VAGINAL NEOPLASIA IN OFFSPRING, CASE REPORTS, 1860
- RNA, CYTOPLASM
  - TESTES, MOUSE, 1880
- TUMOR INDUCTION
  - TESTES, MOUSE, 1880

DIISOPROPYLSULFATE
- CARCINOGENESIS
  - RAT, 1892

9,10-DIMETHYL-1,2-BENZANTHRACENE
- TRANSPORT
  - RAT, 1825

DIMETHYL 2,2-DICHLOROVINYL PHOSPHATE
- AFLATOXIN B1 BIOSYNTHESIS INHIBITION
  - ASPERGILLUS PARASITICUS, 1854

N,N-DIMETHYL-P-(M-TOLYLAZO)ANILINE
- HEPATOCARCINOGENESIS
  - RAT, 1845

1-(4-DIMETHYLAMINO-BENZYLIDINE)-INDENE
- MEGALOCYTOSIS
  - ULTRASTRUCTURE, LIVER, MOUSE, 1840

DIMETHYLAMINOAZOBENZENE
- HEPATOCARCINOGENESIS
  - RNA METHYLASE ACTIVITY, RAT, HAMSTER, 1850
- RNA METHYLASE ACTIVITY
  - LIVER CELLS, RAT, HAMSTER, 1850

N,N-DIMETHYLAMINOAZOBENZENE
- LIPID TRANSPORT
  - PRENEOPLASTIC LIVER CELLS, RAT, 2131

7,12-DIMETHYLBENA(A)ANTHRACENE
- ADRENAL GLAND
  - ULTRASTRUCTURE, RAT, 1909*

7,12-DIMETHYLBENZ(A)ANTHRACENE
- CHROMOSOME ABERRATIONS
  - ANTIOXIDANT PROTECTION, 1877
- FIBROSARCOMA
  - MOUSE, 1824
- KERATOACANTHOMA
  - EPIDERMAL ANTIGENS, RABBIT, RAT, 2078
- LEUKEMIA, MYELOID
  - RAT, TRANSPLANTABLE, 1879
- LYMPHOSARCOMA
  - MOUSE, 1824
- MAMMARY TUMOR
  - RAT, 2067
  - STEROID ADMINISTRATION, RAT, 1826
- MONONUCLEAR CELLS, INDUCED TUMOR
  - ULTRASTRUCTURE, RAT, 1906*
- OVARIAN CARCINOMA
  - ENZYME HISTOCHEMISTRY, MOUSE, 1907*
- PAPILLOMA, SQUAMOUS
  - EPIDERMAL ANTIGENS, RABBIT, RAT, 2078
- SKIN TUMOR
  - UV RADIATION, MOUSE, 2141
- SQUAMOUS CELL CARCINOMA
  - EPIDERMAL ANTIGENS, RABBIT, RAT, 2078
  - MOUSE, 1824
- TRANSPLACENTAL CARCINOGENESIS
  - EMBRYONIC LUNG TISSUES, HAMSTER, 1838
- TUMORIGENESIS
  - MOUSE, 1824

9,10-DIMETHYLBENZ(A)ANTHRACENE
- PLACENTA
  - FETUS, RAT, 1893
- TRANSPLACENTAL CARCINOGENESIS
  - MOUSE, 1858

1,2-DIMETHYLHYDRAZINE
- LARGE BOWEL NEOPLASMS
  - MOUSE, 1868

DIMETHYLNITROSAMINE
- DNA
  - METHYLATED PURINE FORMATION, RAT LIVER, 1822
- DNA ALTERATION
  - FIBROBLASTS, HUMAN, 1876
- DNA REPAIR SYNTHESIS
  - FIBROBLASTS, HUMAN, 1876
- DNA TRANSCRIPTION
  - MISCODING, RAT, 1822
- GENETIC DAMAGE POTENTIAL
  - EVALUATION, MITOTIC CROSSING OVER TECHNIQUE, 1883
- HEPATOCARCINOGENESIS
  - CARBON TETRACHLORIDE, RAT, 1818
  - EFFECT OF AMINOACETONITRILE, ULTRASTRUCTURE, LIVER CELLS, RAT 1863
- KIDNEY TUMOR INDUCTION
  - CARBON TETRACHLORIDE, RAT, 1818
- RIBOSOMAL DISSOCIATION
  - MOUSE LIVER, 1903*
- TRANSPLACENTAL CARCINOGENESIS
  - EPITHELIUM, MOUSE, 1851
- TUMOR INDUCTION
  - BLOOD VESSELS, MOUSE, 1836
  - SEX FACTORS, MOUSE, 1836

DNA
- ADENOVIRUS 2-SV40 HYBRID
  - BIOPHYSICAL PROPERTIES, 1973
- ALTERED METABOLISM
  - LEUKEMIA, CYTOGENETIC AND ULTRASTRUCTURAL EVIDENCE, 2337*
- BACTERIOPHAGE PS8
  - TUMOR INDUCTION, PLANTS, 2366*
- BINDING
  - 3-METHYLCHOLANTHRENE, MOUSE, 1856

BROMODEOXYRUIDINE INCORPORATION AND DEPENDENCE
    HAMSTER MELANOMA, 2355*
CONTENT
    HEPATOMA CELLS, MOUSE, 2142
DAMAGE BY METHYL METHANESULFONATE
    THERMAL ENHANCEMENT, HUMAN, 1843
DIMETHYLNITROSAMINE
    METHYLATED PURINE FORMATION, RAT LIVER, 1822
INTERACTION WITH BENZO(A)PYRENE
    SPECTROPHOTOMETRIC STUDIES, 1839
INTERACTION WITH BENZO(E)PYRENE
    SPECTROPHOTOMETRIC STUDIES, 1839
MAMMARY TUMOR
    CYTOPHOTOMETRY, 2343*
METHYLATION
    CARCINOGENESIS, RAT LIVER, 1822
MURINE LEUKEMIA VIRUS-SPECIFIC
    PRESENCE IN NORMAL CELLS, MOUSE, 2043*
REPAIR ENDONUCLEASE
    MAMMAL, 2322*
REPAIR INHIBITION, MUTATION INDUCTION
    CAFFEINE, HAMSTER, 1918*
REPAIR REPLICATION
    LYMPHOMA CELLS, IRRADIATED, 1914
RNASE AND ALKALI SENSITIVITY
    ASCITES HEPATOMA, RAT, 2299*
SARCOMAS, ADENOCARCINOMAS
    MORPHOCYTOCHEMISTRY, 2383*
SEQUENCES EXPRESSED AS RNA
    GENE AMPLIFICATION, COW, 2182
SINGLE STRAND BREAK
    N-METHYL-N'-NITRO-N-NITROSOGUANIDINE, BACILLUS SUBTILIS, 1900*
STRAND BREAK
    GAMMA RADIATION INDUCED, 1919*
STRAND BREAKAGE, REPAIR, CELL DEATH
    REVIEW, 1915
SUPERCOILING WITHOUT REPLICATION
    SV40 TRANSFORMED MONKEY KIDNEY CELLS, 2033*
SV40
    CLEAVAGE BY RESTRICTION ENDONUCLEASE, 2019*
TEMPLATE ACTIVITY, RNA POLYMERASE
    REACTION MEDIUM DEPENDENCE, 2164
THYMIDINE INCORPORATION
    NOVIKOFF HEPATOMA CELLS, RAT, 2267*
VALUES
    MYELOMA PATIENTS, 2189*
VIRUS, HERPES
    CARCINOGENESIS, REVIEW, 1805
VIRUS, PAPILLOMA
    CARCINOGENESIS, REVIEW, 1805
VIRUS, POLYOMA
    CARCINOGENESIS, REVIEW, 1805
    PROTEIN AND MESSENGER RNA REQUIREMENTS FOR SUPERHELICITY, 2001
VIRUS-SPECIFIC
    ADENOVIRUS 2-SV40 HYBRID, 1956
VIRUS, SV40
    CARCINOGENESIS, REVIEW, 1805

DNA LIGASE
  ENZYME-ADENYLATE COMPLEX
    CALF, 2345*

DNA POLYMERASE
  ACTIVITY
    AUSTRALIA ANTIGEN, HUMAN SERUM, 1983
    CROWN GALL TUMOR, TOBACCO, 2192*
  RNA-DEPENDENT
    RD-114 VIRUS, HYPERIMMUNE GUNIEA PIG ANTISERUM, 1984
  RNA-DIRECTED
    DNA-RNA COMPLEX, CHICKEN EMBRYO, 1942
  SARCOMA 180 ASCITES CELLS
    MOUSE, 2382*
  TEMPLATE RESPONSE, REGENERATING LIVER
    RAT, 2275*
  VIRUS, AVIAN MYELOBLASTOSIS
    INHIBITION BY POLY(U) FRACTIONS, 1963

DNA REPAIR SYNTHESIS
  DIMETHYLNITROSAMINE
    FIBROBLASTS, HUMAN, 1876

DNA REPLICATION
  VIRUS, SV40
    KIDNEY CELLS, MONKEY, 1970

DNA SYNTHESIS, 2167
  CYCLOHEXIMIDE
    POLYOMA VIRUS-INFECTED MOUSE EMBRYO CELLS, 2001
  DIETHYLSTILBESTROL
    TESTES, MOUSE, 1880
  EHRLICH ASCITES TUMOR
    MOUSE, 2246*
  3H-THYMIDINE UPTAKE
    MURINE LEUKEMIA VIRUS-INFECTED CELLS, MOUSE, 1974
  HERPESVIRUS-HOST CELL INTERACTION
    BIOCHEMISTRY, REVIEW, 1809
  INDUCTION, CONTACT-INHIBITED CELLS
    BOVINE ADENOVIRUS, 3, 2037*
  INHIBITION
    5-FLUORODEOXYURIDINE, SV40-INFECTED MONKEY CELL CULTURES, 1960
  3-METHYLCHOLANTHRENE
    MAMMARY NODULE, MOUSE, 1856
  MITOTIC ACTIVITY
    ADENOVIRUS 12-INFECTED RAT EMBRYO CELLS, 2002
  PYRIMIDINE METABOLISM
    NORMAL AND TUMOR TISSUE, MAMMAL, 2365*
  RADIATION, GAMMA
    ISOPROTERENOL, MOUSE SALIVARY GLAND CELLS, 1911
  RNA TEMPLATE
    MOUSE MYELOMA, 2305*
  VIRAL, GENETIC MUTATION
    BACTERIOPHAGE T7 INFECTED CELLS, 2041*
  VIRUS, POLYOMA
    MOUSE, 1927

DNA TRANSCRIPTION

    DIMETHYLNITROSAMINE
        MISCODING, RAT, 1822
    VIRUS, POLYOMA
        KIDNEY CELL, MOUSE, 2005
    VIRUS, SV40
        KIDNEY CELLS, MONKEY, 1970
        RNA POLYMERASE, MONKEY KIDNEY CELLS, 1995

EHRLICH ASCITES CELL
    GROWTH
        BIOCHEMICAL, HISTOCHEMICAL, ULTRASTRUCTURAL CHANGES, 2210*

EHRLICH ASCITES TUMOR
    CALCIUM TRANSPORT
        MOUSE, 2272*
    DNA SYNTHESIS
        MOUSE, 2246*
    GLUTAMIC ACID METABOLISM
        BIOCHEMICAL, HISTOCHEMICAL STUDY, 2332*
    MITOCHONDRIA INTERACTION WITH CALCIUM
        RAT, 2300*
    NICOTINAMIDE-ADENINE DINUCLEOTIDES
        KINETICS, 2388*
    POTASSIUM ION EFFECT
        ELECTRICAL CHARACTERISTICS, 2245*
    RNA POLYMERASE
        PURIFICATION, 2164

ENDOMETRIUM
    ADENOCARCINOMA
        ULTRASTRUCTURE, HUMAN, 2290*
    CARCINOMA SITE DETERMINATION FROM BODY FLUID
        CLINICAL STUDY, 2258*

ENDOPLASMIC RETICULUM
    MEMBRANE ENZYME ACTIVITY
        PLASTOCYTOMA PHENOTYPES, MOUSE, 2216*
    MEMBRANE-POLYSOME INTERACTION, 2202*

ENVIRONMENTAL HAZARD
    CHROMOSOME ABERRATIONS
        CHEMICAL MUTAGENS, HUMAN, REVIEW, 1846

ENZYME
    SEE ALSO NAMES OF
    ALANINE AMINOTRANSFERASE
        HEPATOMA HYBRIDS, 2357*
    ANDENYLATE CYCLASE
        LEUKEMIA, RADIOIMMUNOASSAY, 2190*
    ARGINASE
        UROGENITAL TUMOR, 2396*
    ARYLSULPHATASE ACTIVITY
        LEUKEMIA, CLINICAL STUDY, 2333*
    COENZYME Q LEVELS
        FRIEND LEUKEMIA VIRUS-INFECTED MICE, 1996
    DNA LIGASE
        ENZYME-ADENYLATE COMPLEX, CALF, 2345*
    ERYTHROCYTIC CATALASE
        LUNG CANCER, MOLECULAR STUDY, 2344*
    GUANYL CYCLASE ACTIVITY
        MORRIS HEPATOMA, RAT, 2362*
    BETA-KETOADIPATE PATHWAY MODIFICATION
        N-METHYL-N'-NITRO-N-NITROSOGUANIDINE, 1905*
    LYSOSOMAL SECRETION
        RAT YOSHIDA HEPATOMA, 2262*
    MEMBRANE ACTIVITY
        PLASTOCYTOMA PHENOTYPES, MOUSE, 2216*
    MYELOPEROXIDASE
        SERUM, LEUKEMIA, 2358*
    PATHWAYS, NUCLEOTIDE METABOLISM
        HUMAN LYMPHOCYTE AND LEUKEMIA CELLS, 2264*
    PATTERNS
        MOUSE TUMOR, HUMAN AND MOUSE EMBRYO, 2196*
    REVERSE TRANSCRIPTASE
        TRANSFORMED CELL LINES, HUMAN NEOPLASM, 2228*
    TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE ACTIVITY
        CROWN GALL TUMOR, TOBACCO, 2192*
    TYROSINE HYDROXYLASE
        ADRENAL GLAND, NEUROBLASTOMA, 2391*

EPIDERMIS
    COAL TAR
        GROWTH KINETICS, MOUSE, 1848
    METHOTREXATE
        GROWTH KINETICS, MOUSE, 1848
    SULFUR
        GROWTH KINETICS, MOUSE, 1848

EPIDERMODYSPLASIA VERRUCIFORMIS
    VIRUS, WART-LIKE
        SKIN, CASE REPORTS, 1999

EPIDERMOID CARCINOMA
    DERMAL GRAFT
        CASE REPORT, 2354*

EPITHELIOMA
    CHRONIC PROGRESSIVE KERATO-ATROPHODERMA
        CASE REPORTS, 2361*

EPITHELIUM
    PROSTATE
        HERPES-LIKE VIRUS, HUMAN, 1992
    TRANSPLACENTAL CARCINOGENESIS
        DIMETHYLNITROSAMINE, MOUSE, 1851

EPOXIDES, K-REGION
    BENZ(A)ANTHRACENE 5,6-OXIDE
        METABOLIC FORMATION, HUMAN LUNG PREPARATIONS, 1859

ERYTHROBLASTOSIS
    VIRAL INDUCTION
        THYMIC LYMPHOMA INOCULATION, MOUSE, RAT, 2008*

ERYTHROCYTIC CATALASE
    ACTIVITY
        LUNG CANCER, MOLECULAR STUDY, 2344*

- ERYTHROCYTOSIS
  - PULMONARY CARCINOMA
    - FAMILY STUDY. 2394*
- ESOPHAGEAL CARCINOMA
  - EPIDEMIOLOGY
    - CEYLON. BETEL LEAVE CHEWING. 2150
- ESOPHAGUS
  - CANCER. INTRAABDOMINAL METASTASES
    - LAPAROSCOPIC DIAGNOSIS. 2377*
  - MELANOMA. MALIGNANT
    - ULTRASTRUCTURE. 2390*
- ESTRADIOL
  - VARIATION. TISSUE CULTURE SERA
    - BOVINE. 2279*
- ESTROGEN. 2166
  - RECEPTOR
    - MAMMARY CARCINOMA. HUMAN. 2186
    - MAMMARY TUMOR. RAT AND HUMAN SPECIMENS. 2271*
- ESTRONE
  - VARIATION. TISSUE CULTURE SERA
    - BOVINE. 2279*
- ETHIONINE
  - HEPATOCARCINOGENESIS
    - RNA METHYLASE ACTIVITY. RAT. HAMSTER. 1850
  - RNA METHYLASE ACTIVITY
    - LIVER CELLS. RAT. HAMSTER. 1850
- ETHYLENETHIOUREA
  - THYROID CARCINOMA
    - RAT. 1864
- ETHYLNITROSOUREA
  - CYTOTOXICITY
    - INHIBITION BY CYCLOHEXIMIDE. RAT. 1869
  - NEURAL TISSUE
    - CYTOTOXICITY. RAT. 1869
- EYE
  - CILIARY PIGMENT EPITHELIUM TUMOR
    - ULTRASTRUCTURE. CASE REPORT. 2400*
  - MELANOMA
    - EPIDEMIOLOGY. 2148
- FERRITIN
  - HEPATOMA
    - IMMUNOLOGICAL CHARACTERIZATION. RAT. 1881
- ALPHA-FETOPROTEIN
  - HEPATOMA
    - RADIOIMMUNOASSAY. 2121*
- FIBROADENOMA
  - MAMMARY GLAND
    - STEROID ADMINISTRATION. RAT. 1826
- FIBROBLAST
  - GROWTH
    - INHIBITION. SERUM DEPRIVATION. 2176
- FIBROMA
  - OSSIFYING AND CEMENTIFYING. JAW
    - CLINICAL STUDY. CHILDREN. 2289*
- FIBROSARCOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - MOUSE. 1824
  - MACROPHAGE CULTURE PREPARATION
    - MOUSE. RAT. 2266*
  - PROSTAGLANDIN E2. BONE RESORPTION-STIMULUS
    - MOUSE. 2278*
  - STIMULATED GRANULOCYTOPOIESIS
    - MOUSE. 2211*
- FLUORANTHRENE
  - FORMATION DURING HEATING OF FOODS
    - CONTAMINATION ON GRILLS OVER CHARCOAL. 1820
- N-2-FLUORENYLACETAMIDE
  - CHOLESTEROL SYNTHESIS
    - PRECANCEROUS LIVER. RAT. 2130
  - METABOLISM
    - BUTYLATED HYDROXYTOLUENE EFFECT. RAT. 1865
    - PRECANCEROUS LIVER. RAT. 2130
  - TUMOR INDUCTION
    - INHIBITION BY BUTYLATED HYDROXYTOLUENE. RAT. 1862
- 5-FLUORODEOXYURIDINE
  - DNA SYNTHESIS INHIBITION
    - SV40-INFECTED MONKEY CELL CULTURES. 1960
- 5-FLUOROURACIL
  - POLYOMA VIRUS-INDUCED SURFACE ANTIGEN
    - HAMSTER CELLS. 2107
- FOOD
  - CARCINOGENIC HYDROCARBON FORMATION
    - CONTAMINATION ON GRILLS OVER CHARCOAL. 1820
- GALACTOSYL TRANSFER
  - MASTOCYTOMA
    - MOUSE. 2273*
- GALLBLADDER DISEASE
  - ORAL CONTRACEPTIVES
    - INCIDENCE. BOSTON. 2153
- GASTRIC CARCINOMA
  - N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
    - RAT. SURFACTANT EFFECT. 1886
  - PITUITARY GLAND-ADRENAL CORTEX SYSTEM
    - CLINICAL STUDY. 2342*
- GASTRIC MUCOSA
  - CELL ALTERATIONS
    - MALIGNANT TUMORS. HUMAN. 2140
  - RADON. ORAL ADMINISTRATION
    - RADIATION. RAT. 1924*
- GASTROINTESTINAL CARCINOMA
  - EPIDEMIOLOGY
    - PATHOLOGY. NEW ZEALAND. 2152

GENETICS
  CARCINOID TUMOR METASTASIS
    CASE REPORTS, 2277*
  CONGENITAL TESTICULAR TERATOMA
    MOUSE, 2265*
  DEFICIENT INFORMATION CODED FOR BY
   VIRUS
    SHOPE PAPILLOMA VIRUS, ARGININEMIC
     PATIENT, 2023*
  FAMILIAL OCCURRENCE OF THYMOMA
    CASE REPORT, 2244*
  MEMBRANE FUNCTION REGULATION
    MAMMALIAN CELL CULTURE, 2203*
  PULMONARY CARCINOMA, ABNORMAL
   HEMOGLOBIN
    FAMILY STUDY, 2394*

GENITALIS, HERPES
  CLINICAL STUDY, 2014*
  INDUCED CERVICAL INFECTION AND
   MALIGNANCY
    MONKEY, 2015*

GIANT CELL TUMOR, MALIGNANT
  TENDON SHEATH
    ULTRASTRUCTURE, CASE REPORT, 2284*

GLIOMA
  GROWTH, APPEARANCE
    TISSUE CULTURE, 2212*

GLIOMA, MALIGNANT
  BRAIN STEM
    CLINICAL STUDY, 2213*

GLUTAMIC ACID
  METABOLISM
    EHRLICH ASCITES TUMOR, 2332*

GLUTATHIONE PEROXIDASE
  DIETHYLNITROSAMINE
    HEPATOMA, RAT, 1887

GLUTATHIONE REDUCTASE
  ACTIVITY
    HUMAN, ANIMAL TUMOR, 2168
  DIETHYLNITROSAMINE
    HEPATOMA, RAT, 1887

GLYCERYL ETHERS
  TUMOR
    MOUSE, INSECT, 2194*

GLYCOLIPID
  INCORPORATION OF 2-DEOXY-D-GLUCOSE
   INTO GLYCOLIPIDS
    NORMAL AND SV40 TRANSFORMED
     HAMSTER CELLS, 2371*

GRAFT-VS-HOST REACTION
  LYMPHOMA, MALIGNANT
    AMYLOIDOSIS, MOUSE, 2044

GRANULOCYTE
  LEUKEMIA, CHRONIC MYELOGENOUS
    ENZYME DISTRIBUTION, HUMAN, 2139
  PRECURSORS, LEUKEMIA
    HUMAN, 2139

GRANULOCYTIC SARCOMA
  LEUKEMIA, MYELOGENOUS
    ULTRASTRUCTURE, CASE REPORT, 2292*

GRANULOCYTOSIS
  STIMULATION
    MICE BEARING FIBROSARCOMA, 2211*

GRANULOMATOSIS
  LYMPH NODE
    VASCULAR ALTERATIONS, 2326*

GROWTH
  BRONCHOGENIC CARCINOMA
    CLINICAL STUDY, 2151
  CONTINUOUS
    EXPERIMENTAL TUMOR CELLS, 2204*
  EHRLICH ASCITES CELL
    BIOCHEMICAL, HISTOCHEMICAL,
     ULTRASTRUCTURAL CHANGES, 2210*
  EPIDERMIS
    COAL TAR, METHOTREXATE, SULFUR,
     MOUSE, 1848
  FIBROBLAST
    INHIBITION, SERUM DEPRIVATION,
     2176
  HEPATOMA
    O-AMINOAZOTOLUENE-INDUCED, MOUSE,
     2132
  HUMAN TUMOR TISSUE
    THYMUS ANTISERUM EFFECT, MOUSE,
     2097
  KIDNEY SARCOMA INHIBITION
    LEUKOCYTE, RAT, 2126*
  LUNG CANCER
    CLINICAL STUDY, 2341*
  MENINGEOMA
    CASE REPORT, 2158*
  MORRIS HEPATOMA
    CYCLIC AMP, CYCLIC GMP, RAT, 2191*
  PROLIFERATION KINETICS
    ASCITES TUMOR, MOUSE, 2157*

3H-5ALPHA-DIHYDROTESTOSTERONE
  METABOLISM
    NORMAL AND ADENOMATOUS PROSTATE,
     HUMAN, 2136

3H-TESTOSTERONE
  METABOLISM
    NORMAL AND ADENOMATOUS PROSTATE,
     HUMAN, 2136

HALO NEVUS
  OXYMETHOLONE TREATMENT
    CASE REPORT, 1904*

HEART
  TUMORS, CYSTS
    X-RAY FINDINGS, 2327*

HELA CELLS
  HISTONE MESSENGER RNA
    IDENTIFICATION, 2207*

HEMANGIOPERICYTOMA
  NOSE
    ULTRASTRUCTURE, CASE REPORT, 2260*
  THYROID GLAND

    - CASE REPORT, 2398*

- HEPATOCARCINOGENESIS
  - AFLATOXIN
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - AFLATOXIN B1
    - RAT, 1861
  - 4-AMINOBIPHENYL
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - AURAMINE
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - BENZIDINE
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - BENZO(A)PYRENE
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - CARBON TETRACHLORIDE
    - DIMETHYLNITROSAMINE, RAT, 1818
  - CARCINOGEN, CHEMICAL
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - DIETHYLNITROSAMINE
    - COMPETITIVE RNA-DNA HYBRIDIZATION, RAT, 1834
    - EFFECT OF AMINOACETONITRILE, ULTRASTRUCTURE, LIVER CELLS, RAT, 1863
  - DIETHYLNITROSAMINE DEALKYLATION ACTIVITY
    - MOUSE LIVER, 1849
  - N,N-DIMETHYL-P-(M-TOLYLAZO)ANILINE
    - RAT, 1845
  - DIMETHYLAMINOAZOBENZENE
    - RNA METHYLASE ACTIVITY, RAT, HAMSTER, 1850
  - DIMETHYLNITROSAMINE
    - CARBON TETRACHLORIDE, RAT, 1818
    - EFFECT OF AMINOACETONITRILE, ULTRASTRUCTURE, LIVER CELLS, RAT, 1863
  - ENZYME ACTIVITY CHANGES
    - RATS, 1890
  - ETHIONINE
    - RNA METHYLASE ACTIVITY, RAT, HAMSTER, 1850
  - 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    - CARCINOGEN-BINDING PROTEIN DISTRIBUTION, RAT, 1831
    - SULFATE ESTER FORMATION, RAT, 1852
  - 2-NAPHTHYLAMINE
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - STILBESTROL
    - HAMSTER, RAT, MOUSE, REVIEW, 1802
  - URETHANE
    - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830

- HEPATOMA
  - SEE ALSO LIVER
  - O-AMINOAZOTOLUENE
    - GROWTH KINETICS, MOUSE, 2132
  - DIFFERENTIATED FUNCTIONS IN HYBRIDS
    - ALANINE AMINOTRANSFERASE, 2357*
  - DNA CONTENT
    - TUMOR CELL NUCLEI, MOUSE, 2142
  - DYSFIBRINOGENEMIA
    - CASE REPORT, 2393*
  - ENZYME ACTIVITY IN GLUCOSE METABOLISM
    - CLINICAL STUDY, AUTOPSY, 2285*
  - ALPHA-FETOPROTEIN
    - RADIOIMMUNOASSAY, 2121*
  - LIPID ANTIOXIDANT ACTIVITY
    - EXPERIMENTAL CARCINOGENESIS, MOUSE, 2160
  - 3'-METHYL-4-(DIMETHYLAMINO)AZOBENZENE
    - RAT, 1881
  - PHENYLALANINE HYDROXYLASE
    - RAT, 2274*
  - PYRUVATE KINASE
    - KINETIC STUDY, ADULT AND FETAL LIVER, 2306*
  - RIBOSOMAL RNA, NUCLEOTIDE SEQUENCE
    - LIVER, MOUSE, 2193*
  - RNA, TRANSFER
    - FETAL RAT, 2171
  - TRANSFER RNA
    - RAT, 2222*

- HEPATOMA, ASCITES
  - CLOSED CIRCULAR DNA
    - RNASE AND ALKALI SENSISTIVITY, RAT, 2299*

- HEPATOMA, MORRIS
  - N-ACETYLNEURAMINIC ACID SYNTHESIS
    - RAT, 2270*
  - CYCLIC AMP, CYCLIC GMP
    - RAT, 2191*
  - GUANYL CYCLASE ACTIVITY
    - RAT, 2362*
  - MINERAL CONTENT
    - NECROSIS, RAT, 2170
  - NMR OF WATER, 2385*

- HEPATOMA, MOUSE
  - LIVER
    - RIBOSOMAL RNA, NUCLEOTIDE SEQUENCE, 2193*

- HEPATOMA, NOVIKOFF
  - THYMIDINE TRANSPORT, INCORPORATION INTO DNA
    - RAT, 2267*

- HEPATOMA, YOSHIDA
  - LYSOSOMAL ENZYME ACTIVITY
    - RAT, 2262*

- HEPATOMA, ZAJDELA
  - TRANSFER RNA ACCEPTOR CAPACITY
    - RAT, 2178

- HISTOCOMPATABILITY
  - LYMPHOCYTE
    - MOUSE, MAN, 2115*

- HISTOCOMPATIBILITY
  - ANTIBODY
    - LYMPHOID CELLS, COMPLEMENT, HUMAN, 2101
  - ANTIGEN
    - FRIEND VIRUS-INDUCED TUMOR, ULTRASTRUCTURE, RAT, 2104
    - LOCALIZATION, CELL MEMBRANE, MOUSE, 2103
    - TROPHOBLASTIC TUMORS, 2050

- HISTONE
  - LYSINE-RICH
    - NORMAL, NEOPLASTIC TISSUE, 2374*

- RNA, MESSENGER
  - IDENTIFICATION, HELA CELLS, 2207*

HODGKIN'S DISEASE
- ANTIGEN
  - CHARACTERIZATION, 2087
- BRUCELLOSIS
  - CASE REPORT, 2199*
- CELL LINES
  - CASE REPORT, 2348*
- LEUKEMIA DEVELOPMENT AFTER CHEMOTHERAPY
  - CASE REPORTS, 1898*
- LUNG SCANNING
  - CLINICAL STUDY, 2376*
- NUCLEOPROTEINS, GLYCOGEN
  - MORPHOCYTOCHEMISTRY, 2383*
- PATTERN OF INVOLVEMENT
  - CLINICAL STUDY, 2269*
- RNA LABELING
  - DIAGNOSTIC, PROGNOSTIC, 2319*
- SERUM COMPLEMENT LEVELS
  - CLINICAL STUDY, 2106
- SERUM COPPER LEVEL
  - CLINICAL STUDY, 2317*
- TRANSFORMATION, LYMPHOCYTE
  - PHYTOHEMAGGLUTININ, POKEWEED MITOGEN, 2099
- VASCULAR ALTERATIONS
  - HISTOPATHOLOGY, 2326*

HORMONE
- SEE ALSO NAMES OF
- HORMONE-PRODUCING CARCINOMA
  - ADRENAL GLAND, CASE REPORT, 2197*
- MAMMARY CARCINOMA, GROWTH REQUIREMENT
  - RAT, CELL CULTURE, 2163
- VARIATION, TISSUE CULTURE SERA
  - BOVINE, 2279*

HORMONE, STEROID
- 7,12-DIMETHYLBENZ(A)ANTHRACENE INDUCED MAMMARY TUMORS
  - RAT, 1826

HYALINE GLOBULES
- NEOPLASMS
  - CASE REPORTS, 2282*

HYBRIDIZATION
- DNA-RNA
  - DIETHYLNITROSAMINE
    - HEPATOCARCINOGENESIS, RAT, 1834
- HUMAN LYMPHOID LEUKEMIA PERMANENT CELL LINE
  - MOUSE FIBROBLAST CELLS, 2095
- REVERSION
  - SV40-TRANSFORMED HAMSTER AND MOUSE CELLS, 1955
- RNA
  - MURINE MYELOMA TUMORS, MOUSE, 2183

HYDROCARBON, POLYCYCLIC
- CARCINOGENESIS
  - REVIEW, 1801
- FORMATION DURING HEATING OF FOODS
  - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820

N-HYDROXY-2-ACETYLAMINOFLUORENE
- ESTERIFICATION DEPRESSION
  - AMINOAZO DYE CARCINOGENESIS, LIVER, RAT, 1852

N-HYDROXY-N-2-FLUORENYLACETAMIDE
- METABOLISM
  - BUTYLATED HYDROXYTOLUENE EFFECT, RAT, 1865
- TUMOR INDUCTION
  - INHIBITION BY BUTYLATED HYDROXYTOLUENE, RAT, 1862

HYPERCALCEMIA
- MICROANGIOPATHIC HEMOLYTIC ANEMIA
  - RAT TUMOR, 2397*
- MULTIPLE MYELOMA
  - CASE REPORT, 2198*

HYPERCALCEMIA, RAT
- TUMOR
  - MICROANGIOPATHIC HEMOLYTIC ANEMIA, 2397*

HYPERNEPHROIC CARCINOMA
- ANTIGEN, TUMOR-SPECIFIC
  - DETECTION, HUMAN, 2096

HYPERPLASIA
- ORAL FOCAL EPITHELIAL
  - INCIDENCE, GEOGRAPHIC ASPECTS, 2146

ILEUM
- CARCINOID TUMOR METASTASIS
  - FAMILIAL OCCURRENCE, CASE REPORTS, 2277*

IMMUNE RESPONSE
- ANTIGEN
  - ANTIBODY ADMINISTRATION, MOUSE, 2082
  - RENAL CELL CARCINOMA, HUMAN, 2062
- ANTIGEN, TUMOR
  - CANCER PATIENTS, 2110*
- ANTISERUM
  - RADIATION-INDUCED LYMPHOMAS, 1913
- BACILLUS CALMETTE-GUERIN
  - POLYOMA TUMOR, RAT, 2053
- CELL-MEDIATED
  - MURINE SARCOMA VIRUS, MOUSE, 2056
- METHYLCHOLANTHRENE
  - SPLEEN CELLS, MOUSE, 1821
- SERUM, IMMUNE
  - TUMOR GROWTH, CONTROL, MOUSE, 2063
- TRANSPLANTATION ANTIGENS
  - MURINE SARCOMA VIRUS, MOUSE, 2080
- VIRUS, MURINE SARCOMA
  - CELL-MEDIATED, MOUSE, 2056

IMMUNITY
- ANTITUMOR CELL-MEDIATED
  - METASTASIS CONTROL, MOUSE, 2063
- ANTITUMOR SERUM-MEDIATED
  - METASTASIS CONTROL, MOUSE, 2063
- CELL BOUND, AGAINST NORMAL GLIAL ANTIGEN
  - INFILTRATING GLIAL TUMOR, HUMAN, 2119*

  CELL-MEDIATED
    HERPESVIRUS HOMINIS, 2051
    LEUKOCYTE MIGRATION TEST, 2089
    LEUKOPLAKIA, HUMAN, 2089
    LYMPHOCYTE, HUMAN, CARCINOMA, 2070
    NERVOUS SYTEM TUMORS, HUMAN, 2057
    RENAL CELL CARCINOMA, HUMAN, 2062
    ROUS SARCOMA, RAT, 2072
  HOST RANGE CONVERSION
    MURINE SARCOMA-LEUKEMIA COMPLEX,
      2118*
  HUMORAL ANTIBODY
    LYMPHOCYTE, HUMAN, CARCINOMA, 2070
  LYMPHOCYTE
    GLIOMA, HUMAN, 2057
  LYMPHOCYTE CYTOTOXICITY
    POLYOMA TUMOR, RAT, 2053
  SERUM BLOCKING ACTIVITY
    POLYOMA TUMOR, RAT, 2053
  SERUM-MEDIATED
    FACILITATION OF PULMONARY
      METASTASES, MOUSE, 2135
  TRANSPLANTATION ANTIGENS
    MURINE SARCOMA VIRUS, MOUSE, 2080
  TUMOR, POLYOMA
    RAT, 2053
  TUMOR-SPECIFIC
    MURINE LEUKEMIA VIRUS, RAT, 2066
    ROUS SARCOMA, RAT, 2072
  VIRUS, ROUS SARCOMA
    AGE FACTOR, RAT, INOCULATION, 2072

IMMUNITY, ACTIVE
  INDUCTION
    SENDAI VIRUS-FUSED MULTINULCEATED
      TUMOR CELLS, MOUSE, 2047

IMMUNIZATION, TUMOR
  RADIATION, X-RAY
    MOUSE, 2086

IMMUNOBLASTOMA, MALIGNANT
  INDUCTION, HAMSTER
    TRANSPLANTED CELLS, HUMAN, 2074

IMMUNODEPRESSION
  METHYLCHOLANTHRENE
    MOUSE, 1821

IMMUNOGENICITY
  ADENOMATA
    LUNG, MOUSE, 2088
  MAMMARY TUMOR
    TRANSPLANTATION, MOUSE, 2084
  METHYLCHOLANTHRENE-INDUCED SARCOMA
    MOUSE, 1832

IMMUNOGLOBULIN
  BINDING SITE
    MYELOMA, MOUSE, 2108
  MAREK'S DISEASE
    IGG AND IGM ANTIBODY, 2049
  MYELOMA
    BINDING INHIBITION, ANTISERUM,
      HUMAN, 2081
  SYNTHESIS
    LYMPHOBLASTOID CELL LINES, HUMAN,
      2074

IMMUNOGLOBULIN FC
  RECEPTORS
    MALIGNANT TISSUES, HUMAN, 2109

IMMUNOGLOBULIN IGG
  HEMADSORPTION
    MALIGNANT TISSUES, HUMAN, 2109

IMMUNOLOGY
  C-TYPE VIRUS
    ANTIGENS, PRIMATE, 2055
  VIRUS, ONCORNA
    HUMAN CANCER CELL LINE, 1952
  VIRUS, ROUS SARCOMA
    HIGH ONCOGENICITY VARIANTS, MOUSE,
      2105

IMMUNOSUPPRESSION
  PYRAN COPOLYMER
    ANTI-LYMPHOCYTE SERUM, LEUKEMIA,
      MOUSE, 2079
  VIRUS, MAREK'S DISEASE
    ANTIBODY, 2049

INHALATION CARCINOGENESIS
  AUTOMOTIVE EXHAUST
    RAT, LONG-TERM STUDY, 1827

INSULIN
  VARIATION, TISSUE CULTURE SERA
    BOVINE, 2279*

INTERFERON
  INDUCTION
    RAUSCHER LEUKEMIA VIRUS, MOUSE,
      1977

INTESTINE
  MALAKOPLAKIA
    CASE REPORT, 2351*

INTESTINE, SMALL
  LYMPHOMA
    CASE REPORT, 2316*
  TUMOR RARITY, 2373*

5-IODO-2'-DEOXYURIDINE
  CONVERSION OF NONPERMISSIVE CELLS
    CYTOMEGALOVIRUS, HUMAN, 1996

ISOPROTERENOL
  DNA SYNTHESIS
    GAMMA RADIATION, MOUSE SALIVARY
      GLAND CELLS, 1911

JAW
  BENIGN FIBRO-OSSEOUS LESIONS
    CLINICAL STUDY, CHILDREN, 2289*
  MYXOMA
    CASE STUDY, 2255*
  PRIMARY AND SECONDARY MALIGNANT TUMORS
    CLINICAL STUDY, CHILDREN, 2187*

KERATOACANTHOMA
  ANTIGEN, EPIDERMAL
    7,12-DIMETHYLBENZ(A)ANTHRACENE,
      RABBIT, RAT, 2078
  7,12-DIMETHYLBENZ(A)ANTHRACENE
    EPIDERMAL ANTIGENS, RABBIT, RAT,

    2078

KIDNEY
  ADENOVIRUS ISOLATION
    FROG, 1953
  CALCIUM LOSS, MULTIPLE MYELOMA
    CASE REPORT, 2198*
  JUXTAGLOMERULAR CELL TUMOR
    ULTRASTRUCTURE, 2370*
  RENAL CARCINOMA
    AFLATOXIN B1, ULTRASTRUCTURE, RAT, 1844
  RENAL CELL CARCINOMA
    CASE REPORTS, 2234*
  RENIN-SECRETING TUMOR
    ULTRASTRUCTURE, 2370*
  TUMOR INDUCTION
    DIMETHYLNITROSAMINE, CARBON TETRACHLORIDE, RAT, 1818

KIDNEY TUMOR
  ANTIGEN, TUMOR-ASSOCIATED
    CHARACTERIZATION, HUMAN, 2071

LACTATE DEHYDROGENASE
  ACTIVITY, STOMACH CANCER
    CLINICAL STUDY, 2276*
  ISOENZYMES
    TESTICULAR INTERSTITIAL TUMOR GRAFTS, RAT, 2335*

LARYNX
  CARCINOMA
    CLINICAL STUDY, 2399*

LEUKEMIA
  ACUTE PROMYELOCYTIC
    PROCOAGULANT, FIBRINOLYTIC ACTIVITY, 2386*
  ADENYLATE CYCLASE ACTIVITY, LEUKOCYTE
    RADIOIMMUNOASSAY, 2190*
  ALTERED DNA METABOLISM
    CYTOGENETIC, ULTRASTRUCTURAL EVIDENCE, 2337*
  ARYLSULPHATASE ACTIVITY
    CLINICAL STUDY, 2333*
  ASCITES ALLOGRAFT REJECTION BY LYMPHOCYTES
    IN VITRO STUDY, MOUSE, 2257*
  AUTOPSY STUDY
    ATOMIC BOMB SURVIVORS, HIROSHIMA-NAGASAKI, 1917*
  BACILLUS CALMETTE-GUERIN
    MOUSE, 2092
  BONE MARROW CELL CO-CULTIVATION
    OSTEOSARCOMA CELLS, TRANSFORMING ACTIVITY, 2208*
  CYTOTOXIC LYMPHOCYTE
    ANTIBODY, MOUSE, 2077
  EFFECT, CROWDING OR ISOLATION
    MOUSE, 2238*
  EOSINOPHILIA
    MORPHOLOGY, CYTOCHEMISTRY, CASE REPORT, 2334*
  INTRADERMAL AND SUBCUTANEOUS TUMORS
    ULTRASTRUCTURE, GUINEA PIGS, 2247*
  LACTOFERRIN
    MEASUREMENT IN SERUM, 2359*
  MISSING Y CHROMOSOME
    HUMAN MALES, 2209*
  MIXED LEUKOCYTE CULTURE REACTIVITY
    HUMAN, 2177
  MUCOPOLYSACCHARIDE SULFATION, LEUKOCYTES
    CLINICAL STUDY, 2235*
  MYELOBLAST CELL COLONY
    AVIAN MYELOBLASTOSIS VIRUS RELEASE, 2035*
  MYELOPEROXIDASE
    MEASUREMENT IN SERUM, 2358*
  NUCLEOTIDE METABOLISM
    HUMAN ENZYMATIC PATHWAYS, 2264*
  PERIANAL, ANORECTAL COMPLICATIONS
    CLINICAL STUDY, 2311*
  RHEOLOGY
    LEUKOCYTES, LEUKOCYTE SUSPENSIONS, BLOOD, 2314*
  SERUM, ANTI-LYMPHOCYTE
    PYRAN COPOLYMER, MOUSE, 2079
  SPLEEN CELLS
    ERYTHROCYTE ACCUMULATION, MOUSE, 1990
  SUSCEPTIBILITY
    BONE MARROW CULTURE TEST, HUMAN, 2134
  VIRUS, ABELSON
    RAT, 1934
  VIRUS, C-TYPE
    GUINEA PIG, 1989, 2000
  VIRUS, HERPES-LIKE
    GUINEA PIG, 1989
  VIRUS, MOLONEY LEUKEMIA
    SEROCHEMOTHERAPY, MOUSE, 1988

LEUKEMIA, ACUTE
  BONE MARROW
    DEFECTIVE COLONY FORMATION, HUMAN, 2175
  INDUCTION, HAMSTER
    TRANSPLANTED CELLS, HUMAN, LYMPHOBLASTIC LEUKEMIA, 2074
  MYELOCYTE PROLIFERATION
    AUTORADIOGRAPHY, 2395*
  SERUM MURAMIDASE
    CLINICAL STUDY, 2360*

LEUKEMIA, ACUTE LYMPHOCYTIC
  CENTRAL NERVOUS SYSTEM
    ARACHNOID, 2320*
  CHROMOSOME TRANSLOCATION
    FAMILIAL CASE REPORT, 2380*

LEUKEMIA, ACUTE MYELOBLASTIC
  CHEMOTHERAPY, HODGKIN'S DISEASE
    CASE REPORTS, 1898*
  MONOBLAST
    ULTRASTRUCTURE, 2221*

LEUKEMIA, ACUTE MYELOCYTIC
  RIEDER CELL MICROTUBULES
    ULTRASTRUCTURE, 2392*

LEUKEMIA, ACUTE MYELOGENOUS
  BONE MARROW CELLS
    MATURATION DEFECT, HUMAN, 2134

LEUKEMIA, ACUTE MYELOID
  TISSUE CULTURE STUDY

- HUMAN, REVIEW, 1803

LEUKEMIA, CHRONIC MYELOGENOUS
- GRANULOCYTE
  - ENZYME DISTRIBUTION, HUMAN, 2139
- TRANSFORMATION, ACUTE
  - MONOCLONAL, HUMAN, 2137

LEUKEMIA, CHRONIC MYELOID
- TISSUE CULTURE STUDY
  - HUMAN, REVIEW, 1803

LEUKEMIA, CLINICAL STUDY
- ARYLSULPHATASE
  - ACTIVITY, 2333*
- ENZYME
  - ARYLSULPHATASE ACTIVITY, 2333*

LEUKEMIA, LYMPHOBLASTIC
- TERMINAL TRANSFERASE
  - CELL STUDY, 2307*

LEUKEMIA, LYMPHOCYTIC
- LIPID ANTIOXIDANT ACTIVITY
  - EXPERIMENTAL CARCINOGENESIS, MOUSE, 2160

LEUKEMIA, MONOCYTIC
- LEUKOCYTE ALKALINE PHOSPHATASE
  - CASE REPORTS, 2389*

LEUKEMIA, MYELOCYTIC
- ERYTHROBLASTIC PHASE
  - AUTONOMOUS ERYTHROPOIESIS, CASE REPORTS, 2356*

LEUKEMIA, MYELOGENOUS
- GRANULOCYTIC SARCOMA
  - ULTRASTRUCTURE, CASE REPORT, 2292*

LEUKEMIA, MYELOID
- CHROMOSOME ABERRATIONS
  - CYTOGENETIC STUDY, CASE REPORT, 2349*
- CHRONIC JUVENILE TYPE
  - ULTRASTRUCTURE, CLINICAL STUDY, 2232*
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - RAT, TRANSPLANTABLE, 1879

LEUKEMIA, RETICULOENDOTHELIAL
- CELL CHARACTERISTICS
  - HUMAN, 2054
- CELL ULTRASTRUCTURE
  - HUMAN, 2054
- IMMUNOGLOBULIN, SURFACE-BOUND
  - CELLS, HUMAN, 2054

LEUKEMOGENESIS
- N-NITROSOBUTYLUREA
  - THETA ANTIGEN, MOUSE, 2045
- RADIATION-INDUCED, PROTECTION
  - SERA, IMMUNE AND NONIMMUNE, 1913

LEUKOCYTE
- CYTOTOXICITY
  - KIDNEY SARCOMA, RAT, 2126*
- REACTIVITY
  - LEUKEMIC CELLS, HUMAN, 2177

LEUKOPLAKIA
- IMMUNITY, CELL-MEDIATED
  - LEUKOCYTE MIGRATION TEST, HUMAN, 2089

LIGAND
- BINDING SITE
  - MYELOMA PROTEIN, MOUSE, 2108

LIP
- ADENOID SQUAMOUS CELL CARCINOMA
  - CASE REPORTS, 2331*
- BASAL CELL ADENOMA
  - CASE REPORT, 2243*

LIPID
- EFFECTS OF CARCINOGENIC HYDROCARBONS
  - PROTOPLASMIC MEMBRANES, MITOCHONDRIA, YEAST, 1867
- GLYCERYL ETHERS
  - MOUSE, INSECT, 2194*
- TRANSPORT INTO INTERPHASE NUCLEI
  - PRENEOPLASTIC RAT LIVER, 2131

LIVER
- BENZPYRENE HYDROXYLASE ACTIVITY
  - CANNABIS ADMINISTRATION, RAT, 1814
- CANCER INCIDENCE
  - AFLATOXIN INGESTION, KENYA, 2147
- CARCINOMA
  - ABO BLOOD GROUPS, 2122*
- CHARACTERIZATION OF CYTOCHROME P-450 IN MICROSOMES
  - RAT, 1896*
- HEPATOCARCINOGENESIS
  - DIETHYLNITROSAMINE DEALKYLATION ACTIVITY, MOUSE, 1849
  - DIMETHYLNITROSAMINE, CARBON TETRACHLORIDE, RAT, 1818
  - PHOSPHOKINASE ACTIVITY, CHROMATIN, RAT, 1845
- HEPATOCELLULAR TUMOR
  - ULTRASTRUCTURE, 2328*
- INTRACYTOPLASMIC HYALIN GLOBULES
  - CASE REPORTS, 2282*
- INTRAHEPATIC CHOLANGIOCARCINOMA, CLONORCHIASIS
  - CLINICAL STUDY, 2295*
- LIPID ANTIOXIDANT ACTIVITY
  - EXPERIMENTAL CARCINOGENESIS, MOUSE, 2160
- LIPID TRANSPORT
  - PRENEOPLASTIC CELLS, RAT, 2131
- MEGALOCYTOSIS
  - 1-(4-DIMETHYLAMINO-BENZYLIDINE)-INDENE, ULTRASTRUCTUREM MOUSE, 1840
- MINERAL CONTENT
  - RAT, 2170
- BETA-NAPHTHYLAMINE
  - IMMUNOLOGICAL AND MORPHOLOGICAL CHANGES, DOG, MOUSE, 1823
- PRIMARY CARCINOMA
  - EPIDEMIOLOGY, UGANDA, 2155
- PROTEIN, NUCLEAR
  - THIOACETAMIDE-TREATED RATS, 1833
- PROTEIN SYNTHESIS INHIBITION
  - AFLATOXIN B1, RAT, 1819
- PROTEINS, BILEAFLET NUCLEAR MEMBRANE

    - RAT, BIOCHEMICAL STUDY, 2268*
  - PYRUVATE KINASE
    - KINETIC STUDY, ADULT AND FETAL LIVER AND HEPATOMA, 2306*
  - REGENERATION AND HEPATOMA
    - PROTEIN SYNTHESIS, 2330*
  - RIBOSOMAL RNA, NUCLEOTIDE SEQUENCE
    - HEPATOMA, MOUSE, 2193*
  - RNA SYNTHESIS INHIBITION
    - URETHANE, RAT, 1841
  - TEMPLATE RESPONSE OF DNA POLYMERASE
    - REGENERATING RAT, 2275*

- LIVER, FETAL
  - BENZO(A)PYRENE METABOLISM
    - MATERNAL TOBACCO SMOKING, HUMAN, 1815
  - N-METHYLANILINE METABOLISM
    - MATERNAL TOBACCO SMOKING, HUMAN, 1815

- LUNG
  - ADENOMATA
    - URETHANE-INDUCED, MOUSE, 2088
  - ALVEOLAR CELL CARCINOMA
    - NUCLEAR INCLUSIONS, 2286*
  - BENZPYRENE HYDROXYLASE ACTIVITY
    - CANNABIS ADMINISTRATION, RAT, 1814
  - BRONCHIAL MUCOSA
    - HUMAN ORGAN CULTURE, 2263*
    - NORMAL, METAPLASTIC AND DYSPLASTIC STATE MAINTENANCE, 2263*
  - CARCINOMA SITE DETERMINATION FROM BODY FLUID
    - CLINICAL STUDY, 2258*
  - EMBRYONAL TISSUE
    - URETHANE, TRANSPLACENTAL ACTION, 1908*
  - HAMARTOMAS
    - X-RAY DIAGNOSIS, 2367*
  - HERPESVIRUS HOMINIS 2-ABORTIVE INFECTION
    - HUMAN EMBRYONIC CELLS, 1959
  - INTRACYTOPLASMIC HYALIN GLOBULES
    - CASE REPORTS, 2282*
  - MESENCHYMOMA, MALIGNANT
    - CASE REPORT, 2280*
  - 4-NITROQUINOLINE-1-OXIDE, BRONCHIOLE SUSCEPTIBILITY
    - ULTRASTRUCTURE, MOUSE, 1902*
  - OAT-CELL CARCINOMA, BRONCHUS
    - HYPONATREMIA, CASE REPORT, 2364*
  - PLASMA CELL GRANULOMA
    - CASE REPORTS, 2230*
    - CLINICAL STUDY, 2252*
  - PULMONARY CARCINOMA, ABNORMAL HEMOGLOBIN
    - FAMILY STUDY, 2394*
  - PULMONARY METASTASIS
    - SERUM-MEDIATED, MOUSE, 2135
  - SCANNING
    - HODGKIN'S DISEASE, CLINICAL STUDY, 2376*
  - TRANSPLACENTAL CARCINOGENESIS
    - EMBRYONIC TISSUES, HAMSTER, 1838

- LUNG CANCER
  - ARTERIAL SCANNING
    - CLINICAL DIAGNOSIS, 2375*
  - ASBESTOS
    - CLINICAL STUDY, 2143
  - ERYTHROCYTIC CATALASE ACTIVITY
    - MOLECULAR STUDY, 2344*
  - GROWTH, METASTASES
    - CLINICAL STUDY, 2341*
  - TNM-CLASSIFICATION SYSTEM
    - CLINICAL STUDY, 2309*
  - TOBACCO SMOKING
    - CLINICAL STUDY, 1899*
    - REVIEW, 1810*

- LYMPH NODE
  - GRANULOMATOSIS
    - VASCULAR ALTERATIONS, 2326*
  - SECONDARY ANTIBODY RESPONSE
    - ANTIBODY INHIBITORY MATERIAL, RABBIT, 2102

- LYMPHOBLAST
  - HUMAN B
    - SPECIFIC MEMBRANE ANTIGEN, 2116*
  - RESPONSE, CANCER CELLS
    - INHIBITION, PLASMA SIALOGLYCOPROTEINS, 2219*
  - VIRUS, EPSTEIN-BARR
    - HALOGENATED PYRIMIDINE, 2060

- LYMPHOCYTE
  - ARYL HYDROCARBON HYDROXYLASE INDUCTION
    - 3-METHYLCHOLANTHRENE, HUMAN, 1829
  - B-CELL PRECURSOR
    - SPECIFICITY RESTRICTION, MOUSE, 2093
  - BONE MARROW-DERIVED
    - MONOSPECIFICITY, MOUSE, 2094
  - CYTOTOXIC
    - ANTIBODY, LEUKEMIA, MOUSE, 2077
  - CYTOTOXICITY
    - CARCINOMA, HUMAN, 2070
  - CYTOTOXICITY, CELL-MEDIATED
    - TUMOR, MOUSE, 2129*
  - GROUP- AND SPECIES-SPECIFIC ANTIGENIC SITES
    - HUMAN, 2114*
  - HISTOCOMPATABILITY
    - MOUSE, MAN, 2115*
  - IMMUNITY
    - GLIOMA, HUMAN, 2057
  - LEUKEMIC, NORMAL
    - RESPONSE, HL-A COMPLEX, 2113*
  - NUCLEOTIDE METABOLISM
    - HUMAN ENZYMATIC PATHWAYS, 2264*
  - T-CELL
    - TRANSPLANTATION TOLERANCE, RAT, 2100
  - TRANSFORMATION
    - IMMUNE RESPONSE, CELL-MEDIATED, MOUSE, 2056
    - PHYTOHEMAGGLUTININ, POKEWEED MITOGEN, HODGKIN'S DISEASE, 2099
    - POST-SURGERY, CANCER PATIENTS, 2112*
    - REGIONAL NODE CELLS, 2168*
    - SCOPE AND LIMITATIONS OF PLASMA FREE MEDIUM, 2318*

- LYMPHOCYTES
  - DESTRUCTION OF ASCITES LEUKEMIA

- ALLOGRAFT
  - IN VITRO STUDY, MOUSE, 2257*

LYMPHOCYTES, AUTOSENSITIZED
- TUMORIGENESIS
  - MOUSE, 2052

LYMPHOID HYPERPLASIA
- EXTRATHORACIC ANGIOFOLLICULAR
  - MYASTHENIA GRAVIS, CASE REPORT, 2256*

LYMPHOID TUMOR
- RADIO-IRON INCORPORATION
  - CHICKEN, 2248*

LYMPHOMA
- DIBUTYRYL CYCLIC AMP
  - CYTOLYSIS AND RESISTANT STRAIN, MOUSE, 2347*
- FREQUENCEY
  - ZYMOSAN TREATED MICE, 2117*
- HERPES-LIKE VIRUS
  - RABBIT, 2017*
- INDUCTION
  - HERPESVIRUS SAIMIRI, MONKEY, 1943
- INTESTINE, SMALL
  - CASE REPORT, 2316*
- VIRUS, RADIATION LEUKEMIA
  - MOUSE, 1913

LYMPHOMA, MALIGNANT
- AMYLOIDOSIS
  - GRAFT-VS-HOST REACTION, MOUSE, 2044
- AUTOIMMUNE DISORDERS
  - HUMAN, 2091
- CELL MEMBRANE
  - BINDING SITE, CONCANAVALIN A, 2165

LYMPHOMAGENESIS
- GRAFT-VS-HOST REACTION
  - MOUSE, 2044

LYMPHOSARCOMA
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - MOUSE, 1824

MACROPHAGE
- FIBROSARCOMA, CULTURE PREPARATION
  - MOUSE, RAT, 2266*

MALAKOPLAKIA
- INTESTINE
  - CASE REPORT, 2351*

MALIGNANCY
- DIAGNOSIS
  - NUCLEOLAR MORPHOLOGY, 2161

MALIGNOLIPIN
- BLOOD
  - MALACHITE POWDER METHOD, 2201*
- ISOLATION FAILURE
  - PICRATE METHOD, 2200*

MAMMARY CARCINOMA
- CHRONICITY
  - CASE REPORTS, 2363*
- ESTROGEN RECEPTOR
  - HUMAN, 2166, 2186
- HORMONE GROWTH REQUIREMENT
  - RAT, CELL CULTURE, 2163
- INFILTRATING LOBULAR
  - CLINICAL STUDY, 2249*
- LOBULAR
  - IN SITU DIAGNOSIS, CLINICAL STUDY, 2312*
- METASTASIS
  - AUTOPSY STUDY, 2237*
- PROTEIN, DNA-ASSOCIATED
  - SYNTHESIS ALTERATION, MOUSE, RAT, 2180
- SEBUM, 17-OXOSTEROID
  - CLINICAL STUDY, 2206*
- SITE DETERMINATION, PLEURAL AND PERITONEAL FLUID
  - CLINICAL STUDY, 2258*
- TRITIATED THYMIDINE PERFUSION
  - ULTRASTRUCTURE, MOUSE, 2321*
- URINARY EXCRETION, CORTICOSTEROID SULPHATES
  - CLINICAL STUDY, 2313*

MAMMARY GLAND
- ADENOCARCINOMA
  - STEROID ADMINISTRATION, RAT, 1826
- FIBROADENOMA
  - STEROID ADMINISTRATION, RAT, 1826
- HYPERPLASTIC NODULE, PLACENTAL LACTOGEN
  - OVARIECTOMIZED MOUSE, 2159
- 3-METHYLCHOLANTHRENE
  - TUMORIGENICITY, MOUSE, 1856
- POLYPLOID SEX CHROMATIN TUMORS
  - DNA CONTENT, HUMAN, 2343*
- TUMOR
  - ALDOLASE ACTIVITY, HUMAN, MOUSE, 2329*

MAMMARY TUMOR
- ANTIBODY
  - IMMUNOFLUORESCENCE, RAT, 2067
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - RAT, 2067
  - STEROID ADMINISTRATION, RAT, 1826
- ESTROGEN RECEPTOR
  - RAT AND HUMAN SPECIMENS, 2271*
- ETIOLOGY, VIRAL
  - HUMAN, 1991
- IMMUNOGENICITY
  - TRANSPLANTATION, MOUSE, 2084
- INHIBITION, TEREPHTHALIC ACID
  - MOUSE, 2225*
- INTRACYTOPLASMIC HYALIN GLOBULES
  - CASE REPORTS, 2282*
- ORAL CONTRACEPTIVES
  - INCIDENCE, BOSTON, 2153
- SPONTANEOUS
  - DOG, 2133
- VIRUS, RNA
  - HUMAN, MOUSE, 1991

MAREK'S DISEASE
- IMMUNOGLOBULIN
  - IGG AND IGM ANTIBODY, 2049
- ONCOGENESIS
  - CHICKEN, REVIEW, 1806

- MASTOCYTOMA
    - GALACTOSYL TRANSFER
        - MOUSE, 2273*

- MATERNAL TOBACCO SMOKING, HUMAN FETAL LIVER, PLACENTA
    - BENZO(A)PYRENE
        - METABOLISM, 1815
    - N-METHYLANILINE
        - METABOLISM, 1815

- MEDULLARY CARCINOMA
    - THYROID
        - GRANULES AND AMYLOID, CALCITONIN DISTRIBUTION, CASE REPORTS, 2293*
    - THYROID GLAND
        - ULTRASTRUCTURE, 2353*

- MEDULLOEPITHELIOMA
    - CEREBRAL
        - CASE REPORT, 2297*

- MEGALOCYTOSIS
    - 1-(4-DIMETHYLAMINO-BENZYLIDINE)-INDENE
        - ULTRASTRUCTURE, LIVER, MOUSE, 1840

- MELANOMA
    - BROMODEOXYRUIDINE INCORPORATION AND DEPENDENCE
        - HAMSTER, 2355*
    - EFFECT, CROWDING OR ISOLATION
        - MOUSE, 2238*
    - EYE
        - EPIDEMIOLOGY, 2148
    - METASTATIC CELL LINES
        - MOUSE, 2169
    - PREMELANOSOMES AND MELANOSOMES
        - HUMAN AND MOUSE, 2381*

- MELANOMA, HARDING-PASSY
    - PIGMENT FRACTIONS
        - MOUSE, 2339*

- MELANOMA, MALIGNANT
    - CYSTEINYL DOPA
        - CASE REPORT, 2384*
    - ESOPHAGUS
        - ULTRASTRUCTURE, 2390*

- MEMBRANE
    - DYNAMICS
        - ANTI-HL-A ANTIBODY, LEUKEMIC AND NORMAL LYMPHOCYTE, 2113*
    - ENZYME ACTIVITY
        - PLASTOCYTOMA PHENOTYPES, MOUSE, 2216*
    - FUNCTION, GENETIC REGULATION
        - MAMMALIAN CELL CULTURE, 2203*
    - INTRACELLULAR
        - POLYSOME INTERACTION, 2202*
    - SUGARS
        - CELL ALTERATION, RAT, 2346*

- MEMBRANE, CELL
    - LECTIN-BINDING SITES
        - SV40-TRANSFORMED CELL, 1946

- MEMBRANE, PLASMA
    - CLEAVAGE INHIBITION
        - NORMAL AND SV40 TRANSFORMED MOUSE CELLS, 2010*
    - MALIGNANT CELLS
        - RESEARCH, REVIEW, 2162

- MENINGEOMA
    - GROWTH
        - CASE REPORT, 2158*

- MENINGIOMA, MALIGNANT
    - EXTRACRANIAL METASTASES, CASE REPORTS
        - CASE REPORTS, 2352*

- MESENCHYMOMA, MALIGNANT
    - LUNG
        - CASE REPORT, 2280*

- METABOLISM
    - BENZO(A)PYRENE
        - MATERNAL TOBACCO SMOKING, HUMAN FETAL LIVER, PLACENTA, 1815
    - CALCIUM
        - EXPERIMENTAL TUMORS, 2214*
    - DEAMINATION IMPAIRMENT, NORMALIZATION
        - TUMOR-BEARING MICE, 2223*
    - DNA, ALTERED
        - LEUKEMIA, CYTOGENETIC AND ULTRASTRUCTURAL EVIDENCE, 2337*
    - ENZYME ACTIVITY IN GLUCOSE METABOLISM
        - MALIGNANT HEPATOMA, 2285*
    - N-2-FLUORENYLACETAMIDE
        - BUTYLATED HYDROXYTOLUENE EFFECT, RAT, 1865
        - PRECANCEROUS LIVER, RAT, 2130
    - GLUTAMIC ACID
        - EHRLICH ASCITES TUMOR, 2332*
    - 3H-5ALPHA-DIHYDROTESTOSTERONE
        - NORMAL AND ADENOMATOUS PROSTATE, HUMAN, 2136
    - 3H-TESTOSTERONE
        - NORMAL AND ADENOMATOUS PROSTATE, HUMAN, 2136
    - N-HYDROXY-N-2-FLUORENYLACETAMIDE
        - BUTYLATED HYDROXYTOLUENE EFFECT, RAT, 1865
    - INCORPORATION OF 2-DEOXY-D-GLUCOSE INTO GLYCOLIPIDS
        - NORMAL AND SV40 TRANSFORMED HAMSTER CELLS, 2371*
    - N-METHYLANILINE
        - MATERNAL TOBACCO SMOKING, HUMAN FETAL LIVER, PLACENTA, 1815
    - NUCLEIC ACID
        - TESTES, DIETHYLSTILBESTROL, MOUSE, 1880
    - NUCLEOTIDE
        - HUMAN LYMPHOCYTE AND LEUKEMIA CELLS, 2264*
    - PYRIMIDINE
        - NORMAL AND TUMOR TISSUE, MAMMAL, 2365*

- METASTASIS
    - BROWN-PEARS CARCINOMA
        - THYROID, ADRENAL CORTEX CHANGE, RABBIT, 2227*
    - CARCINOID TUMOR, ILEUM
        - FAMILIAL OCCURRENCE, CASE REPORTS,

        2277*
    DIAGNOSIS
        NUCLEOLAR MORPHOLOGY, 2161
    ESOPHAGEAL CANCER
        LAPAROSCOPIC DIAGNOSIS, 2377*
    EXTRACRANIAL, MALIGNANT MEINGIOMA
        CASE REPORTS, 2352*
    INHIBITION BY WARFARIN
        MOUSE, 2136
    LUNG CANCER
        CLINICAL STUDY, 2341*
    LYMPH NODE, CERVICAL
        CLINICAL STUDY, 2310*
    MAMMARY CARCINOMA
        AUTOPSY STUDY, 2237*
    MELANOMA, CELL LINE SELECTION
        MOUSE, 2169
    PROSTATE CARCINOMA TO TESTIS
        CASE REPORTS, 2240*
    PULMONARY
        SERUM-MEDIATED, MOUSE, 2135

METHOTREXATE
    EPIDERMIS
        GROWTH KINETICS, MOUSE, 1848

3'-METHYL-4-DIMETHYL-AMINOBENZENE
    DNA PYRIMIDINE SYNTHESIS
        LIVER, RAT, 2167

3'-METHYL-4-(DIMETHYLAMINO)AZOBENZENE
    HEPATOMA
        RAT, 1881

3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    HEPATOCARCINOGENESIS
        CARCINOGEN-BINDING PROTEIN
          DISTRIBUTION, RAT, 1831
        SULFATE ESTER FORMATION, RAT, 1852
    ISOLATION OF BINDING PROTEIN
        ALCOHOL DEHYDROGENASE, 1828

METHYL METHANESULFONATE
    DNA DAMAGE
        THERMAL ENHANCEMENT, HUMAN, 1843

N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
    DNA SINGLE STRAND BREAK
        BACILLUS SUBTILIS, 1900*
    ENZYME MODIFICATION
        BETA-KETOADIPATE PATHWAY, 1905*
    GASTRIC CARCINOMA
        RAT, SURFACTANT EFFECT, 1886

N-METHYL-N-NITROSO-N'-NITRO-GUANIDINE
    MUTAGENESIS
        MOUSE, DOMINANT-LETHAL ASSAY, 1888

N-METHYL-N-NITROSOUREA
    RESPIRATORY SYSTEM TUMORS
        ULTRASTRUCTURE, HAMSTER, 1870
    SQUAMOUS CELL CARCINOMA
        LUNG, HAMSTER, 1870

N-METHYLANILINE
    METABOLISM
        MATERNAL TOBACCO SMOKING, HUMAN
          FETAL LIVER, PLACENTA, 1815

METHYLATION
    DNA
        CARCINOGENESIS, RAT LIVER, 1822

METHYLCHOLANTHRENE
    CARCINOGENESIS
        MOUSE, 1821
    IMMUNE RESPONSE
        SPLEEN CELLS, MOUSE, 1821
    IMMUNODEPRESSION
        MOUSE, 1821
    SARCOMA
        IMMUNOGENICITY, MOUSE, 1832
    TUMOR INDUCTION
        MOUSE, 2135

3-METHYLCHOLANTHRENE
    ARYL HYDROCARBON HYDROXYLASE INDUCTION
        HUMAN LYMPHOCYTES, 1829
    DNA BINDING
        MAMMARY NODULE, MOUSE, 1856
    DNA SYNTHESIS
        MAMMARY NODULE, MOUSE, 1856
    MAMMARY GLAND
        TUMORIGENICITY, MOUSE, 1856

METHYLNITROSOUREA
    CYTOTOXICITY
        INHIBITION BY CYCLOHEXIMIDE, RAT,
          1869
    NERVOUS SYSTEM TUMORS
        RAT, 1871
    NEURAL TISSUE
        CYTOTOXICITY, RAT, 1869

MICROSOME
    CHARACTERIZATION OF CYTOCHROME P-450
        RAT LIVER, 1896*

MILK
    VIRUS, ONCORNA
        PRODUCTION OF CORES, HUMAN, 1972

MITOCHONDRIA
    CALCIUM
        EHRLICH ASCITES TUMOR, RAT, 2300*
    CONCANAVALIN A BINDING
        RAT LIVER, 2046

MITOGEN, POKEWEED
    TRANSFORMATION, LYMPHOCYTE
        PHYTOHEMAGGLUTININ, HODGKIN'S
          DISEASE, 2099

MITOMYCIN C
    POLYOMA VIRUS-INDUCED SURFACE ANTIGEN
        HAMSTER CELLS, 2107

MITOSIS
    DNA SYNTHESIS
        ADENOVIRUS 12-INFECTED RAT EMBRYO
          CELLS, 2002

MORPHOLOGY
    VIRUS, C-TYPE
        GUINEA PIG, 2000

MUCOPOLYSACCHARIDE
    SULFATION, LEUKEMIC LEUKOCYTES

- CLINICAL STUDY, 2235*

MURAMIDASE
- SERUM LEVELS
  - ACUTE LEUKEMIA, CLINICAL STUDY, 2360*

MUTAGEN, CHEMICAL
- CHROMOSOME ABERRATIONS
  - HUMAN, REVIEW, 1846

MUTAGENESIS
- 5'-BROMODEOXYURIDINE
  - MURINE SARCOMA VIRUS-TRANSFORMED CELL LINE, 1935
- N-METHYL-N-NITROSO-N'-NITRO-GUANIDINE
  - MOUSE, DOMINANT-LETHAL ASSAY, 1888
- N-NITROSO-N-METHYLUREA
  - MOUSE, DOMINANT-LETHAL ASSAY, 1888
- N-NITROSOMORPHOLINE
  - MOUSE, DOMINANT-LETHAL ASSAY, 1888

MUTATION
- BROMODEOXYURIDINE DEPENDENCE
  - HAMSTER MELANOMA, 2355*

MUTATION, CHEMICAL
- DETECTION SYSTEM
  - SACCHAROMYCES CEREVISIAE, MITOTIC CROSSING O, 1883

MYCOPLASMA
- ALVEOLAR CELL CARCINOMA
  - NUCLEAR INCLUSIONS, 2286*

MYELOMA
- ANTIGEN
  - DEFICIENCY, HUMAN, 2081
- ANTISERUM
  - IMMUNOGLOBULIN, HUMAN, 2081
- DNA VALUES
  - CLINICAL STUDY, 2189*
- IMMUNOGLOBULIN
  - BINDING INHIBITION, ANTISERUM, HUMAN, 2081
- IMMUNOGLOBULIN BINDING SITE
  - 2,4-DINITRO- AND 2,4,6-TRINITROPHENYL LIGANDS, MOUSE, 2108
- PHOSPHOLIPIDS AND DIMETHYLSULFOXIDE EFFECT ON RNA POLYMERASE
  - MOUSE, 2242*
- RNA HYBRIDIZATION
  - MOUSE, 2183
- RNA ISOLATION, DNA SYNTHESIS
  - MOUSE, 2305*

MYELOMA, MULTIPLE
- CALCIUM LOSS, KIDNEY
  - CASE REPORT, 2198*

MYELOPEROXIDASE
- LEUKEMIA
  - MEASUREMENT IN SERUM, 2358*

MYOEPITHELIOMA
- SALIVARY GLAND
  - ULTRASTRUCTURE, CASE REPORT, 2283*

MYXOMA
- JAW
  - CASE STUDY, 2255*

1-NAPHTHYLAMINE
- GENETIC DAMAGE POTENTIAL
  - EVALUATION, MITOTIC CROSSING OVER TECHNIQUE, 1883

2-NAPHTHYLAMINE
- GENETIC DAMAGE POTENTIAL
  - EVALUATION, MITOTIC CROSSING OVER TECHNIQUE, 1883
- HEPATOCARCINOGENESIS
  - HAMSTER, RAT, MOUSE, REVIEW, 1802

BETA-NAPHTHYLAMINE
- LIVER
  - IMMUNOLOGICAL AND MORPHOLOGICAL CHANGES, DOG, MOUSE, 1823

NEOPLASMS
- HYALINE GLOBULES
  - CASE REPORTS, 2282*
- LARGE BOWEL
  - 1,2-DIMETHYLHYDRAZINE, MOUSE, 1868
- NERVOUS SYSTEM, CENTRAL
  - CHROMOSOMAL CHARACTERISTICS, 2205*
- REVERSE TRANSCRIPTASE
  - TRANSFORMED CELL LINES, HUMAN, 2228*

NEPHROBLASTOMA
- ANTIGEN, TUMOR-ASSOCIATED
  - CHARACTERIZATION, HUMAN, 2071

NERVE TUMOR
- PNEUMOGASTRIC
  - CASE REPORTS, 2369*

NERVOUS SYSTEM
- BURKITT'S LYMPHOMA
  - CLINICAL STUDY, 2007*
  - CLINICAL STUDY, KENYA, 2006*

NERVOUS SYSTEM, CENTRAL
- ARACHNOID
  - ACUTE LYMPHOCYTIC LEUKEMIA, 2320*
- BRAIN STEM, MALIGNANT GLIOMA
  - CLINICAL STUDY, 2213*
- CEREBRAL MEDULLOEPITHELIOMA
  - CASE REPORT, 2297*
- MALIGNANT GLIOMA
  - DETECTION, 2119*
- NEOPLASMS
  - CHROMOSOMAL CHARACTERISTICS, 2205*

NERVOUS SYSTEM TUMORS
- ANIMAL MODEL, RAT
  - HUMAN BRAIN TUMOR COMPARISON, 1871
- IMMUNITY, CELL-MEDIATED
  - HUMAN, 2057
- METHYLNITROSOUREA
  - RAT, 1871
- PROTEINS, SOLUBLE
  - RAT, DETERMINATION, 1871
- SCHWANNOMA, MALIGNANT
  - CLINICAL STUDY, 2251*

- NEUROBLASTOMA
  - CHOLINERGIC CELLS
    - HUMAN, 2218*
  - TYROSINE HYDROXYLASE
    - BIOCHEMISTRY, 2391*
- N-(4-(5-NITRO-2-FURYL)-2-THIOZOLYL) ACETAMIDE
  - CARCINOGENESIS
    - MOUSE, 1891
- 4-NITROBENZENE
  - CARCINOGENESIS
    - MOUSE, 1891
- 5-NITROFURAN
  - CARCINOGENESIS
    - MOUSE, 1891
- NITROGEN OXIDE
  - AUTOMOTIVE EXHAUST
    - TUMORIGENESIS, RAT, LONG-TERM STUDY, 1827
- 4-NITROQUINOLINE-1-OXIDE
  - CARCINOGENIC ACTIVITY
    - CORRELATION WITH STRUCTURAL PROPERTIES, 1882
  - CHROMOSOME ABERRATIONS
    - CULTURED HUMAN LYMPHOCYTES, 1817
  - TERMINAL BRONCHIOLE
    - ULTRASTRUCTURE, MOUSE, 1902*
- NITROSAMINES
  - FORMATION FROM LECITHIN AND NITRITE, 1866
- NITROSAMINES, CYCLIC
  - NUCLEIC ACID
    - ALKYLATION, RAT, 1895
- N-NITROSO-N-METHYLUREA
  - MUTAGENESIS
    - MOUSE, DOMINANT-LETHAL ASSAY, 1888
- N-NITROSOBUTYLUREA
  - LEUKEMOGENESIS
    - THETA ANTIGEN, MOUSE, 2045
- NITROSOMETHYL UREA
  - TRANSPLACENTAL CARCINOGENESIS
    - EMBRYONIC LUNG TISSUES, HAMSTER, 1838
- N-NITROSOMORPHOLINE
  - MUTAGENESIS
    - MOUSE, DOMINANT-LETHAL ASSAY, 1888
- NOSE
  - HEMANGIOPERICYTOMA
    - ULTRASTRUCTURE, CASE REPORT, 2260*
- NUCLEAR MAGNETIC RESONANCE
  - CANCER DETECTION
    - MOUSE, 2199*
- NUCLEIC ACID
  - CARBON TETRACHLORIDE
    - BINDING, RAT, MOUSE, LIVER, 1894
  - METABOLISM
    - TESTES, DIETHYLSTILBESTROL, MOUSE, 1880
  - NITROSAMINES, CYCLIC
    - ALKYLATION, RAT, 1895
  - VIRUS, C-TYPE RNA
    - POLYPEPTIDE COMPOSITION, 1961
  - VIRUS, RETICULOENDOTHELIOSIS
    - POLYPEPTIDE COMPOSITION, 1961
- OCCUPATIONAL HAZARD
  - ASBESTOS
    - RESPIRATORY CANCER, CLINICAL STUDY, 2144
  - ASBESTOSIS
    - LUNG CANCER, CLINICAL STUDY, 2143
  - URINARY BLADDER TUMOR
    - SUSCEPTIBILITY, AGE FACTOR, 2154
- OOCYTE
  - INTRACISTERNAL A PARTICLES
    - PREIMPLANTATION STAGE, ULTRASTRUCTURE, MOUSE, 1965
- ORAL CONTRACEPTIVES
  - GALLBLADDER DISEASE
    - INCIDENCE, BOSTON, 2153
  - MAMMARY TUMOR
    - INCIDENCE, BOSTON, 2153
  - VENOUS THROMBOEMBOLISM
    - INCIDENCE, BOSTON, 2153
- OSTEOSARCOMA
  - TRANSFORMING ACTIVITY
    - CO-CULTIVATION, LEUKEMIC BONE MARROW CELLS, 2208*
- OVARIAN CARCINOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - ENZYME HISTOCHEMISTRY, MOUSE, 1907*
- OXYMETHOLONE
  - HALO NEVUS DEVELOPMENT
    - CASE REPORT, 1904*
- PAGET'S DISEASE
  - ADENOCARCINOMA OF BARTHOLIN'S GLAND
    - CASE REPORT, 2253*
- PANCREAS
  - ZOLLINGER-ELLISON SYNDROME
    - CALCITONIN, GASTRIN, CLINICAL STUDY, 2372*
- PAPILLOMA, SQUAMOUS
  - ANTIGEN, EPIDERMAL
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RABBIT, RAT, 2078
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - EPIDERMAL ANTIGENS, RABBIT, RAT, 2078
- PARATHYROID GLAND
  - ADENOMAS, OXYPHILIC
    - CLINICAL, BIOCHEMICAL STUDY, 2259*
- PEROXIDATION
  - CHROMOSOME BREAKAGE

- AGING, 1877

PESTICIDE TOXICITY
- PROTEIN SYNTHESIS
  - HELA CELLS, 1857
- RNA SYNTHESIS
  - HELA CELLS, 1857

PHAGOLYSOSOME
- DIGESTION OF CELLULAR FRAGMENTS
  - BASAL-CELL SKIN CARCINOMA, ULTRASTRUCTURE, 2220*

PHENANTHRENE
- FORMATION DURING HEATING OF FOODS
  - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820

PHENYLALANINE HYDROXYLASE
- HEPATOMA
  - RAT, 2274*

PHOSPHOKINASE
- ACTIVITY
  - CHROMATIN, LIVER TUMOR, RAT, 1845

PHOSPHOLIPID
- CALCIUM BINDING IN TUMOR CELLS
  - BIOCHEMICAL STUDY, 2261*
- RNA POLYMERASE IN MYELOMA CELLS
  - MOUSE, 2242*
- UPTAKE
  - EXPERIMENTAL RAT TUMORS, 2308*

PHYTOHEMAGGLUTININ
- TRANSFORMATION, LYMPHOCYTE
  - POKEWEED MITOGEN, HODGKIN'S DISEASE, 2099

PINOCYTOSIS
- POLYOMA VIRUS SYNTHESIS
  - ULTRASTRUCTURE, MOUSE EMBRYO CELLS, 2004

PITUITARY GLAND
- ADRENAL CORTEX
  - GASTRIC CANCER, CLINICAL STUDY, 2342*
- CHROMOPHOBE ADENOMA
  - BLOOD VESSEL ULTRASTRUCTURE, 2298*

PLACENTA
- BENZO(A)PYRENE METABOLISM
  - MATERNAL TOBACCO SMOKING, HUMAN, 1815
- 9,10-DIMETHYLBENZ(A)ANTHRACENE
  - FETUS, RAT, 1893
- N-METHYLANILINE METABOLISM
  - MATERNAL TOBACCO SMOKING, HUMAN, 1815
- VIRUS, C-TYPE PARTICLE
  - FETAL TISSUES, MONKEY, 1930

PLASMA CELL GRANULOMA
- LUNG
  - CASE REPORTS, 2230*
  - CLINICAL STUDY, 2252*

PLASMOCYTOMA
- MEMBRANE ENZYME ACTIVITY
  - MOUSE CELL CULTURE, 2216*

POLLUTION, AIR
- ATMOSPHERIC CARCINOGENS
  - TRANSFORMATION, EMBRYO CELLS, MOUSE, RAT, 1835
- AUTOMOTIVE EXHAUST
  - TUMORIGENESIS, RAT, LONG-TERM STUDY, 1827

POLY(ADENOSINE DIPHOSPHATE RIBOSE)
- SYNTHESIS
  - TRANSFORMED LUNG CELLS, HAMSTER, 1901*

POTASSIUM
- ION EFFECT ON EHRLICH ASCITES TUMOR
  - ELECTRICAL CHARACTERISTICS, 2245*

PROGESTERONE
- VARIATION, TISSUE CULTURE SERA
  - BOVINE, 2279*

PROSTAGLANDIN
- LEVELS
  - POLYOMA VIRUS-TRANSFORMED HAMSTER KIDNEY CELLS, 1971

PROSTAGLANDIN E2
- BONE RESORPTION-STIMULUS
  - FIBROSARCOMA, MOUSE, 2278*

PROSTATE
- ADENOMA
  - 3H-5ALPHA-DIHYDROTESTOSTERONE METABOLISM,HUMAN, 2136
  - 3H-TESTOSTERONE METABOLISM, HUMAN, 2136
- CARCINOMA
  - CLINICAL STUDY, 2145
- CARCINOMA, METASTASIS TO TESTIS, 2240*
- EMBRYONAL RHABDOMYOSARCOMA
  - ULTRASTRUCTURE, CASE REPORT, 2294*
- NEOPLASM
  - URINARY CHOLESTEROL EXCRETION, HUMAN, 2224*
- VIRUS, HERPES-LIKE
  - EPITHELIUM, HUMAN, 1992

PROTEIN
- ACIDIC CHROMATIN
  - MAMMARY TUMOR, MOUSE, RAT, 2180
- BILEAFLET NUCLEAR MEMBRANE
  - LIVER, BIOCHEMICAL STUDY, RAT, 2268*
- SARCOMAS, ADENOCARCINOMAS
  - MORPHOCYTOCHEMISTRY, 2383*
- SYNTHESIZING MACHINERY
  - RNA TUMOR VIRUS, CHICKEN, 2032*
- VIRUS, AVIAN MYELOBLASTOSIS
  - IDENTIFICATION OF LARGE PRECURSOR, CHICKEN, 2031*
- VIRUS, C-TYPE RNA
  - PURIFICATION AND CHARACTERIZATION, 1940
- VIRUS, RAUSCHER LEUKEMIA
  - PURIFICATION AND CHARACTERIZATION, 1940

- PROTEIN KINASE
  - CYTOLYSIS, RESISTANCE
    - MOUSE LYMPHOMA, 2347*
- PROTEIN, NUCLEAR
  - LIVER
    - THIOACETAMIDE-TREATED RATS, 1833
- PROTEIN SYNTHESIS
  - AFLATOXIN B1
    - LIVER, RAT, 1819
  - CYCLOHEXIMIDE
    - POLYOMA VIRUS-INFECTED MOUSE EMBRYO CELLS, 2001
  - HERPESVIRUS-HOST CELL INTERACTION
    - BIOCHEMISTRY, REVIEW, 1809
  - INHIBITION
    - AFLATOXIN B1, MONKEY KIDNEY CELL CULTURES, 1847
  - PESTICIDE TOXICITY
    - HELA CELLS, 1857
  - THIOACETAMIDE
    - LIVER NUCLEI, RAT, 1833
  - VIRUS, MURINE SARCOMA-LEUKEMIA
    - POLYRIBOSOMES, TRANSFORMED RAT CELLS, 1964
- PULMONARY CARCINOMA
  - ERYTHROCYTOSIS
    - FAMILY STUDY, 2394*
- PYRAN COPOLYMER
  - IMMUNOSUPPRESSION
    - ANTI-LYMPHOCYTE SERUM, LEUKEMIA, MOUSE, 2079
- PYRENE
  - FORMATION DURING HEATING OF FOODS
    - CONTAMINATION ON GRILLS OVER CHARCOAL, 1820
- PYRIMIDINE
  - METABOLISM
    - NORMAL AND TUMOR TISSUE, MAMMAL, 2365*
- PYRIMIDINE, HALOGENATED
  - EPSTEIN-BARR VIRUS
    - ANTIGEN COMPONENT, HUMAN LYMPHOBLASTOID CELL, 2060
- PYRUVATE KINASE
  - LIVER AND HEPATOMA
    - KINETIC STUDY, ADULT AND FETAL LIVER, 2306*
- QUINOLINES
  - CARCINOGENIC ACTIVITY
    - CORRELATION WITH STRUCTURAL PROPERTIES, 1882
- RADIATION
  - CARCINOGENESIS
    - POPULATION HETEROGENEITY HYPOTHESIS, 1912
  - CELL SURVIVAL
    - MOLECULAR THEORY, MATHEMATICAL MODEL, 1910
  - CHROMOSOME BREAKAGE, SITE
    - HUMAN LYMPHOCYTE, 1923*
  - LEUKEMIA AUTOPSY STUDY
    - ATOMIC BOMB SURVIVORS, HIROSHIMA-NAGASAKI, 1917*
  - RADON, ORAL ADMINISTRATION
    - GASTRIC MUCOSA, RAT, 1924*
  - THYROID GLAND
    - MOUSE, ADULT, 1921*
    - MOUSE, FETAL, 1922*
- RADIATION, GAMMA
  - DNA STRAND BREAK FORMATION
    - MECHANISM, 1919*
  - DNA SYNTHESIS
    - ISOPROTERENOL, MOUSE SALIVARY GLAND CELLS, 1911
- RADIATION, UV
  - SKIN TUMOR INITIATION
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE, MOUSE, 2141
- RADIATION, X-RAY
  - CHROMOSOME ABBERATIONS
    - DOSIMETRY, CELL CULTURE, 1920*
  - IMMUNIZATION, TUMOR
    - MOUSE, 2086
- RADIATION ,X-RAY
  - LYMPHOMA CELLS
    - REPAIR REPLICATION, DNA, 1914
- RADIOIMMUNOASSAY
  - VIRUS, C-TYPE
    - PRIMATE, 2055
- RECTAL CARCINOMA
  - CIRCULATING MALIGNANT CELLS
    - LONG-TERM CLINICAL STUDY, 2254*
- RENAL ADENOCARCINOMA
  - VIRUS, HERPES
    - TEMPERATURE STUDY, TUMOR CELLS, FROG, 2016*
- RENAL CARCINOMA
  - AFLATOXIN B1
    - ULTRASTRUCTURE, RAT, 1844
- RENAL CELL CARCINOMA
  - CASE REPORTS, 2234*
  - IMMUNITY, CELL-MEDIATED
    - HUMAN, 2062
- RESISTANCE
  - CYTOLYSIS, DIBUTYRYL CYCLIC AMP
    - MOUSE LYMPHOMA, 2347*
- RESPIRATORY SYSTEM
  - ASBESTOS
    - CLINICAL STUDY, 2144
- RESPIRATORY SYSTEM TUMORS
  - BENZO(A)PYRENE
    - HAMSTER, 1816
    - INDUCTION, HAMSTERS, 1837
  - N-DIBUTYLNITROSAMINE
    - X-RAY DETERMINATION, HAMSTER, 1878
  - N-DIETHYLNITROSAMINE

    - X-RAY DETERMINATION, HAMSTER, 1878
  - N-METHYL-N-NITROSOUREA
    - ULTRASTRUCTURE, HAMSTER, 1870
  - URETHANE
    - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830

- RETICULOENDOTHELIAL SYSTEM
  - ZYMOSAN TREATMENT
    - TUMOR FREQUENCY, MOUSE, 2117*

- RETICULUM CELL SARCOMA
  - VIROPEXIS OF TYPE E PARTICLES
    - MOUSE, 1978

- RETINOBLASTOMA
  - HL-A ANTIGEN
    - CLINICAL STUDY, 2128*

- REVERSE TRANSCRIPTASE
  - TRANSFORMED CELL LINES
    - HUMAN NEOPLASMS, 2228*

- RHABDOMYOSARCOMA
  - EMBRYONAL, PROSTATE
    - ULTRASTRUCTURE, CASE REPORT, 2294*

- RIBONUCLEASE
  - ACTIVITY
    - ROUS SARCOMA VIRUS-INFECTED CHICK CELLS, 1931
  - RNA-DNA HYBRID-SPECIFIC
    - THYMUS, CALF, 2181

- RNA
  - DOUBLE STRANDED
    - VIRUS INDUCED, PREPARATION, 2034*
  - EXPRESSION OF DNA SEQUENCE
    - GENE AMPLIFICATION, COW, 2182
  - HYBRIDIZATION
    - MURINE MYELOMA TUMORS, MOUSE, 2183
  - ISOLATION, DNA SYNTHESIS
    - MOUSE MYELOMA, 2305*
  - LABELING
    - HODGKIN'S DISEASE DIAGNOSIS, PROGNOSIS, 2319*
  - RIBONUCLEASE RESISTANT
    - CHARACTERISTICS, BIOSYNTHESIS, 2174
  - SARCOMAS, ADENOCARCINOMAS
    - MORPHOCYTOCHEMISTRY, 2383*
  - VIRUS, MURINE SARCOMA
    - RAT TUMOR CELL CULTURE, 1950
  - VIRUS, MURINE SARCOMA-LEUKEMIA
    - INFECTIVITY, AUTOINTERFERENCE, 1925
  - VIRUS, RETICULOENDOTHELIOSIS
    - POLYPEPTIDE COMPOSITION, 1961
  - VIRUS-SPECIFIC
    - ADENOVIRUS 2-SV40 HYBRID, 1956

- RNA, DOUBLE STRANDED
  - INHIBITOR OF PROTEIN SYNTHESIS SUBSTRATE NUCLEASE
    - KREBS II ASCITES CELLS, 2226*

- RNA, MESSENGER
  - ASCITES TUMOR
    - MOUSE, 2301*
  - HISTONE
    - IDENTIFICATION, HELA CELLS, 2207*
  - TRANSPORT
    - CYTOSOL-MEDIATED, RAT LIVER, 2172
  - VIRUS, MURINE SARCOMA-LEUKEMIA
    - POLYRIBOSOMES, TRANSFORMED RAT CELLS, 1964

- RNA METHYLASE
  - ACTIVITY
    - DIMETHYLAMINOAZOBENZENE, LIVER CELLS, RAT HAMSTER, 1850
    - ETHIONINE, LIVER CELLS, RAT HAMSTER, 1850

- RNA POLYMERASE
  - EHRLICH ASCITES TUMOR
    - PURIFICATION, 2164
  - P1 PHAGE DEVELOPMENT
    - E.COLI, 1980
  - PHENOLS
    - LIVER, RAT, 2185
  - PHOSPHOLIPIDS AND DIMETHYLSULFOXIDE IN MYELOMA CELLS
    - MOUSE, 2242*
  - SV40 DNA TRANSCRIPTION
    - MONKEY KIDNEY CELLS, 1995

- RNA, RIBOSOMAL
  - NUCLEOTIDE SEQUENCE
    - NORMAL LIVER, HEPATOMA, MOUSE, 2193*

- RNA SYNTHESIS
  - HERPESVIRUS-HOST CELL INTERACTION
    - BIOCHEMISTRY, REVIEW, 1809
  - INHIBITION BY URETHANE
    - REGENERATING LIVER, RAT, 1841
  - PARTICIPATION IN TUMORIGENESIS
    - KALANCHOE LEAVES, 2303*
  - PESTICIDE TOXICITY
    - HELA CELLS, 1857
  - VIRAL, ALTERED PATTERN
    - SINDBIS VIRUS INFECTED CELLS, 2042*
  - VIRUS, ROUS SARCOMA
    - QUANTITATIVE STUDY, CHICKEN CELLS, 1926
  - VIRUS, SENDAI
    - EHRLICH TUMOR CELLS, 1957

- RNA TRANSCRIPTION
  - ADENOVIRUS 2-SV40 HYBRID
    - CHARACTERIZATION, 1954
  - BURKITT'S LYMPHOMA
    - HUMAN CELLS, 1944

- RNA, TRANSFER
  - ACCEPTOR CAPACITY
    - RAT LIVER AND ZAJDELA HEPATOMA, 2178
  - ACTIVITY
    - ROUS SARCOMA VIRUS, CHICKEN, 2012*
  - HEPATOMA
    - FETAL RAT, 2171
  - LIVER, ZAJDELA HEPATOMA
    - RAT, 2222*

- ROSENTHAL FIBER

- ASTROCYTOMA, SPONGIOBLASTOMA
    - ULTRASTRUCTURE, HISTOLOGY, 2296*

SALIVARY GLAND
- ADENOCARCINOMA
    - ONCOGENICITY, BITTNER VIRUS, MOUSE, 1933
- ADENOID CYSTIC CARCINOMA
    - CLINICAL STUDY, 2250*
- ADENOMAS, OXYPHILIC
    - CLINICAL, BIOCHEMICAL STUDY, 2259*
- BASAL CELL ADENOMA
    - CASE REPORT, 2243*
- MYOEPITHELIOMA
    - ULTRASTRUCTURE, CASE REPORT, 2283*
- SCLEROSING INFLAMMATION, KUETTNER TUMOR
    - ETIOLOGY, 2229*

SARCOMA
- INDUCTION, FOREIGN BODY
    - INTRACISTERNAL TYPE A PARTICLES, MOUSE, 1981
- METHYLCHOLANTHRENE
    - IMMUNOGENICITY, MOUSE, 1832
- NUCLEOPROTEINS, GLYCOGEN
    - MORPHOCYTOCHEMISTRY, 2383*

SARCOMA, SUBCUTANEOUS
- LIPID ANTIOXIDANT ACTIVITY
    - EXPERIMENTAL CARCINOGENESIS, MOUSE, 2160

SCHIZOPHRENIA
- CANCER
    - REVIEW, 1812*

SCHWANNOMA, MALIGNANT
- NERVOUS SYSTEM TUMORS
    - CLINICAL STUDY, 2251*

SERUM
- ACIDIFIED
    - DIFFERENTIAL TOXICITY TO TUMOR CELLS, 2011*
- ANTIGEN, CARCINOEMBRYONIC
    - RADIOIMMUNOELECTROPHORETIC BINDING ASSAY, 2083
- COMPLEMENT LEVELS
    - HODGKIN'S DISEASE PATIENTS, 2106
- DNA POLYMERASE ACTIVITY
    - AUSTRALIA ANTIGEN, HUMAN, 1983
- MURAMIDASE LEVELS
    - ACUTE LEUKEMIA, CLINICAL STUDY, 2360*

SERUM, ANTI-LYMPHOCYTE
- LEUKEMIA
    - PYRAN COPOLYMER, MOUSE, 2079

SERUM, IMMUNE
- IMMUNE RESPONSE
    - TUMOR GROWTH, CONTROL, MOUSE, 2063

SIALIDASE
- ACTIVITY
    - TRANSFORMED HAMSTER CELLS, 1939

SKIN
- BASAL CELL CARCINOMA
    - PHAGOLYSOSOME DIGESTION OF CELLULAR FRAGMENTS, 2220*
- CHRONIC PROGRESSIVE KERATOATROPHODERMA
    - CASE REPORTS, 2361*
- CLEAR CELL ACANTHOMA
    - CASE REPORT, 2379*
- EPIDERMODYSPLASIA VERRUCIFORMIS
    - WART-LIKE VIRUS, CASE REPORTS, 1999
- NEVOID BASAL CELL EPITHELIOMA
    - ULTRASTRUCTURE, CASE REPORTS, 2231*
- TUMOR DISTRIBUTION
    - DOMESTIC ANIMALS, 2288*

SKIN TUMOR
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - UV RADIATION, MOUSE, 2141

SKULL
- MENINGEOMA
    - CASE REPORT, 2158*

SODIUM CYCLAMATE
- CHROMOSOME ABERRATIONS
    - ANTIOXIDANT PROTECTION, 1877

SPLEEN
- CYTOTOXIC FACTOR
    - MYELOCYTIC LEUKEMIA, HYPERSPLENISM, TRAUMA PATIENTS, 2325*

SPLENECTOMY
- EFFECT ON SARCOMA GROWTH
    - SYNGENEIC TRANSPLANTATION, MICE, 2075

SPLENOMEGALY
- VIRUS, RAUSCHER LEUKEMIA
    - ANEMIA DEVELOPMENT, MOUSE, 1990

SPONGIOBLASTOMA
- ASTROCYTOMA, ROSENTHAL FIBERS
    - ULTRASTRUCTURE, HISTOLOGY, 2296*

SQUAMOUS CELL CARCINOMA
- ANTIGEN, EPIDERMAL
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RABBIT, RAT, 2078
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
    - . EPIDERMAL ANTIGENS, RABBIT, RAT, 2078
    - MOUSE, 1824
- N-METHYL-N-NITROSOUREA
    - LUNG, HAMSTER, 1870

SQUAMOUS DYSPLASIA
- CERVICAL CARCINOMA
    - EPIDEMIOLOGY, 2156

STILBESTROL
- HEPATOCARCINOGENESIS
    - HAMSTER, RAT, MOUSE, REVIEW, 1802

STOMACH
- ARGYROPHYL CELL CARCINOID
    - CASE REPORT, 2233*

- CANCER
  - ELECTROGASTROGRAM, 2378*
- CANCER, LACTATE DEHYDROGENASE ACTIVITY
  - CLINICAL STUDY, 2276*
- CARCINOMA SITE DETERMINATION FROM BODY FLUID
  - CLINICAL STUDY, 2258*
- GASTRIC CARCINOMA
  - PITUITARY GLAND-ADRENAL CORTEX SYSTEM, 2342*
  - SURFACTANT EFFECT, RAT, 1886
- GASTRIC MUCOSA
  - CELL ALTERATIONS, MALIGNANT TUMORS, HUMAN, 2140
- GLOMIC TUMORS
  - CASE REPORTS, 2368*
- TRANSPLANTATION OF VX2 CARCINOMA
  - RABBIT, 2241*

SULFUR
- EPIDERMIS
  - GROWTH KINETICS, MOUSE, 1848

TENDON SHEATH
- GIANT CELL TUMOR, MALIGNANT
  - ULTRASTRUCTURE, CASE REPORT, 2264*

TERATOMA
- TESTIS
  - CONGENITAL, 2265*

TEREPHTHALIC ACID
- MAMMARY TUMOR INHIBITION
  - MOUSE, 2225*

TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE ACTIVITY
- ENZYME
  - CROWN GALL TUMOR, TOBACCO, 2192*

TESTIS
- CANCER INCIDENCE
  - FINLAND, REVIEW, 1811*
- ESTROGEN DEPENDENT CARCINOMA
  - ANDROGEN ACTIVITY, RAT, 2324*
- INTERSTITIAL-CELL TUMOR
  - STEROID METABOLISM, RAT, 2323*
- LACTATE DEHYDROGENASE ISOENZYMES
  - TESTICULAR INTERSTITIAL TUMOR GRAFTS, RAT, 2335*
- MESTASTASIS FROM PROSTATE CARCINOMA, 2240*
- NEOPLASM
  - URINARY CHOLESTEROL EXCRETION, HUMAN, 2224*
- TERATOMA
  - CONGENITAL, 2265*

TESTOSTERONE
- VARIATION, TISSUE CULTURE SERA
  - BOVINE, 2279*

TETRAHYDROCANNABINOL
- BENZPYRENE HYDROXYLASE ACTIVITY
  - LIVER, LUNG, RAT, 1814

THECA GRANULOSA CELL TUMOR
- ADRENAL GLAND
  - CASE REPORT, 2315*

THIOACETAMIDE
- PROTEIN SYNTHESIS
  - LIVER NUCLEI, RAT, 1833

THYMIDINE
- TRANSPORT AND INCORPORATION INTO DNA
  - NOVIKOFF HEPATOMA CELLS, RAT, 2267*

THYMIDINE KINASE
- ACTIVITY
  - AVIAN SARCOMA VIRUS TRANSFORMED CELL, HAMSTER, 2021*
  - RAT TUMOR AND GROWING TISSUE, 2304*
- VIRUS, HERPES SIMPLEX
  - ACTIVATION AND SUPPRESSION OF THE VIRAL ENZYME, MOUSE CELLS, 1945

THYMOMA
- FAMILIAL OCCURRENCE
  - CASE REPORT, 2244*

THYROID
- ADRENAL CORTICAL CHANGE
  - METASTASIS FORMATION, RABBIT, 2227*
- MEDULLARY CARCINOMA
  - CALCITONIN, GASTRIN, CLINICAL STUDY, 2372*
  - GRANULES AND AMYLOID, CALCITONIN DISTRIBUTION, CASE REPORTS, 2293*
- TUMOR
  - MICROSOMAL ANTIGEN DISTRIBUTION, 2111*

THYROID CARCINOMA
- CLASSIFICATION
  - CLINICAL STUDY, 2350*
- ETHYLENETHIOUREA
  - RAT, 1864

THYROID GLAND
- ADENOMAS, OXYPHILIC
  - CLINICAL, BIOCHEMICAL STUDY, 2259*
- HEMANGIOPERICYTOMA
  - CASE REPORT, 2398*
- MEDULLARY CARCINOMA
  - ULTRASTRUCTURE, 2353*
- RADIATION
  - MOUSE, ADULT, 1921*
  - MOUSE, FETAL, 1922*

TOBACCO
- CIGARETTE SMOKE
  - CARCINOGENIC PROPERTIES, 1873
- CIGARETTES
  - LUNG CANCER, HUMAN, AGE FACTOR, 1873
- CROWN GALL TUMOR
  - DNA POLYMERASE ACTIVITY, 2192*

TOBACCO SMOKE CONDENSATE
- URINARY BLADDER TUMOR
  - RAT, 1874

TOBACCO SMOKING
- CARBOXYHEMOGLOBIN LEVELS

- HUMAN, 1842
- CHOLESTEROL LEVELS
  - HUMAN, 1842
- LUNG CANCER
  - CLINICAL STUDY, 1899*
  - REVIEW, 1810*

TOBACCO SMOKING, MATERNAL
- BENZO(A)PYRENE METABOLISM
  - HUMAN FETAL LIVER, PLACENTA, 1815
- N-METHYLANILINE METABOLISM
  - HUMAN FETAL LIVER, PLACENTA, 1815

TRANSFORMATION
- CARCINOGEN, ATMOSPHERIC
  - EMBRYO CELLS, MOUSE, RAT, 1835
- GANGLIOSIDE PATTERNS
  - HAMSTER CELLS, 1939
- LYMPHOCYTE
  - IMMUNE RESPONSE, CELL-MEDIATED, MOUSE, 2056
  - PHYTOHEMAGGLUTININ, POKEWEED MITOGEN, HODGKIN'S DISEASE, 2099
  - POST-SURGERY, CANCER PATIENTS, 2112*
  - REGIONAL NODE CELLS, 2188*
  - SCOPE AND LIMITATIONS OF PLASMA FREE MEDIUM, 2318*
- NUCLEIC ACID HYPERMETHYLATION
  - E.COLI, 2025*
- SIALIC ACID CONTENT
  - HAMSTER CELLS, 1939
- SIALIDASE ACTIVITY
  - HAMSTER CELLS, 1939
- SV40 TRANSFORMED HAMSTER FIBROBLASTS
  - INCORPORATION OF 2-DEOXY-D-GLUCOSE INTO GLYCOLIPIDS, 2371*
- VIRUS, FELINE LEUKEMIA
  - AMNION CELL, HUMAN, 1929
- VIRUS, HERPES HOMINIS 2
  - HUMAN EMBRYONIC LUNG CELLS, 1959
- VIRUS, HERPES-LIKE
  - HAMSTER EMBRYO CELLS, 1989
- VIRUS, MOLONEY SARCOMA
  - AMNION CELL, HUMAN, 1929
  - CONTACT INHIBITION OF MOVEMENT, MOUSE CELLS, 1966
- VIRUS, MURINE SARCOMA
  - 5'-BROMODEOXYURIDINE-INDUCED CELL MUTANTS, MOUSE, 1935
- VIRUS, POLYOMA
  - PROSTAGLANDIN LEVELS, HAMSTER KIDNEY CELLS, 1971
- VIRUS, RD-114
  - AMNION CELL, HUMAN, 1929
- VIRUS, ROUS SARCOMA
  - HEXOSE TRANSPORT, CHICK EMBRYO CELLS, 1979
- VIRUS, SV40
  - TEMPERATURE-SENSITIVE MUTANT, MOUSE CELLS, 1967

TRANSFORMATION, ACUTE
- LEUKEMIA, CHRONIC MYELOGENOUS
  - MONOCLONAL, HUMAN, 2137

TRANSFORMATION, MALIGNANT
- LEUKOPLAKIA
  - HUMAN, HERPES VIRUS HOMINIS ROLE, 2051
- 4-NITROQUINOLINE-1-OXIDE
  - CULTURED HUMAN LYMPHOCYTES, 1817

TRANSPLACENTAL CARCINOGENESIS
- DIETHYLNITROSAMINE
  - EMBRYONIC LUNG TISSUES, HAMSTER, 1838
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - EMBRYONIC LUNG TISSUES, HAMSTER, 1838
- 9,10-DIMETHYLBENZ(A)ANTHRACENE
  - MOUSE, 1858
- DIMETHYLNITROSAMINE
  - EPITHELIUM, MOUSE, 1851
- NITROSOMETHYL UREA
  - EMBRYONIC LUNG TISSUES, HAMSTER, 1838
- URETHANE
  - EMBRYONAL LUNG TISSUE, 1908*
  - MOUSE, 1830

TRANSPLANTATION
- ANTIGEN
  - IMMUNE RESPONSE, MAMMARY TUMOR, MOUSE, 2084
  - IMMUNE RESPONSE, MURINE SARCOMA TUMOR CELLS, 2080
  - IMMUNE RESPONSE, MURINE TUMORS, RAT, 2066
  - ROUS SARCOMA, RAT, 2072
  - TUMOR CELL, MOUSE, 2103
- TOLERANCE
  - T-LYMPHOCYTE, RAT, 2100
- TRANSFORMED CELLS, INDUCED CHANGES
  - CONCANAVALIN A, 2120*
- VX2 STOMACH CARCINOMA
  - RABBIT, 2241*

TRIAZENES
- MUTAGEN, INDIRECT
  - GENETIC TEST SYSTEM STUDIES, 1889

TUMOR
- ALPHA-AMANITIN INHIBITION OF INDUCTION
  - KALANCHOE LEAVES, 2303*
- GLYCERYL ETHERS
  - MOUSE, INSECT, 2194*
- MICROANGIOPATHIC HEMOLYTIC ANEMIA
  - HYPERCALCEMIA, RAT, 2397*

TUMOR, POLYOMA
- IMMUNITY
  - RAT, 2053

TUMOR VIRUS
- SEE VIRUS

TUMORIGENESIS
- IMMUNITY, CELL-MEDIATED TUMOR-SPECIFIC
  - ROUS SARCOMA VIRUS, RAT, 2072
- LYMPHOCYTES, AUTOSENSITIZED
  - MOUSE, 2052
- TUMOR CELL-CONTAINING DIFFUSION CHAMBER
  - INTRAPERITONEAL, RAT, 2336*

TYROSINE HYDROXYLASE
- NEUROBLASTOMA, ADRENAL GLAND

        - BIOCHEMISTRY, 2391*

- URETHANE
    - ADENOMATA
        - LUNG, MOUSE, 2088
    - HEPATOCARCINOGENESIS
        - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830
    - RESPIRATORY SYSTEM TUMORS
        - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830
    - RNA SYNTHESIS INHIBITION
        - REGENERATING LIVER, RAT, 1841
    - TRANSPLACENTAL CARCINOGENESIS
        - EMBRYONAL LUNG TISSUE, 1908*
        - MOUSE, 1830
    - UROGENITAL TRACT CARCINOMA
        - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830

- URINARY BLADDER CARCINOMA
    - BILHARZIOSIS
        - CLINICAL STUDY, EGYPT, 2236*
    - LYMPHOCYTE CYTOTOXICITY
        - THYMUS-DERIVED EFFECTOR CELLS, 2073

- URINARY BLADDER TUMOR
    - 4-AMINOBIPHENYL
        - DOG, 1855
    - APPEARANCE, BONE AND CARTILAGE, 2239*
    - N-BUTYL-N-(4-HYDROXYBUTYL)NITROSAMINE
        - METABOLITE CONCENTRATION, URINE, 1884
        - RAT, AGE FACTOR, 1884
        - RAT, GUINEA PIG, HAMSTER, 1884
    - OCCUPATIONAL HAZARD
        - SUSCEPTIBILITY, AGE FACTOR, 2154
    - RNA VIRUS ISOLATION
        - HUMAN, 1937
    - TOBACCO SMOKE CONDENSATE
        - RAT, 1874
    - URINE AND CALCULUS FORMATION
        - RAT, 1853

- UROGENITAL TRACT CARCINOMA
    - URETHANE
        - TRANSPLACENTAL CARCINOGENESIS, MOUSE, 1830

- UROGENITAL TUMOR
    - ARGINASE
        - BIOCHEMISTRY, 2396*

- UTERUS
    - ADENOSQUAMOUS CARCINOMA, ENDOMETRIUM
        - ULTRASTRUCTURE, HUMAN, 2290*

- VAGINA
    - MESONEPHROMA IN OFFSPRING
        - MATERNAL STILBESTROL
            - ADMINISTRATION, CASE REPORTS, 1860

- VENOUS THROMBOEMBOLISM
    - ORAL CONTRACEPTIVES
        - INCIDENCE, BOSTON, 2153

- VIRUS
    - ADENOVIRUS 2-SV40 HYBRID
        - BIOLOGICAL PROPERTIES, 1956
        - DNA, BIOPHYSICAL PROPERTIES, 1973
        - RNA TRANSCRIPTION, 1954
    - DNA REPLICATION
        - BACTERIOPHAGE T7 INFECTED CELLS, 2041*
    - HOST RANGE CONVERSION
        - MURINE SARCOMA-LEUKEMIA COMPLEX, 2118*
    - INTRACISTERNAL A PARTICLES
        - OOCYTE, PREIMPLANTATION STAGE, ULTRASTRUCTURE, MOUSE, 1965
    - INTRACISTERNAL TYPE A PARTICLES
        - FOREIGN BODY-INDUCED SARCOMAS, MOUSE, 1981
    - P1 PHAGE DEVELOPMENT
        - RNA POLYMERASE, E.COLI, 1980
    - WART-LIKE
        - SKIN CANCER, CASE REPORTS, 1999

- VIRUS, ABELSON
    - ANEMIA
        - RAT, 1934
    - LEUKEMIA
        - RAT, 1934

- VIRUS, ADENO
    - ISOLATION
        - KIDNEY, FROG, 1953
    - TUMORIGENESIS
        - ADENO-ASSOCIATED SATELLITE VIRUS, HAMSTER, 1928

- VIRUS, ADENO 12
    - INFECTION
        - DNA SYNTHESIS, RAT EMBRYO FIBROBLASTS, 2002
        - MITOTIC ACTIVITY, RAT EMBRYO FIBROBLASTS, 2002

- VIRUS, ADENO-ASSOCIATED SATELLITE
    - ADENOVIRUS-INDUCED TUMORS
        - HAMSTER, 1928

- VIRUS, AVIAN
    - GENETIC INTERACTIONS
        - CHICK EMBRYO CELLS, 1994

- VIRUS, AVIAN LEUKOSIS-SARCOMA
    - VIRUS, RETICULOENDOTHELIOSIS
        - COMPARISON, CHARACTERIZATION, 1958

- VIRUS, AVIAN MYELOBLASTOSIS
    - DNA POLYMERASE INHIBITION
        - POLY(U) FRACTIONS, 1963
    - PROTEIN
        - IDENTIFICATION OF LARGE PRECURSOR, CHICKEN, 2031*
    - RELEASE
        - LEUKEMIC MYELOBLAST COLONY, 2035*

- VIRUS, AVIAN SARCOMA
    - TEMPERATURE-SENSITIVE
        - SIX MUTANT CATEGORIES, CHICKEN, 2018*
    - TRANSFORMED CELLS
        - BROMODEOXYURIDINE RESISTANCE, THYMIDINE KINASE ACTIVITY,

    - HAMSTE. 2021*

- VIRUS, BITTNER
  - SALIVARY GLAND ADENOCARCINOMA
    - ONCOGENICITY, MOUSE, 1933

- VIRUS, BOVINE ADENO 3
  - DNA SYNTHESIS INDUCTION
    - CONTACT-INHIBITED CELLS, HAMSTER, 2037*

- VIRUS, C-TYPE
  - ANTIGEN, GROUP-SPECIFIC
    - INDIRECT IMMUNOPEROXIDASE STAINING, 2069
  - ANTIGEN, SPECIES-SPECIFIC
    - PRIMATE, 2055
  - CELL-SURFACE ANTIGENIC PROPERTIES
    - TRANSFORMED MOUSE EMBRYO CELLS, 1982
  - CHARACTERIZATION
    - GUINEA PIG, 2000
  - ENVELOPE ANTIGENIC PROPERTIES
    - TRANSFORMED MOUSE EMBRYO CELLS, 1982
  - GENOME
    - TRANSFORMED MOUSE EMBRYO CELLS, 1982
  - LEUKEMIA
    - GUINEA PIG, 1989, 2000
  - MORPHOLOGY
    - GUINEA PIG, 2000
  - PRODUCTION
    - TRANSFORMED MOUSE EMBRYO CELLS, 1982
  - RADIOIMMUNOASSAY
    - PRIMATE, 2055
  - RNA-DEPENDENT DNA POLYMERASE
    - HYPERIMMUNE GUINEA PIG ANTISERUM, 1984

- VIRUS, C-TYPE PARTICLE
  - PLACENTA
    - FETAL TISSUES, MONKEY, 1930

- VIRUS, C-TYPE RNA
  - ACTIVATION, GENETIC LOCI
    - MOUSE EMBRYO CELLS, 1968
  - ANTIGEN
    - COMMON TO MURINE AND FELINE VIRUSES, 2065
  - ANTIGEN, INTERSPECIES
    - PURIFICATION AND CHARACTERIZATION, 1940
  - NUCLEIC ACID
    - POLYPEPTIDE COMPOSITION, 1961
  - PROTEIN
    - PURIFICATION AND CHARACTERIZATION, 1940

- VIRUS, CYTOMEGALO-
  - ANTIBODY
    - SERUM, HUMAN SOLID TUMOR DONOR, 1947

- VIRUS, CYTOMEGALO
  - CONVERSION OF NONPERMISSIVE CELLS
    - 5-IODO-2'-DEOXYRUIDINE, HUMAN, 1998

- VIRUS, EPSTEIN-BARR
  - ANTIBODY
    - SERUM, HUMAN SOLID TUMOR DONOR, 1947
  - ANTIGEN COMPONENT
    - HALOGENATED PYRIMIDINE, HUMAN LYPHOBLASTOID CELLS, 2060
  - LYMPHOBLAST
    - HALOGENATED PYRIMIDINE, 2060

- VIRUS, ERYTHROBLASTOSIS
  - THYMIC LYMPHOMA INOCULATION
    - MOUSE, RAT, 2008*

- VIRUS, FELINE LEUKEMIA
  - HORIZONTAL TRANSMISSION
    - CAT, 1938
  - TRANSFORMATION
    - AMNION CELL, HUMAN, 1929

- VIRUS, FRIEND LEUKEMIA
  - INFECTION
    - COENZYME Q LEVELS, MOUSE, 1996
  - TUMORIGENESIS
    - RAT, 2104

- VIRUS, GAZDAR MURINE SARCOMA
  - ANTIGEN
    - HAMSTER TUMOR CELL, 2061

- VIRUS, HERPES
  - DNA
    - CARCINOGENESIS, REVIEW, 1805
  - FAMILIAL CHARACTERISTICS
    - REVIEW, 2039*
  - GENITAL INFECTIONS
    - CLINICAL STUDY, 2014*
  - HOST CELL-VIRUS INTERACTION
    - BIOCHEMISTRY, REVIEW, 1809
  - RENAL ADENOCARCINOMA
    - TEMPERATURE STUDY, TUMOR CELLS, FROG, 2016*

- VIRUS, HERPES 2
  - ANTIGEN, TUMOR-SPECIFIC
    - CERVICAL CARCINOMA, HUMAN, 2003
  - CERVICAL CARCINOMA
    - HUMAN, 1949
    - SEROEPIDEMIOLOGICAL STUDIES, 2149
  - INDUCED CERVICAL INFECTION AND MALIGNANCY
    - MONKEY, 2015*

- VIRUS, HERPES HOMINIS
  - IMMUNITY, CELL-MEDIATED
    - CANCER PATIENTS, HUMAN, 2051

- VIRUS, HERPES HOMINIS 2
  - ABORTIVE INFECTION
    - HUMAN EMBRYONIC LUNG CELLS, 1959
  - TRANSFORMATION
    - HUMAN EMBRYONIC LUNG CELLS, 1959

- VIRUS, HERPES-LIKE
  - LEUKEMIA
    - GUINEA PIG, 1989
  - LYMPHOMA
    - RABBIT, 2017*
  - PROSTATE

    - EPITHELIUM, HUMAN, 1992
  - TRANSFORMATION
    - HAMSTER EMBRYO CELLS, 1989

- VIRUS, HERPES MICROTUS
  - ISOLATION
    - MOUSE, 2036*

- VIRUS, HERPES SAIMIRI
  - ANTIBODY
    - PRIMATE, 2064
  - ANTIGEN
    - ANTIBODY RESPONSE, PRIMATE, 2064
  - INFECTION
    - ANTIBODY RESPONSE, 2064
  - LYMPHOMA INDUCTION
    - MONKEY, 1943

- VIRUS, HERPES SIMPLEX
  - CERVICAL CARCINOMA
    - ETIOLOGY, HUMAN, 1951
  - 2-DEOXY-D-GLUCOSE
    - REPLICATION, 2027*
  - GROWTH
    - TRANSFORMED GLIAL AND NEURONAL CELLS, ULTRAS, 2038*
  - TEMPERATURE SENSITIVE MUTANTS
    - ISOLATION, 2028*
  - THYMIDINE KINASE
    - ACTIVATION AND SUPPRESSION OF THE VIRAL ENZYME, MOUSE CELLS, 1945

- VIRUS, HERPES SIMPLEX 2
  - ANTIBODY
    - CERVICAL CARCINOMA PATIENTS, 2127*

- VIRUS, MAREK'S DISEASE
  - ANTIBODY
    - IMMUNOSUPPRESSION, 2049
  - MEMBRANE ANTIGEN
    - ANTIBODY, MOUSE, CHICKEN, 2125*

- VIRUS, MOLONEY LEUKEMIA
  - LEUKEMIA
    - SEROCHEMOTHERAPY, MOUSE, 1988
  - TEMPERATURE-SENSITIVE MUTANTS
    - SELECTIVE ISOLATION, MOUSE, 2020*

- VIRUS, MOLONEY SARCOMA
  - TRANSFORMATION
    - AMNION CELL, HUMAN, 1929
    - CONTACT INHIBITION OF MOVEMENT, MOUSE CELLS, 1966

- VIRUS, MURINE LEUKEMIA
  - ACTIVATION, GENETIC LOCI
    - MOUSE EMBRYO CELLS, 1968
  - ANTIGEN
    - CHROMOSOME, MOUSE, 2059
    - TUMOR TRANSPLANT, VIRUS-TOLERANT RAT, 2066
  - ANTIGEN, CELL-SURFACE
    - RAT, 2090
  - 3H-THYMIDINE UPTAKE
    - LYMPHOID CELLS, MOUSE, 1974
  - VIRUS-SPECIFIC DNA
    - PRESENCE IN NORMAL CELLS, MOUSE, 2043*

- VIRUS, MURINE SARCOMA
  - IMMUNE RESPONSE
    - CELL-MEDIATED, MOUSE, 2056
  - RNA SUBUNIT COMPOSITION
    - RAT TUMOR CELL CULTURE, 1950
  - TRANSFORMATION
    - 5'-BROMODEOXYURIDINE-INDUCED CELL MUTANTS, MOUSE, 1935

- VIRUS, MURINE SARCOMA-LEUKEMIA
  - ANEMIA
    - RAT, 1934
  - NASCENT POLYPEPTIDES
    - POLYRIBOSOMES, TRANSFORMED RAT CELLS, 1964
  - PROTEIN SYNTHESIS
    - POLYRIBOSOMES, TRANSFORMED RAT CELLS, 1964
  - RNA
    - INFECTIVITY, AUTOINTERFERENCE, 1925
  - RNA, MESSENGER
    - POLYRIBOSOMES, TRANSFORMED RAT CELLS, 1964

- VIRUS, MURINE SATCOMA
  - TRANSPLANTATION ANTIGENS
    - IMMUNE RESPONSE, MOUSE, 2080

- VIRUS, ONCORNA
  - ISOLATION
    - HUMAN CANCER CELL LINE, 1952
  - MILK
    - PRODUCTION OF CORES, HUMAN, 1972

- VIRUS, PAPILLOMA
  - DNA
    - CARCINOGENESIS, REVIEW, 1805

- VIRUS, PICORNA
  - RNA, DOUBLE STRANDED
    - PREPARATION, 2034*

- VIRUS, POLYOMA
  - ANTIGEN, SURFACE
    - HAMSTER CELLS, 2107
  - CELL PROLIFERATION INHIBITED IN NONTRANSFORMED CELLS
    - RAT, 2030*
  - DNA
    - CARCINOGENESIS, REVIEW, 1805
    - PROTEIN AND MESSENGER RNA REQUIREMENTS FOR SUPERHELICITY, 2001
  - DNA SYNTHESIS
    - MOUSE, 1927
  - DNA TRANSCRIPTION
    - KIDNEY CELL, MOUSE, 2005
  - INFECTION
    - DNA SYNTHESIS, CYCLOHEXMIDE, MOUSE EMBRYO CELLS, 2001
    - PROTEIN SYNTHESIS, CYCLOHEXIMIDE, MOUSE EMBRYO CELLS, 2001
  - SYNTHESIS
    - ULTRASTRUCTURE, MOUSE EMBRYO CELLS, 2004
  - TRANSFORMATION
    - PROSTAGLANDIN LEVELS, HAMSTER KIDNEY CELLS, 1971

- TRANSFORMED CELL SURFACES
  - LECTIN BINDING AND AGGLUTINATION. REVIEW. 1808

VIRUS. PRIMATE SARCOMA
- ISOLATION AND CHARACTERIZATION
  - MECHANISM OF RESCUE. 1969

VIRUS. RABIES
- SERUM FACTOR
  - ROUS SARCOMA VIRUS INHIBITION. CHICKS. 2009*

VIRUS. RADIATION LEUKEMIA
- ANTISERUM
  - LYMPHOMA INHIBITION. MOUSE. 1913
- LYMPHOMA
  - MOUSE. 1913

VIRUS. RAUSCHER LEUKEMIA
- ANTIGEN. INTERSPECIES
  - PURIFICATION AND CHARACTERIZATION. 1940
- INFECTION
  - SPLEEN CELLS. MOUSE. 1990
- INTERFERON INDUCTION
  - MOUSE. 1977
- PROTEIN
  - PURIFICATION AND CHARACTERIZATION. 1940
- SPLENOMEGALY
  - ANEMIA DEVELOPMENT. MOUSE. 1990
- STEM CELLS IN PERIPHERAL BLOOD
  - MOUSE. 2029*

VIRUS. RD-114
- RNA-DEPENDENT DNA POLYMERASE
  - HYPERIMMUNE GUINEA PIG ANTISERUM. 1984
- TRANSFORMATION
  - AMNION CELL. HUMAN. 1929

VIRUS. RETICULOENDOTHELIOSIS
- NUCLEIC ACID
  - POLYPEPTIDE COMPOSITION. 1961
- RNA
  - POLYPEPTIDE COMPOSITION. 1961
- VIRUS. AVIAN LEUKOSIS-SARCOMA
  - COMPARISON. CHARACTERIZATION. 1958

VIRUS. RNA
- ISOLATION
  - URINARY BLADDER TUMOR. HUMAN. 1937
- MAMMARY TUMOR
  - HUMAN. MOUSE. 1991

VIRUS. RNA TUMOR
- BASE SPECIFIC METHYLASE ACTIVITY
  - E.COLI. 2025*
- GENETIC INTERACTIONS
  - CHICK EMBRYO CELLS. 1994
- PROTEIN SYNTHESIZING MACHINERY
  - CHICKEN. 2032*

VIRUS. ROUS SARCOMA
- ADENYLATE CYCLASE ACTIVITY
  - TRANSFORMED CHICK CELLS. 1976
- BRAIN TUMOR INDUCTION
  - CLASSIFICATION. RAT. 1986
- HIGH ONCOGENICITY VARIANTS
  - IMMUNOLOGICAL STUDIES. MOUSE. 2105
- IMMUNITY
  - AGE FACTOR. RAT. INOCULATION. 2072
- INHIBITION
  - RABIES-INDUCED SERUM FACTOR. CHICKS. 2009*
- PRODUCTION AND PURIFICATION
  - CHICK EMBRYO FIBROBLASTS. 1936
- RIBONUCLEASE ACTIVITY
  - TRANSFORMED CHICK CELLS. 1931
- RNA SYNTHESIS
  - QUANTITATIVE STUDY. CHICKEN CELLS. 1926
- TEMPERATURE-SENSITIVE MUTANT
  - DEFECTIVE FOR REPLICATION. CHICK CELLS. 1957
- TEMPERATURE SENSITIVE MUTANT
  - SELECTIVE ISOLATION. CHICK EMBRYO. 2024*
- TRANSFER RNA ACTIVITY
  - CHICKEN. 2012*
- TRANSFORMATION
  - HEXOSE TRANSPORT. CHICK EMBRYO CELLS. 1979
- TUMORIGENESIS
  - RAT. 2072

VIRUS. SENDAI
- -FUSED MULTINUCLEATED TUMOR CELLS
  - ACTIVE IMMUNITY INDUCTION. MOUSE. 2047
- RNA SYNTHESIS
  - EHRLICH TUMOR CELLS. 1997

VIRUS. SHOPE PAPILLOMA
- ARGINASE ACTIVITY
  - ARGININEMIC PATIENT. 2023*

VIRUS. SINBIS
- RNA SYNTHESIS
  - ALTERED PATTERN. 2042*

VIRUS. SINDBIS
- DEFECTIVE-INTERFERING PARTICLES
  - CHICK EMBRYO. 2040*

VIRUS. SV40
- AMINO ACID COMPOSITION. 2013*
- ANTIGEN INDUCTION
  - ANTINOMYCIN D. 2124*
- CYCLIC AMP
  - MOUSE CELLS. 1932
- CYTOCHROME
  - TRANSFORMED AND NORMAL CELLS. COMPARATIVE STUDY. 1975
- DNA
  - CARCINOGENESIS. REVIEW. 1805
  - SUPERCOILING WITHOUT REPLICATION. MONKEY KIDNEY CELLS. 2033*
- DNA CLEAVAGE BY RESTRICTION ENDONUCLEASE
  - HEMOPHILUS PARAINFLUENZAE. 2019*
- DNA REPLICATION
  - KIDNEY CELLS. MONKEY. 1970
- DNA SYNTHESIS INHIBITION
  - 5-FLUORODEOXYURIDINE. MONKEY CELL CULTURES. 1960
- DNA TRANSCRIPTION

    - KIDNEY CELLS, MONKEY, 1970
    - RNA POLYMERASE, MONKEY KIDNEY CELLS, 1995
  - HYBRID REVERSION
    - TRANSFORMED HAMSTER AND MOUSE CELLS, 1955
  - INFECTION
    - RNA SYNTHESIS, MONKEY CELLS, 1970
  - RESCUE OF GENOME
    - MONKEY, 2026*
  - TEMPERATURE SENSITIVE MUTANT
    - DNA SYNTHESIS, GENETIC EXPRESSION, 1970
  - TEMPERATURE-SENSITIVE MUTANT
    - TRANSFORMED MOUSE CELLS, 1967
  - TRANSFORMED CELL MEMBRANE
    - LECTIN-BINDING SITES, 1946
  - TRANSFORMED CELL SURFACES
    - LECTIN BINDING AND AGGLUTINATION, REVIEW, 1808
  - TRANSFORMED MOUSE CELLS
    - CYTOCHALASIN B, CYTOPLASMIC CLEAVAGE INHIBITION, 2010*

- VIRUS, WOUND TUMOR
  - DEPRECIATION OF SPECIFIC INFECTIVITY
    - CLOVER, 2022*

- WARBURG THEORY
  - CARCINOGENESIS
    - REVIEW, 1807

- WARFARIN
  - TUMOR METASTASIS INHIBITION
    - MOUSE, 2138

- XANTHOMA
  - RETROPERITONEAL XANTHOGRANULOMA
    - CASE REPORTS, 2291*
  - XANTHOSARCOMA
    - CASE REPORTS, 2291*

- XANTHOSARCOMA
  - XANTHOMA
    - CASE REPORTS, 2291*

- YOLK SAC
  - CARCINOMA INDUCED
    - MOUSE, 2281*

LIBRARY U. OF I. URBANA-CHAMPAIGN

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list

DHEW Publications No. (NIH) 72-301

405

Vet Med

MAY 1973
Abstract & Citation Nos. 2401-3000

VOL. 11
NO. 5

THE LIBRARY OF THE
FEB [illegible] 1974
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institutes of Health

LIBRARY U. OF I. URBANA-[illegible]

Volume XI, No. 5
May

Abstract and Citation Nos. 2401 - 3000

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

THE LIBRARY OF THE
FEB 27 1974
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Editor
Robert Love, M.D.
Jefferson Medical College, Philadelphia

Associate Editor
George P. Studzinski, M.D.
Jefferson Medical College, Philadelphia

NCI Staff Consultants
Elizabeth Weisburger, Ph.D.
Sidney Siegel, Ph.D.

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

LIBRARY U. OF I. URBANA-CHAMPAIGN

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Etiology Area
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication
approved by the Director of the Bureau of
the Budget on July 25, 1967.

LIBRARY U. OF I.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	U	unit(s)
kg	kilogram(s)	UV	ultraviolet
LD_{50}	median lethal dose(s)	WBC	white blood cells (leukocytes), white blood count
m	meter(s)		
M	molar	wk	week(s)
mEq	milliequivalent(s)	wt	weight(s)
mM	millimolar	yr	year(s)
μM	micromolar		
mC,μC	milli-,microcurie(s)		

LIBRARY U. OF I. URBANA-CHAMPAIGN

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	2401-2478	363
CHEMICAL CARCINOGENESIS	(Chem)	2479-2582	370
PHYSICAL CARCINOGENESIS	(Phys)	2583-2611	387
VIRAL CARCINOGENESIS	(Viral)	2612-2690	390
IMMUNOLOGY	(Immun)	2691-2890	405
PATHOGENESIS	(Path)	2891-2914	432
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	2915-2951	436
MISCELLANEOUS	(Misc)	2952-3000	443
AUTHOR INDEX			
SUBJECT INDEX			

LIBRARY U. OF I. URBANA [illegible]

2401 RADIATION, IMMUNITY, AND CANCER. *(E.)*

Anonymous. *Lancet* 7770:217-218, 1972.

Recent findings on the relationship of cancer and the immune system are briefly reviewed. The distinction between thymus-derived lymphocytes associated with cell-mediated immunity (T cells), and bursa-derived antibody-secreting lymphocytes (B cells) is discussed. The increased incidence of cancer in patients with diminished immunological competence is emphasized, and it is speculated that radiation, because it may affect the immune system, may be a dangerous predisposer to cancer. In support of this suggestion, it is noted that irradiation for mammary cancer can produce lymphopenia which diminishes the effectiveness of immune surveillance for other cancers. (19 references)

2402 INTRODUCTORY REVIEW. MOLECULAR MECHANISMS IN VIRAL NEOPLASIA OF ANIMALS AND THEIR IMPLICATIONS IN THE ORIGIN OF CANCER IN MAN. *(E.)*

Dmochowski, L. (M. D. Anderson Hosp. Tumor Inst., Houston, Texas). *Am J Clin Pathol* 60(1):3-18, 1973.

Diverse methods such as biologic, morphologic, biophysical, immunologic, and biochemical have led to the demonstration, isolation, and identification of animal tumor viruses of both the ribonucleic acid (RNA) and deoxyribonucleic acid (DNA) carrying types. Similar methods have been applied to search for viruses in human neoplasms of many types. Morphologic studies have demonstrated virus-like particles, similar to those found to be the causative agents of animal neoplasia of diverse types, in many types of human tumors. Immunologic studies have demonstrated antibodies to tumor cells, and in some cases, to virus particles, in sera of patients with tumors such as Burkitt's lymphoma. Biochemical studies have demonstrated characteristic enzymes and a heavy or fast sedimenting RNA in all known tumor viruses of animals. Application of such studies to human neoplasms (leukemia, mammary cancer and other solid tumors) has demonstrated the presence of genetic information similar to that present in corresponding animal tumors of known viral origin. Thus, there exists in certain types of human neoplasms viral information which shows remarkable similarity to that from corresponding tumors of animals. (77 references)

2403 LATE EFFECTS OF RADIATION: CARCINOGENESIS.

(E.) Mole, R. H. (Med. Res. Council, Harwell, England). *Brit Med Bull* 29(1):78-83, 1973.

Aspects of cancer developing as a late effect of radiation are discussed. The growth of radiation-induced cancer from a single cell, rather than as a result of derangement of tissue architecture and function, is described. The hypothesis that radiation may affect the immune defenses of the body and may activate latent viruses is discussed. Clinical observations of radiation-induced cancer are reviewed; exposure to radiation in radiotherapy and in diagnostic radiology are surveyed and nuclear bomb survivors are discussed. Age as a factor in radiosensitivity to carcinogenesis is treated. (53 references)

2404 HERPESVIRUS: WOLF IN SHEEP'S CLOTHING?

(E.) Chedd, G. (*New Scientist*, Washington, D.C.). *New Scientist* 58(852):808-810, 1973.

Evidence which suggests an etiologic role for herpesviruses (HSV) in certain human cancers is reviewed. An association between HSV type 2 (HSV-2) which causes genital lesions and carcinoma of the cervix was first suggested by studies in the early 1960's. HSV-2 was shown to have the capacity to transform hamster cells *in vitro*, and immunological studies using anti-HSV-2 antiserum indicated that virus was present in cells taken from cervical cancer patients. Circulating antibody levels in cancer patients correlated well with the state of the disease. Nonviron antigens to both HSV-1 and HSV-2 which are free from contamination by structural components of the virus have been produced in the laboratory. Antibodies to the non-virion antigens have been found in the sera of patients with advanced cancers of the lip, mouth, oropharynx, nasopharynx, kidney, bladder, prostate, cervix and vulva. These nine cancers cluster anatomically near the sights at which HSV-1 and HSV-2 cause primary infections. Sera from patients with 20 other cancers were clear. (1 reference)

2405 ARENE OXIDES AND THE NIH SHIFT: THE METABOLISM, TOXICITY AND CARCINOGENICITY OF AROMATIC COMPOUNDS. *(E.)*

Daly, J. W. (Natl. Inst. Arthritis, Metabolism, Digestive Dis., Bethesda, Md.), D. M. Jerina and B. Witkop. *Experientia* 28(10):1129-1149, 1972.

In a lengthy review, the so-called NIH Shift is discussed and all known examples of the shift as brought about by monooxygenase are tabulated. The NIH shift plays a role in the metabolism of aromatic compounds. It involves the spontaneous isomerization of naphthalene-1,2 oxide to 1-naphthol accompanied by migration of deuterium from the 1- to 2-position. (189 references)

2406 EARLY EVENTS IN CELL-ANIMAL VIRUS INTERACTIONS. *(E.)*

Dales, S. (Public Hlth. Res. Inst. City New York, N.Y.). *Bacteriol Rev* 37(2): 103-135, 1973.

The complexity and variety of events associated with early cell-virus interactions are reviewed. Major topics covered include: the interpretation of electron microscope images and the problem of artifacts; fusion, lysis, and repair of cellular membranes; mechanisms for internalization of the inoculum; activities of virion-associated enzymes and their effect on the host; vectorial movement of the inoculum to sites of genome replication; uncoating; the action of antibody and specific inhibitors; subviral components; the role of lysosomes in the early process of infection; and host- and virus-dependent modifications and restrictions. The following viruses are specifically discussed in the section on mechanisms for internalization of the inoculum: herpesviruses, poxviruses, adenoviruses, papovaviruses, reoviruses, rhabdoviruses, myxoviruses, paramyxoviruses, picornaviruses and oncornaviruses. (39 references)

2407 THE EPIDEMIOLOGY OF CANCER: FOUR PERSPECTIVES. *(E.)* Fritsch, W. (U. Iowa Coll. Med., Iowa City), D. Culp, N. Rossi and H. J. Buchsbaum. *J Iowa Med Soc* 63(5):199-205, 1973.

Epidemiology of cancer in four specific areas is reviewed: skin, penile, lung and cervical cancer. UV light is the major causitive factor of skin cancer, particularly basal cell carcinoma, squamous cell carcinoma and actinic keratosis. Therefore, occupation, geography, weather and genetics (such as light skin or genetic disorders) are important epidemiological factors. The steep increase of skin cancer incidence after the age of 55-59 yr indicates that the carcinogenic effects of UV light are cumulative. Chemical carcinogens, especially relating to occupation, and benign skin lesions can also contribute to development of skin cancer. Poor hygiene is probably the largest contributor to cancer of the penis, especially in the uncircumcised. Penile cancer is rare among those males circumcised at birth. Smegma, which can accumulate in the closed preputial space, has been linked to the production of penile cancer by a study in which smegma was applied to exposed surfaces and buried skin tunnels of Paris R^3 strain mice. The biggest factor in lung cancer incidence is smoking. There are no good means of early detection of lung cancer. The emphasis should be on prevention, then, rather than detection of symptoms which mean a far-advanced state of the lesion. Studies of cervical carcinoma reveal no evidence of a hereditary component, nor is there any correlation with hormone status or menstrual irregularities. Cervical carcinoma is rare in celibate women and six times more common among prostitutes, suggesting a correlation between coitus and cervical carcinoma. It does not appear that the physical act of coitus plays a causitive role, since a group of lesbians who practiced mechanical masturbation with a phallus were found to have an incidence no higher than among celibate women. Cervical carcinoma incidence varies with geographic location, but may follow ethnic and economic lines rather than political borders. The incidence is significantly higher in societies where circumcision is uncommon. Also, women living in high population and industrial environments are at greater risk than women living in rural areas. (No references)

2408 RADIATION CARCINOGENESIS. *(E.)* Ilbery, P. L. T. (Sch. Pub. Hlth. Trop. Med., U. Sydney, Australia) and A. B. Rickinson. *Australas Radiol* 17(1):66-77, 1973.

Classical discoveries in human radiation carcinogenesis are reviewed. X-ray induced skin cancer in humans was first described in 1902; osteomyelitis of mandible and maxilla in radium dial painters in 1924. The time of tumor appearance was dose-dependent; the apparent threshold of 1000 rad may reflect long latent periods at low doses. Uranium miners showed susceptibility to lung cancer, with 16 yr latency. Thorotrast induced cancer at site of injection and in organs concentrating the compound. Local irradiation of children for thymic enlargement increases incidence of thyroid tumors, with 20 yr latency. Radioactive iodine given internally induces mostly benign nodules. Whole-body irradiation, as of Hiroshima and Nagasaki atom bomb subjects, caused increased leukemia and thyroid, lung, and breast tumors. A dose-dependent increase in acute and chronic myeloid leukemia in the survivors peaks some 5-7 yr after exposure. The high incidence of leukemia resulting from the Hiroshima explosion relates to a neutron component essentially absent from the purer gamma radiation of the Nagasaki explosion. Leukemia incidence among radiologists is 8-10 times greater that among physicians in general. Children exposed to X-rays *in utero* show 2-fold increase in leukemia; parental exposure prior to conception constitutes a similar risk. Radiophosphorus increases leukemia in polycythemia vera patients, as also does radiotherapy of ankylosing spondylitis patients. The question of whether a threshold dose of radiation exists for human radiation leukemogenesis remains unanswered. In experiments with radiation-induced leukemia in mice, chromosomal changes recognizable in the leukemic cells represent a secondary manifestation of the neoplasia and not the cause of it. In general, thymectomy of mice prior to irradiation abolishes the leukemic response to whole-body exposure, but subsequent thymic grafts restore susceptibility. Leukemia in the grafted animals arises, in half the cases, from the unexposed graft cells. This evidence, together with the presence of viral particles in leukemic cells and the induction of leukemia by acellular transfer, indicates a role of viruses in radiation leukemogenesis. Radiation also reduces immune competence, thus reinforcing the leukemogenic process. Doses above optimal level reduce the incidence of leukemia induction, due to inactivation of newly transformed cells. Application of murine models to man is complciated by heritable and environmental heterogeneity of human populations. (96 references)

2409 LEUKAEMIA ANTIGENS AND IMMUNITY IN MAN. *(E.)* Harris, R. (St. Mary's Hosp., Manchester, England). *Nature* 241(5385):95-100, 1973.

Research on the nature of human leukemia antigens is briefly reviewed. Immune reactions to autochthonous and allogeneic leukemia material suggest such antigens exist. Antibody responses to leukemia in man and cell mediated immune responses are described. Leukemia-associated antigens discovered to date are characterized with emphasis on variations seen in normal iso-antigens in leukemia. Studies on embryonic antigens and on the behavior of HL-A antigens in leukemia are described. Immunodepression in connection with leukemia is also discussed. (92 references)

2410 MYCOTOXINS. *(E.)* Pitt, J. I. (CSIRO Div. Food Res., North Ryde, NSW, Australia). *Food Techno Aust* 125(6):291-294, 1973.

In contrast to microbial toxins which traditionally are antigenic proteins, fungal toxins are nonantigenic polycyclic compounds. They have been shown to cause sickness and death in epidemic proportions in humans, turkeys, ducklings, and trout. The classic example of mycotoxicosis is alimentary toxic aleukia, which affected Russians living in the grain belt and was due to grain which had overwintered in the field and became moldy with species of *Fusarium* and *Cladosporium*. In other examples, a toxin of *Aspergillus flavus* which grew in improperly dried peanuts caused "Turkey X" disease, while toxin from the same fungus growing on cottonseed caused hepatomas in trout. These and related toxins are called aflatoxins. Aflatoxins also can cause hepatomas when administered to rats. Other carcinogenic mycotoxins are islanditoxin and luteoskyrin, produced by *Penicillium islandicum*, a comtaminant in mouldy rice, and patulin, produced by *Penicillium expansum*, the cause of a common apple rot. Evidence is mounting that some human liver cancer, which has an exceptionally high incidence in parts of Africa and Asia, may be caused by mycotoxins in foodstuffs. Analysis of agricultural or food commodities for the presence of aflotoxins, whether by chemical, chromatographic, or toxicological procedures, is difficult due to interfering compounds, sampling problems, and slowness of the assays. (4 references)

2411 FOREWORD. *(E.)* Wilbanks, G. (Rush Presbyterian-St. Luke's Med. Center, Chicago, Ill.). *Cancer Res* 33:1352, 1973.

A meeting of the American Cancer Society was held to review recent research on the association between herpes virus type 2 and cervical cancer, as well as studies of related viruses and other human and animal cancers. Many concepts of cancer as a spectrum of disease, beginning with minor but recognizable *in situ* changes progressing to invasive, have been developed in studies of cervical cancer. These concepts, originally applied to neoplastic lesions of the cervix, are now applied to the majority of neoplastic diseases. (No references)

2412 SUMMARY OF RECENT OBSERVATIONS ON THE MOLECULAR BIOLOGY OF RNA TUMOR VIRUSES AND ATTEMPTS AT APPLICATION TO HUMAN LEUKEMIA. *(E.)* Gallo, R. C. (Natl. Cancer Inst., Bethesda, Md.). *Am J Clin Pathol* 60(1):80-87, 1973.

RNA tumor viruses apparently replicate through a DNA intermediate (provirus), a reaction catalyzed by the polymerase of these viruses, the so-called "reverse transcriptase." The viral genomic RNA is large (about 70S) and contains large tracts of poly A. These viruses can be induced by some halogenated pyrimidines from some non-producer cells. The induction of these viruses can be blocked by 3'-deoxyadenosine (cordycepin) but only at a very specific time (12 to 18 hours after induction). The mechanism of this block in induction may be due to interference with normal processing of the viral RNA after its transcription from proviral DNA, perhaps by blocking poly A formation. A reverse transcriptase is present in cytoplasmic particulate fractions from some patients with acute leukemia. This enzyme is very similar to the reverse transcriptase of viruses and may be viral in origin. Some criteria for its detection and features which distinguish it from major DNA polymerases of cells are reviewed. Some rationales and approaches for inhibition of viral replication are discussed. (30 references)

2413 RIBOFLAVIN AND CANCER: A REVIEW. *(E.)* Rivlin, R. S. (Columbia U. Coll. Physicians Surg., New York, N.Y.). *Cancer Res* 33(9):1977-1986, 1973.

Several studies indicate that riboflavin deficiency inhibits tumor growth in experimental animals and possibly in man, but the precise mechanisms involved have not been elucidated. Azo dye carcinogenesis in liver appears to be a special case in that riboflavin deficiency increases the potency of these drugs in tumor causation, probably in large measure because flavin cofactors are involved in their metabolic degradation. Riboflavin influences uptake of chemotherapeutic drugs in at least one instance (methotrexate) into neoplastic cells. The fact that folic acid metabolism depends upon flavin cofactors may have implications for the use of folate antagonists in cancer chemotherapy. Neoplastic liver appears to lack certain mechanisms that regulate metabolism in normal tissues. In addition, it is more resistant than normal liver to riboflavin deficiency, as reflected in the relatively higher concentrations of flavin mononucleotide and flavin adenine dinucleotide measured in samples of tumor than in the host rat liver. By contrast, the concentration of free riboflavin is greatly depressed in Novikoff hepatoma grown in riboflavin deficient animals. The loss of this relatively dispensable flavin fraction may be one of the mechanisms that enable tumor to retain the more critical flavin mononucleotide and flavin adenine dinucleotide fractions when dietary riboflavin is diminished. The observation that certain patients with cancer excrete less riboflavin than do normal individuals requires further confirmation. (75 references)

2414 CELLULAR IMMUNITY TO HUMAN TUMOR-ASSOCIATED ANTIGENS. A REVIEW. *(E.)* Herberman, R. B. (Natl. Cancer Inst., Bethesda, Md.). *Isr J Med Sci* 9(3):300-307, 1973.

Tumor-specifity of antigens which are seemingly associated with the neoplastic state of human tissue was studied. It was demonstrated that peripheral blood lymphocytes from patients with a variety of tumors could inhibit *in vitro* colony formation by tissue culture cells derived from the tumors. Allogeneic lymphocytes from patients with the same histological types of tumors gave positive reactions, suggesting that reactivity was directed against antigens common with each of the tumor types. By means of delayed skin reactions to extracts of a variety of human tumors reactivity to tumor-associated antigens was detected, although in some cases it appeared that at least some of the observed reactivity was directed against tissue-specific antigens. Correlation with clinical course was found. Lymphocyte stimulation tests have been less useful in this study since control tests with approriate normal cells have generally not been performed. Contact between immune lymphocytes and antigen has been shown in many systems to result in inibition of migration of leukocytes from a capillary tube. There have been three recent reports of success of this method. In one case extracts of breast cancers inhibited the migration of the autologous leukocytes in 8 out of 22 patients.

The specificity of these reactions was well controlled. The tumor extracts did not inhibit the migration of leukocytes from normal individuals and extracts of autologous, noninvolved breast tissue caused no migration of the patients' leukocytes. In the small number of patients studied there was no correlation of reactivity with extent of tumor involvement. The other two tests of this sort showed similar results. These different assay methods show a general lack of correlation in their results. Various assays for cellular immunity may be measuring reactivity to different antigenic specificities, or may be reflecting involvement of different components of the immune response. (49 references)

2415 EPIGENETIC TRANSFORMATION BY RNA FROM HUMAN NEOPLASTIC CELLS. *(E.)* DeCarvalho, S. (Belmont Med. Clin., Bellflower, Ca.). *Oncology* 27(1):3-29, 1973.

Studies of neoplastic disorganization at the level of RNA-protein systems are reviewed and original experiments are described. Investigations of the transfer of information, including antigenic information, from cell to cell via RNA are discussed. In original experiments, RNA prepared from normal and leukemic human cells was exposed to enzymes and the effects were observed. In another experiment it was found that under the influence of leukemic RNA human amniotic cells in culture produce leukemic antigens. The morphologic aspects of the effect of leukemic RNA are described. (76 references)

2416 ON THE ETIOLOGY OF CANCER OF THE LOWER LIP. *(E.)* Ju, D. M. C. (Columbia-Presbyterian Med. Ctr., New York, N.Y.). *Plast Reconstr Surg* 52(2):151-154, 1973.

A composite picture of the person most likely to develop cancer of the lower lip is drawn from the characteristics of 26 private patients treated over a 15-yr period for this disease. It is a man who has a light complexion, who is not a professional athlete, but who loves sports. During the summer, and on weekends and vacations, he participates in those outdoor sports which are best performed on good sunny days. He has been sunburned several times, particularly on the lower lip. He has never used chewing tobacco, or any oral irritant, and he does not smoke a pipe. (20 references)

2417 NUCLEIC ACID HYBRIDIZATION AND THE RELATIONSHIP BETWEEN CERVICAL CANCER AND HERPES SIMPLEX VIRUS TYPE 2. *(E.)* Goodheart, C. R. (Bio-Labs, Inc., Northbrook, Ill.). *Cancer Res* 33(6): 1548-1551, 1973.

Although seroepidemiology has clearly indicated that herpes simplex virus type 2 (HSV-2) is worth investigating as a possible etiological agent in cervical cancer, it is doubtful whether such an approach can ever provide definitive evidence of a causal relationship. A major problem is that if both cervical carcinoma and HSV-2 are transmitted venereally, without any other feature in common, seroepidemiological surveys will indicate a spurious relationship. It is recommended that molecular hybridization methods be used to obtain more conclusive evidence for the implication of HSV-2 in cervical carcinoma, and various approaches are described such as competition-type tests using mRNA from tumor tissue, *in vitro* labeling of viral DNA in desquamated cervical cells, hybridization of DNA extracted tumor tissue with labeled cRNA, and hybridization of labeled viral DNA with DNA extracted from tumor tissue. Data on the number of genomes per malignant cell, the relationship of the viral genetic material to the cellular DNA, and the function of the viral genetic material in terms of the messenger RNA molecules transcribed are also needed to properly evaluate the role of the HSV-2 genome in the malignant process. (29 references)

2418 INFECTIVE AGENTS IN THE CAUSATION OF HODGKIN'S DISEASE. A REVIEW OF THE EPIDEMIOLOGICAL EVIDENCE. *(E.)* Abramson, J. H. (Hadassah Med. Sch., Jerusalem, Israel). *Isr J Med Sci* 9(7):932-953, 1973.

Epidemiological observations with a bearing on the possible role of infective agents in the causation of Hodgkin's disease are reviewed, with special reference to a number of possibly relevant factors. A bimodal age incidence with peaks in the decade from 20 to 30 yr and in old age has been interpreted as a reflection of two different diseases, as supported by geographical, histological, clinical and prognostic considerations. International variation in frequency has been adduced as evidence for the role of environmental factors. Possible effects of social class are not convincing. In the United States, incidence is greater among non-whites than among whites. In Brooklyn, Jews showed twice the risk after 50 yr of age, but no excess in the 15-34 yr group. The disease is rare at all ages in Japan but more common among Japanese in Hawaii. Evidence for high incidence of clinical onset in winter has been disputed, and may reflect operation of exogenous factors, such as viral infections. It has been reported that among males under 15 yr who died of Hodgkin's disease in the United States in 1960-64, significantly more than the expected number were born in July and August, but this seasonal variation was not confirmed by several other studies. There is a tendency for cases occurring at closely related times to be located close to one another in space which would suggest the influence of transitory environmental factors, such as infective agents. High incidences of Hodgkin's disease for a number of years after 1953 in Albany county, New York, and between 1968 and 1971 in Union County, Ohio, have been interpreted by some analysts, however, as reasonable chance situations within a random distributions. The apparent epidemic in Albany county provided some of the strongest support for the hypothesis of person-to-person transmission but this and considerable other evidence is refuted by studies showing no evidence. Familial incidence, where apparently statistically significant, was so low that heredity could have played only a minor role and "infectious agents" were suggested as more important. In certain studies, higher antibodies to Epstein-Barr virus (EBV) have been observed in patients with Hodgkin's disease then in patients with other lymphomas or in normal controls. Aside from this suggestive laboratory evidence, the only epidemiological evidence for a linkage between EBV and Hodgkin's disease is that provided by reports of a number of cases of occurrence of infectious mononucleosis (a disease possibly caused by EBV) before or at the same time as recognition of Hodgkin's disease. Since evidence of infection with EBV is not found in all cases of Hodgkin's disease, it may be postulated that other viruses, possibly similar to EBV, may play a similar role. Evidence for association of Hodgkin's disease with tonsillectomy, appendectomy and contact with animals cannot be regarded as well established. Thus the weight of the epidemiological evidence is consistent with, but does not prove, the hypothesis that viral infection plays a role in the causation of the disease. (160 references)

2419 WHY IS PANCREATIC CANCER INCIDENCE UP; STOMACH CANCER DOWN? *(E.)* Krain, L. S. (U. California Los Angeles Med. Ctr.). *Geriatrics* 28(4): 140-145, 1973. (No references)

2420 ANTIBODY ACTIVITY OF HUMAN MYELOMA GLOBULINS. *(E.)* Seligmann, M. (Saint-Louis Hosp., Paris, France) and J. C. Brouet. *Semin Hematol* 10(2):163-177, 1973. (92 references)

2421 IMMUNOGLOBULIN STRUCTURE DETERMINED FROM PRODUCTS OF PLASMA CELL NEOPLASMS. *(E.)* Solomon, A. (U. Tennessee Mem. Res. Ctr., Hosp., Knoxville) and C. L. McLaughlin. *Semin Hematol* 10(1):3-17, 1973. (129 references)

2422 CAN VIRUSES CAUSE CANCER? *(E.)* Nutter, R. L. (Dept. Microbiol., Loma Linda U., Calif.). *Life Health* 88(7):18-24, 1973. (No references)

2423 STASTICAL ASPECTS OF A CO-OPERATIVE TRIAL ON THE TREATMENT OF OVARIAN CARCINOMA. *(E.)* MacRae, K. D. (Dept. Med. Statistics, Queens, U., Belfast, Ireland). *Postgrad Med J* 49(568):78-80, 1973. (7 references)

2424 OVARIAN CANCER CLINICAL SURVEY. *(E.)* Harcus, A. W. (Schering Chem. Limited, Burgess Hill, Sussex, England). *Postgrad Med J* 49(568): 81-82, 1973. (No references)

2425 LEUKAEMIC ANTIGENS. *(E.)* Pegrum, G. D. (Fulham Hosp., London, England). *Br J Haematol* 24(1):1-6, 1973. (49 references)

2426 CARCINOGENS AND FOOD: A BROAD ASSESSMENT. *(E.)* Roe, F. J. C. (Tobacco Res. Council, London, England). *Proc R Soc Med* 66(1):23-26, 1973. (16 references)

2427 TALC: A POSSIBLE OCCUPATIONAL AND ENVIRONMENTAL CARCINOGEN. *(E.)* Blejer, H. P. (California Dept. Publ. Hlth., Los Angeles) and R. Arlon. *J Occup Med* 15(2):92-97, 1973. (26 references)

2428 CHROMOSOMAL CHANGES DUE TO VIRUSES. *(E.)* Nichols, W. W. (Inst. Med. Res., Camden, N. J.). *Triangle* 11(3):103-106, 1972. (25 references)

2429 HEPATOMA IN PAPUA NEW GUINEA. *(E.)* Nugent, R. P. (Fac. Med., U. Papua New Guinea, Boroko). *Aust NZ J Surg* 42(3):251-256, 1973. (18 references)

2430 TUMOUR SPECIFIC TRANSPLANTATION ANTIGENS IN ANIMAL AND HUMAN TUMOURS AND THE THERAPEUTIC IMPLICATIONS OF THE DEVELOPMENT OF HUMORAL AND CELLULAR IMMUNITY TO SUCH ANTIGENS. *(E.)* Sirsi, M. (Microbiol., Pharmacol. Lab., Indian Inst. Sci., Bangalore). *Indian J Cancer* 9(4):337-339, 1972. (No references)

2431 ANTIOXIDANTS IN CEREALS AND IN FOOD PRESERVATIVES AND DECLINING GASTRIC CANCER MORTALITY. *(E.)* Shamberger, R. J. (Cleveland Clin. Fdn., Ohio), S. Tytko and C. E. Willis. *Cleve Clin Q* 39(3):119-124, 1972. (14 references)

2432 EXPERIMENTAL APPORACHES TO ENDOMETRIAL AND OVARIAN CANCER. *(E.)* Brush, M. G. (St. Thomas's Hosp. Med. Sch., London, England). *Postgrad Med J* 49(568):72-75, 1973. (29 references)

2433 THE USE OF OESTRADIOL UPTAKE AND BINDING-SITE STUDIES IN ENDOMETRIAL AND OVARIAN CARCINOMA. *(E.)* Taylor, R. W. (St. Thomas's Hosp. Med. Sch., London, England), M. G. Brush and R. J. B. King. *Postgrad Med J* 49(568):77, 1973. (5 references)

2434 RNA-CONTAINING VIRUSES AS TUMOR PRODUCING AGENTS. *(Ger.)* Weissman, C. (Inst. Molecularbiol., U. Zurich, Switzerland). *Pathol Microbiol* 39(1):14-16, 1973. (3 references)

2435 IMMUNOLOGY AND CANCER. *(E.)* Weksler, M. E. (New York Hosp.- Cornell Med. Ctr., N.Y.). *NY State J Med* 73(4):544-549, 1973. (29 references)

2436 THE ETIOLOGY AND PATHOGENESIS OF ORAL CANCER. *(E.)* Osterkamp, R. W. (St. Louis, Mo.) and J. B. Whitten. *CA* 23(1):28-32, 1973. (2 references)

2437 SUMMARY OF PAPERS DELIVERED AT THE CONFERENCE ON HERPESVIRUS AND CERVICAL CANCER (KEY BISCAYNE, FLORIDA). *(E.)* Klein, G. (Karolinska Inst., Stockholm, Sweden). *Cancer Res* 33(6):1557-1563, 1973. (8 references)

2438 A SELECTIVE REVIEW OF EXPERIMENTAL STUDIES IN CERVICAL CARCINOMA. *(E.)* Wilbanks, G. D. (Rush-Presbyterian-St. Luke's Med. Ctr., Chicago, Ill.). *Cancer Res* 33(6):1379-1381, 1973. (34 references)

2439 SUMMARY OF INFORMAL DISCUSSION ON GENERAL ASPECTS OF CERVICAL CANCER. *(E.)* Wilbanks, G. D. (Rush-Presbyterian-St. Luke's Med. Ctr., Chicago, Ill.). *Cancer Res* 33(6):1382-1384, 1973. (No references)

2440 SUMMARY OF INFORMAL DISCUSSION ON GENERAL ASPECTS OF HERPESVIRUS. *(E.)* Goodheart, C. R. (BioLabs, Inc., Northbrook, Ill.). *Cancer Res* 33(6):1417-1418, 1973. (No references)

2441 SUMMARY OF INFORMAL DISCUSSION OF PART III OF GENITAL HERPESVIRUS AND OF *IN VIVO* ANIMAL STUDIES. *(E.)* Rawls, W. E. (Baylor Coll, Med., Houston, Tex.). *Cancer Res* 33(6):1511-1514, 1973. (No references)

2442 SUMMARY OF INFORMAL DISCUSSION OF EXPERIMENTAL APPROACHES. *(E.)* Deinhardt, F. (Rush-Presbyterian-St. Luke's Med. Ctr., Chicago, Ill.). *Cancer Res* 33(6):1552-1555, 1973. (2 references)

2443 SUMMARY OF INFORMAL DISCUSSION OF PART I OF GENITAL HERPESVIRUS. *(E.)* Josey, W. E. (Emory U. Sch. Med., Atlanta, Ga.). *Cancer Res* 33(6):1464, 1973. (5 references)

2444 ANTIGENS OF HERPES SIMPLEX VIRUS OF ORAL AND GENITAL ORIGIN. *(E.)* Wildy, P. (U. Birmingham Med. Sch., England). *Cancer Res* 33(6):1465-1468, 1973. (36 references)

2445 A REVIEW OF THE IDENTIFICATION AND TITRATION OF ANTIBODIES TO HERPES SIMPLEX VIRUSES TYPE 1 AND TYPE 2 IN HUMAN SERA. *(E.)* Plummer, G. (Loyola U. Stritch Sch. Med., Maywood, Ill.). *Cancer Res* 33(6):1469-1476, 1973. (26 references)

2446 SUMMARY OF INFORMAL DISCUSSION OF PART II OF GENITAL HERPESVIRUS. *(E.)* Nahmias, A. J. (Emory U. Sch. Med., Atlanta, Ga.). *Cancer Res* 33(6):1483-1484, 1973. (7 references)

2447 CARCINOGENS AND IRRITANTS. *(E.)* Anonymous. *J Inst Chem (India)* 44(Part 6): 175-176, 1972. (No references)

2448 CYTOHISTOPATHOLOGY OF CERVICAL CANCER. *(E.)* Stern, E. (Sch. Publ. Hlth., U. Calif., Los Angeles). *Cancer Res* 33(6):1368-1378, 1973. (28 references)

2449 EMPYEMA IN LUNG CANCER -- THE CLOUD WITH A SILVER LINING. *(E.)* Anonymous. *JAMA* 224(12):1644, 1973. (3 references)

2450 TRANSPLACENTAL CARCINOGENESIS. *(E.)* Anonymous. *Lancet* (7817):1425-1426, 1973. (11 references)

2451 PRELEUKEMIA. *(E.)* Anonymous. *Lancet* (7817):1426-1427, 1973. (14 references)

2452 PHOTOCHEMICAL REACTIONS OF NATURAL MACROMOLECULES. PHOTOREACTIONS OF PROTEINS. *(E.)* Neckers, D. C. (U. New Mexico, Albuquerque). *J Chem Educ* 50(3):164-168, 1973. (49 references)

2453 A REVIEW OF CYTOGENETIC STUDIES OF GYNECOLOGIC NEOPLASMS. *(E.)* Gelender, H. (U. Chicago Med. Sch., Ill.) and J. A. Cohen. *Chicago Med Sch Q* 32(1,2,3,4):59-62, 1973. (12 references)

2454 SMOKING AND IMMUNITY. *(E.)* Holt, P. G. (Dept. Microbiol., U. Western Australia, Perth), W. R. Thomas and D. Keast. *Lancet* (7815): 1316, 1973. (11 references)

2455 PYRIMETHAMINE IN CENTRAL-NERVOUS-SYSTEM LEUKAEMIA. *(E.)* Price, L. A. (Roy. Marsden Hosp., London, England). *Lancet* (7815): 1315, 1973. (No references)

2456 INITIAL SITE OF HODGKIN'S DISEASE. *(E.)* Vianna, N. J. (New York St. Dept. Hlth., Albany), P. Greenwald and J. N. P. Davies. *Lancet* (7817):1440, 1973. (13 references)

2457 THE CHALONES. *(Fr.)* Vandermeersch, B. (No affiliation). *Recherche* 4(32):281-283, 1973. (No references)

2458 SUMMARY OF INFORMAL DISCUSSION ON ONCOGENICITY OF OTHER HERPESVIRUSES. *(E.)* Goodheart, C. R. (BioLabs, Inc., Northbrook, Ill.). *Cancer Res* 33(6):1443-1445, 1973. (5 references)

2459 OVARIAN CANCER: SOME NEW ANALYTICAL APPROACHES. *(E.)* Griffiths, K. (St. Thomas's Hosp. Med. Sch., London, England), W. J. Henderson, J. A. Chandler and C. A. F. Joslin. *Postgrad Med J* 49(568):69-72, 1973. (15 references)

2460 IMMUNOLOGY IN HODGKIN'S DISEASE. *(E.)* Gollerkeri, M. P. (Tata Mem. Hosp., Bombay, India). *Indian J Cancer* 9(4):312-315, 1972. (5 references)

2461 THE AETIOLOGY AND NATURAL HISTORY OF HODGKIN'S DISEASE. *(E.)* Karnik, A. M. (Tata Mem. Hosp., Bombay, India). *Indian J Cancer* 9(4):303-306, 1972. (4 references)

2462 *ONCOLOGICA GASTROENTEROLOGICA* AND THE CONDITIONS IN YUGOSLAVIA. *(E.)* Knezevic, S. (U. Zagreb Med. Sch., Yugoslavia). *Acta Med Iugosl* 25(4): 411-416, 1971. (19 references)

2463 PROPERTIES OF ONCOGENIC VIRUS. *(Fr.)* Cuzin, F. (Pasteur Inst., Paris, France). *Bull Cancer* 59(1):15-20, 1972. (No references)

2464 TUMOUR CELL KINETICS. *(E.)* Lamerton, L. F. (Royal Cancer Hosp., Sutton, Surrey, England). *Br Med J* 29(1):23-28, 1973. (34 references)

2465 THE EFFECT OF ALFATOXIN IN ANIMALS AND MAN. *(Ger.)* Bösenberg, H. (Münster U. Dept. Hyg., W. Germany). *Z Lebensm Unters-Forsch* 151(4):245-249, 1973. (10 references)

2466 BIOLOGICAL FEATURES OF TUMOR PROGRESSION. *(Rus.)* Shapot, V. S. (Moscow, USSR). *Vopr Onkol* 19(4):89-92, 1973. (No references)

2467 CARCINOGENIC SUBSTANCES AND CANCER PROPHYLAXIS IN THE U.S.S.R. *(Rus.)* Shabad, L. M. (Inst. Exp. Clin. Oncol., Acad. Med. Sci., Leningrad, USSR). *Vopr Onkol* 18(10):3-10, 1972. (11 references)

2468 CONDITIONS GENERATING SOME OF THE CARCINOGENIC MYCOTOXINS. *(Ger.)* Orth, R. (Fed. Res. Inst. Fresh Food Maintenance, Karlsruhe, W. Germany). *Z Lebensm Unters-Forsch* 151(4): 267-273, 1973. (30 references)

2469 THE DEVELOPMENTAL HISTORY OF THE NEOPLASTIC PLASMA CELL IN MICE: A BRIEF REVIEW OF RECENT DEVELOPMENTS. *(E.)* Potter, M. (Natl. Cancer Inst., Bethesda, Md.). *Semin Hematol* 10(1):19-32, 1973. (81 references)

2470 RECENT FINDINGS RELATED TO THE PHYSIOLOGY AND ENDOCRINOLOGY OF REPRODUCTION IN ANIMALS AND MAN. *(Ger.)* Neumann, F. (Berlin, W. Germany), W. Elger and Y. Nishino. *Naturw Rdsh* 26(5):181-191, 1973. (No references)

2471 GYNECOLOGICAL TUMORS IN CHILDHOOD AND ADOLESCENCE. *(E.)* Barber, H. R. K. (Lenox Hill Hosp., New York, N.Y.) and E. A. Graber. *Obstet Gynecol Surv* 28(5):357-381, 1973. (70 references)

2472 CURRENT STATUS OF VIROLOGICAL RESEARCH IN HUMAN LEUKEMIAS AND SARCOMAS. *(E.)* Boiron, M. (Saint-Louis Hosp., Paris, France). *Pathol Biol* 21(1):77-81, 1973. (21 references)

2473 CANCER AND THE EVOLUTION OF SPECIES: A RANSOM. *(E.)* Grouchy, J. de (Hosp. Sick Children, Paris, France). *Biomedicine* 18:6-8, 1973. (10 references)

2474 THE DISCRIMINATION OF LEUKEMIC FROM NORMAL CELLS. *(E.)* Metcalf, D. (Cancer Res. Unit, Walter & Eliza Hall Inst., Melbourne, Australia). *Biomedicine* 18(4):264-271, 1973. (51 references)

2475 STANDARDIZATION OF CHEMICAL COMPOUNDS FEATURING TUMORIGENIC PROPERTIES IN TERMS OF HYGIENE. *(Rus.)* Litvinov, N. N. (Moscow, USSR). *Gig Sanit* (3):95-102, 1973. (44 references)

2476 EPIDEMIOLOGY OF COLON-RECTAL CANCER 1972. *(E.)* Macdonald, E. J. (U. Texas M.D. Anderson Hosp., Houston). *Cancer Bull* 25(2):33-41, 1973. (No references)

2477 GENETICS OF CHILDHOOD CANCER. *(E.)* Strong, L. C. (U. Texas, Houston, Grad. Sch. Biomed. Sci.). *Cancer Bull* 25(2):26-29, 1973. (No references)

2478 IMMUNOGLOBULIN SYNTHESIS AND TUMOR KINETICS OF MULTIPLE MYELOMA. *(E.)* Salmon, S. E. (U. Arizona Coll. Med., Tucson). *Semin Hematol* 10(2): 135-147, 1973. (29 references)

2479 FORMATION OF ELECTROPHILIC *N*-ACETOXYARYLAMINES IN CYTOSOLS FROM RAT MAMMARY GLAND AND OTHER TISSUES BY TRANSACETYLATION FROM THE CARCINOGEN *N*-HYDROXY-4-ACETYLAMINOBIPHENYL. *(E.)* Bartsch, H. (McArdle Lab., Madison, Wisc.), C. Dworkin, E. C. Miller and J. A. Miller. *Biochim Biophys Acta* 304(1):42-55, 1973.

Rat tissue cytosols were assayed for their capacity to transfer enzymatically the acetyl of an acethydroxamic acid to a hydroxylamine (transacetylase activity). The 105,000 x g supernatant fractions of tissues were known to catalyze the transfer of the *N*-acetyl group of certain carcinogenic aromatic acethydroxamic acids to the O atom of aromatic hydroxylamines. With *N*-hydroxy-2-acetylaminofluorene (*N*-hydroxy-AAF) or *N*-hydroxy-4-aminobiphenyl (*N*-hydroxy-ABP) as substrates, rat liver cytosol had the highest transacetylase activity. Kidney and intestinal mucosa cytosols showed 15-20% of the liver cytosol transacetylase activity. Rat lung and ear duct sebaceous gland cytosol showed no transacetylase with either substrate. Alone among tissues, mammary glands from 16-21 day pregnant rats showed moderate activity with 9 *N*-hydroxy-ABP as acetyl acceptor and no activity with acceptor *N*-hydroxy-AF. Spleen and muscle cytosol had low activity with acceptor *N*-hydroxy-AF or *N*-hydroxy-ABP. The mammary gland transacetylase had a pH optimum of 7.5. This system had 20% the transacetylase activity of liver when *N*-hydroxy-4-acetylaminobiphenyl was used as acetyl donor and *N*-hydroxy-ABP was used as acceptor.

2480 EARLY ENDOMETRIAL CHANGES AND HISTOGENESIS OF MALIGNANT TUMORS IN THE CORPUS UTERI OF THE MOUSE AFTER IMPLANTATION OF 3-METHYLCHOLANTHRENE (I). *(Ger.)* Boquoi, E. (Obstet. Gynecol. Clin., Free U., Berlin, Germany) and H. Ebner. *Arch Gynaekol* 215(2):215-232, 1973.

Using a modification of the method of Taki and Iijima, 3-methylcholanthrene was implanted into the right uterine horn of 65 female NMR 1 mice with intact ovarian function. Mice of the same strain in which beeswax had been implanted by the same method served as controls. Mice were sacrificed after 1, 3 and 7 days and at weekly intervals thereafter (2-22 weeks). Five stages of histological change occurred in the uterus: (1) trauma and toxic necrosis of the endometrial epithelium and acute, nonspecific, exudative inflammation during the first 24 hr; (2) epithelial defects and the onset of a fibroblast reaction in vascular connective tissue of the stroma from days 3 to 5; (3) chronic inflammation in all layers of the corpus uteri with foci of necrosis in the stroma, necrotizing vasculitis and fibrinoid necrosis of the arterioles from weeks 1 through 3; (4) chronic inflammation, arteriolar vasculitis, endometrial hyperplasia, squamous cell metaplasia with keratinization, basal cell hyperplasia and dysplasia from weeks 4 through 7; and (5) the onset of malignant transformation of the epithelium from weeks 8 through 22. Changes observed during the last stage included carcinoma *in situ*, squamous cell carcinoma, adenocarcinoma and, in the cervical region and lower part of the uterus, mucoepidermoid carcinoma.

2481 INCREASED SENSITIVITY OF XERODERMA PIGMENTOSUM CELLS TO SOME CHEMICAL CARCINOGENS AND MUTAGENS. *(E.)* Stich, H. F. (Cancer Res. Ctr., Vancouver, Canada), R. H. C. San and Y. Kawazoe. *Mutat Res* 17(1):127-137, 1973.

The clone-forming capacity and level of DNA repair was examined in normal human cells and repair-deficient Xeroderma pigmentosum (XP) fibroblasts exposed to various chemical carcinogens and mutagens. The cultured fibroblasts were treated for 90 min with the carcinogenic and mutagenic 4-nitroquinoline 1-oxide (4NQO), 4-hydroxyaminoquinoline 1-oxide (4HAQO), 2-methyl-4-nitroquinoline 1-oxide (2-Me-4NQO), 3-methyl-4-nitropyridine 1-oxide (3-Me-4NPO) and the non-carcinogenic 6-nitroquinoline I-oxide (6NQO) dissolved in 100% ethanol and diluted with tissue culture medium. The response of the cells to the *N*-oxides was compared to that induced by UV-irradiation and the mutagen and carcinogen *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine (MNNG) dissolved in distilled water and then diluted with Eagle's minimum essential medium. The XP cells showed (1) a reduced level of DNA repair synthesis when exposed to various carcinogenic *N*-oxides, (2) no unscheduled DNA synthesis following 6NQO and (3) a normal degree of DNA repair synthesis after treatment with MNNG. When the clone-forming capcity was examined the XP cells exhibited (1) an increased sensitivity to the various carcinogenic *N*-oxides, (2) no reduction in the clone formation following 6NQO and (3) a sensitivity virtually comparable to that of normal cells after treatment with MNNG. The results suggest a link between extent of DNA damage, level of DNA repair and degree of sensitivity in human cells exposed to various chemical carcinogens and mutagens. The UV-repair deficient XP cells are highly sensitive to chemical agents which induce DNA alterations that cannot be repaired by DNA repair synthesis.

2482 DEOXYRIBONUCLEOSIDE INCORPORATION DURING DNA REPAIR OF CARCINOGEN-INDUCED DAMAGE IN HUMAN DIPLOID FIBROBLASTS. *(E.)* Lieberman, M. W. (Natl. Cancer Inst., Bethesda, Md.) and M. C. Poirier. *Cancer Res* 33(9):2097-2103, 1973.

Incorporation of tritium-labeled deoxyribonucleosides during DNA repair was investigated in confluent human diploid fibroblasts (WI-38) in conditioned medium containing hydroxyurea. After damage with either of two proximate carcinogens, *N*-acetoxy-2-acetylaminofluorene or 7-bromomethylbenz(a)anthracene, incorporation of radioactivity into DNA increased 8- to 45-fold when any of the four labeled deoxyribonucleosides was used as a precursor. Similar results were obtained when UV light-damaged cells were allowed to repair in the presence of deoxyadenosine-^{3}H. Repair synthesis was also demonstrable when deoxyguanosine-^{3}H was used as precursor and bromodeoxyuridine was used as a density label for replicating DNA. Chromatography of nucleosides prepared by enzymatic digestion of repaired DNA's labeled with one of the four tritiated deoxyribonucleosides revealed that virtually all of the radioactivity was recoverable as the expected labeled product. Digestion studies of repaired DNA's with snake venom phosphodiesterase suggested that incorporation was not due to an artifact such as terminal addition. On the basis of these findings we conclude that, during DNA repair synthesis in human diploid cells, all four deoxyribonucleosides are incorporated. These results suggest that repair synthesis represents a faithful restoration of the damaged strand to its original state.

2483 EVIDENCE OF A GENETIC RELATIONSHIP BETWEEN SUSCEPTIBILITY TO 3-METHYLCHOLANTHRENE-INDUCED SUBCUTANEOUS TUMORS AND INDUCIBILITY OF ARYL HYDROCARBON HYDROXYLASE. *(E.)* Kouri, R. E. (Microbiological Assoc., Inc., Bethesda, Md.), H. Ratrie and C. E. Whitmire. *J Natl Cancer Inst* 51(1): 197-200, 1973.

A close correlation was found between susceptibility to 3-methylcholanthrene (MCA)-induced subcutaneous tumors and the genetically mediated presence of aryl hydrocarbon hydroxylase (AAH) induction in C57BL/6Cum (B6), DBA/2Cum (D2), $B6D2F_1$, and $D2B6F_1$ mice of both sexes. At 6 wk, animals were treated s.c. with 150 μg MCA. Typical fibrosarcomas developed at the site of MCA inoculation in some mice 7 months after MCA treatment. In a genetic system where AHH inducibility segregated as a single autosomal dominant gene, mice carrying the Ah^b (or inducible) allele were 5-10 times more sensitive to MCA tumorigenesis than were their noninducible (Ah^d/Ah^d) littermates. In backcross animals [(C57BL/6 X DBA/2) X DBA/2], 10 of 12 (83%) mice with tumors were AHH inducible. This was statistically significant ($P<0.025$) (the expected response was 50%) and clearly indicated a relationship between this enzyme system and MCA carcinogenesis.

2484 THE PRINCIPAL LIVER CARCINOGEN-PROTEIN CONJUGATE AFTER A SINGLE DOSE OF HEPATIC AZOCARCINOGEN. *(E.)* Sorof, S. (Fox Chase Ctr. Cancer Med. Sci., Philadelphia, Pa.) and E. M. Young. *Cancer Res* 33(9):2010-2013, 1973.

The question of whether or not there is a principal protein target of the azocarcinogens that is specific to the beginning events in liver carcinogenesis, one that would give rise to a principal conjugate other than slowly migrating $h_2\text{-}5S$ azoprotein, was examined. Two days after a single intragastric dose of the hepatic carcinogen, 3'-methyl-4-dimethylaminoazobenzene, the principal azoprotein in CFN rat liver cytosol was the $h_2\text{-}5S$ azoprotein. The principal species of azoprotein at two days was the same as that previously found after azocarcinogen was fed for 2.5 through 11 wk. Apparently, the same liver protein serves as the principal protein target of azocarcinogen throughout most of the duration of the liver carcinogenesis.

2485 CARCINOGENIC EFFECT OF DIPROPYLNITROSAMINE AND COMPOUNDS RELATED BY β-OXIDATION. *(E.)* Althoff, J. (U. Nebraska Med. Ctr., Omaha), F. W. Krueger and U. Mohr. *J Natl Cancer Inst* 51(1):287-288, 1973.

Dipropylnitrosamine injected s.c. once weekly for life (at five dose levels, 3.75-60 mg/kg body wt) into Syrian golden hamsters caused a 100% tumor incidence in the respiratory system. The first neoplasms were seen as early as 16 wk. The β-hydroxylated compound (2-hydroxypropyl) propylnitrosamine and the β-oxidized compound (2-oxopropyl) propylnitrosamine, administered by the same route and frequency, showed the same effects. With the β-hydroxylated compound, the first neoplasms in the lower respiratory system were seen 23 wk after treatment was begun; in addition, tumors originating in the liver were seen in 50% of the animals. With the β-oxidized compound, the first neoplasms appeared after 14 wk; tumors in the liver were found in 80% of the animals. Methylpropylnitrosamine injected s.c. once weekly for life lead to similar results. From these findings it was assumed that, in addition to α-hydroxylation, metabolism by β-hydroxylation might be associated with the carcinogenic effect of dipropylnitrosamine.

2486 POLYNUCLEOTIDE LIGASE FROM KIDNEY TUMORS IN RATS INDUCED BY N-NITROSODIMETHYLAMINE. *(E.)* Tsukada, E. (Fac. Pharm. Sci., Toyama U., Japan) and N. Ito. *J Biochem* 72(6):1299-1305, 1972.

Polynucleotide ligase activities were determined in normal BD IX rat kidney and kidney tumors induced by N-nitrosodimethylamine (500 ppm in diet for 2 wk). Ligase activities in the fraction precipitated at pH 5 with acetic acid and in the nuclear fraction of kidney tumors were five and three times higher, resp., than in the corresponding fractions of normal rat kidney. In normal kidney about 50% of the ligase activity was found in the nuclear fraction and 40% in the cytoplasm, while in tumor areas about 30% of the ligase activity was found in the nuclear fraction and 60% in the cytoplasm. On incubation with the ligase of nuclear extracts of tumors or normal kidneys, single strand breaks in DNA were joined and the sedimentation coefficient in alkali increased from 4S to 6-7S. Polynucleotide ligase activity in kidney tumors correlated with increased DNA synthesis, as measured by incorporation of ^{3}H-thymidine.

2487 INTERACTION BETWEEN LYSOSOMES AND CELL NUCLEUS IN DIETHYLNITROSAMINE CARCINOGENESIS. I. CHANGE IN SUBCELLULAR DISTRIBUTION OF THE LYSOSOMAL ENZYMES, ACID DEOXYRIBONUCLEASE AND β-D-GALACTOSIDASE, AFTER ACUTE DIETHYLNITROSAMINE INTOXICATION. *(Ger.)* Schulze, B. (Radiol. Clin. Polyclin., U. Marburg, Germany). *Z Krebsforsch* 79(4):241-248, 1973.

The subcellular distribution of acid deoxyribonuclease (DNAase II) and β-D-galactosidase was investigated in the livers of male inbred rats 24 and 48 hr after administration of diethylnitrosamine (DENA; 40 mg/kg i.p.). In addition, the release of these two enzymes from an enriched lysosome fraction after successive freezing and thawing was measured. Lysosome-bound β-D-galactosidase activity decreased significantly 24 and 48 hr after administration of DENA, but DNAase II activity remained unchanged. After freezing and thawing of the lysosome fraction, β-D-galactosidase activity decreased while DNAase II activity did not change appreciably. However, DNAase II activity increased significantly in both crude and purified nuclear fractions. It is suggested that this increase in DNAase II activity is a manifestation of reactive enzyme repair after DNA had been alkylated by DENA.

2488 THE INDUCTION OF TUMOURS IN THE GUINEA-PIG WITH METHYLCHOLANTHRENE AND DIETHYLNITROSAMINE AND THEIR PROPAGATION *IN VIVO* AND *IN VITRO*. *(E.)* Dale, M. M. (Dept. Pharmacology, U. Coll., London, England), G. C. Easty, R. Tchao, H. Desai and M. Andjargholi. *Br J Cancer* 27(6):445-450, 1973.

Tumor induction with diethylnitrosamine (DEN) and methylcholanthrene (MCA) was studied in three strains of guinea-pig. A DEN concentration of 80 μg/ml drinking water daily proved too toxic but reasonable survival was obtained with 20 μg/ml 3 times/wk in Hartley guinea-pigs and a local inbred strain. Heston Strain 13 guinea-pigs were particularly susceptible to the toxic effects of the diethylnitrosamine. In all three strains, 100% of the animals which survived the early toxic effects subsequently developed hepatomas, the mean time being 15 months. Methylcholanthrene was less toxic but more erratic as a carcinogen, the incidence of tumors in Hartley guinea-pigs varying from 18 to 100% in different experiments, the mean time of tumor development being ten months. Three transplantable hepatomas and three transplantable sarcomas were developed. The hepatomas are all predominantly hepatocellular carcinomas and the sarcomas comprise two liposarcomas and a fibrosarcoma. Successful short-term primary cultures of hepatomas, sarcomas and of normal liver tissues have been accomplished. Established cell lines in tissue culture have been developed from one cholangiocarcinoma from an outbred guinea-pig and one transplanted hepatocellular carcinoma from an inbred guinea-pig.

2489 THE EFFECTS OF AFLATOXIN B_1 AND AMINOAZODYE CARCINOGENS ON RAT AND MOUSE LIVER NUCLEAR ACID DEOXYRIBONUCLEASE. *(E.)* Pitout, M. J. (South African Med. Res. Council, Pretoria), J. J. van der Watt, P. G. Kempff, J. Dijsktra and J. C. Schabort. *Chem Biol Interact* 6(4):227-235, 1973.

The influence of a prolonged low level of administration to rats and mice of the potent hepatocarcinogen, B_1 (aflatoxin B_1) on liver nuclear acid DNAase was investigated. For comparison, the effects of 3'-methyl-*N*,*N*-dimethylaminoazobenzene (3'-MeDAB) (a potent hepatocarcinogen) and 2-methyl-*N*,*N*-dimethylaminoazaobenzene (2-MeDAB) (a weak hepatocarcinogen) was also studied. Rats were dosed weekly with B_1 equivalent to 1/5 of the LD_{50} and mice were fed a diet containing 2 mg/kg feed. Both MeDAB carcinogens were fed to rats at the rate of 0.64 g/kg feed. Groups from each treatment were killed at three weekly intervals until the appearance of neoplasia and the levels of acid DNAase in hepatic nuclei were assayed. Histological alterations were studied throughout the period of treatment. While 3'-MeDAB induced hepatic neoplasia within 26 wk, the first hepatocellular carcinomas were only detected after 40 wk exposure to B_1. Both B_1 and 3'-MeDAB rapidly induced transient increases in the specific activity of acid DNAase in rats. After several months, markedly elevated levels were found, which suddenly declined to normal levels immediately prior to malignant transformation. Histologically, it seemed that early toxic damage to the liver was associated with moderate increases, however, the observation that the appearance of regenerative nodules was accompanied by marked and sustained increases in the specific activity of DNAase appeared to contradict this assumption. In contrast, the weak carcinogen, 2-MeDAB induced only slight changes in the acid DNAase activity of rats. B_1 induced no changes in the mouse, which is resistant to its carcinogenic effects. The inability of 2-MeDAB to increase the activity of acid DNAase to the same levels as the metabolite of 3'-MeDAB is probably due to a much weaker binding of 2-MeDAB to DNA. Indications are that the activity of acid DNAase may be correlated with the morphological alterations occurring during the course of hepatic carcinogenesis. The importance of this study thus lies in the possible clinical application for the early detection of imminent hepatic neoplasia.

2490 THE ADENYL CYCLASE SYSTEM AND CARCINOGENESIS: DECREASED RESPONSIVENESS OF MOUSE EPIDERMIS TO ISOPROTERENOL AFTER 3,4-BENZPYRENE TREATMENT. *(E.)* Murray, A. W. (Sch. Biol. Sci., Flinders U. South Australia, Bedford Park) and A. K. Verma. *Biochem Biophys Res Commun* 54(1):69-75, 1973.

The isoproterenol-dependent accumulation of cyclic AMP in mouse epidermal cells following topical application of 3,4-benzpyrene, a carcinogenic polycyclic hydrocarbon, was examined. The concentration of cyclic AMP in untreated and acetone-treated epidermis was increased 9-11 fold 10 min after the i.p. injection of L-isoproterenol in female Swiss albino mice. The response to isoproterenol was unaffected 30 min after a single application of 3,4-benzpyrene in acetone, but was reduced to a 2.7 fold increase when tested after 24 hr. The increase in epidermal cyclic AMP following isoproterenol injection varied between 2.1 and 2.5 fold with repeated applications of 3,4-benzpyrene over periods of up to 55 days. The response to isoproterenol was unaffected by 1,2-benzpyrene, a non-carcinogenic polycyclic hydrocarbon. The simplest explanation of these results is that application of 3,4-benzpyrene to mouse skin causes a rapid change in the responsiveness of epidermal adenyl cyclase to isoproterenol. The effects are, however, not immediate and it is possible that a metabolite of 3,4-benzpyrene is responsible.

2491 A STUDY OF THE CARCINOGENICITY FOR SKIN OF A POLYPHENYL COOLANT. *(E.)* Henderson, J. S. (Dept. Pathol., U. Manitoba, Winnipeg) and J. L. Weeks. *Ind Med* 42(2):10-21, 1973.

BALB/c mice were skin-painted repeatedly with a hydrogenated terphenyl used as a coolant in nuclear reactors in order to assess to what extent it is an occupational hazard for industrial workers. The agent was applied both in an unirradiated and in an irradiated state, that is before and after use as a coolant, resp. Papillomas developed on only a few mice treated with irradiated or unirradiated coolant, the irradiated agent being somewhat more oncogenic than the unirradiated agent. Although it appears that the hazard to skin of workers exposed to hydrogenated tetraphenyl is not great, necessary protection and precautions must be observed.

2492 SENSITIVITY OF FEEDING TESTS IN DETECTING CARCINOGENIC PROPERTIES IN CHEMICALS: EXAMINATION OF 7,12-DIMETHYLBENZ[a]ANTHRACENE AND OXIDIZED LINOLEATE. *(E.)* Cutler, M. G. (Med. Sch., Birmingham, England) and R. Schneider. *Food Cosmet Toxicol* 11(3):443-457, 1973.

The sensitivity of a normal type of feeding test (Spiller's rat-cake diet) for detecting the carcinogenic risk of 7,12-dimethylbenz(a)anthracene (DMBA) to rats was compared with that of a test in which a high-fat diet containing 1% polyoxyethylene monostearate was administered and feeding of the carcinogen was begun at an earlier age. Female Wistar rats given 2.5 mg DMBA at age 6 wk and the high-fat diet showed an increased incidence of malignant mammary tumors and malignant tumors in other organs. No significant increase in malignant tumors occurred in females given a similar dose of DMBA and the normal diet. The high-fat diet also enabled the tumorigenic properties of oxidized linoleate to be detected more rapidly in female rats. An increased proportion of female rats had cervical sarcomas and mammary adenocarcinoma after addition of oxidized linoleate to their diet; in male rats the incidence of interstitial cell tumors of the testis was raised. Feeding of oxidized linoleate to CBA mice resulted in an increase in benign ovarian tumors and other benign tumors among the females. The possible risk to the population from the autoxidation of lipids is discussed.

2493 THE METABOLISM OF REPEATEDLY ADMINISTERED TRANS-4-DIMETHYLAMINOSTILBENE AND 4-DIMETHYLAMINOBIBENZYL. *(E.)* Neumann, H.-G. (Inst. Pharmacology Toxicology, U. Wurzburg, Germany). *Z Krebsforsch* 79(1):60-70, 1973.

The metabolic fates of initial doses of the carcinogen *trans*-4-dimethylaminostilbene (*trans*-DAS) and the noncarcinogen 4-dimethylaminobibenzyl (DABB) were compared with the metabolism of the thirteenth doses administered after six wk of twice weekly dosage by stomach tube to female rats. No significant changes were observed after prolonged feeding in either case. A slight increase in the urinary excretion of N-hydroxy-acetylaminostilbene glucuronide was found to occur. The pattern of unbound metabolites (5 hr) and the amount of bound metabolites in the liver and kidney as well as urinary and fecal metabolites excreted in 48 hr were analyzed. The results indicate that (1) in this particular experimental system the fate of a single dose is representative also of subsequent administrations, (2) major changes in enzyme activities in the liver are not involved in the process of tumor induction by *trans*-DAS, (3) carcinogenic doses of ear duct tumor-inducing *trans*-DAS appear to be less toxic to the liver than liver tumor-inducing 2-acetylaminofluorene in the rat.

2494 A NEW MODEL FOR INDUCING MALIGNANT OVARIAN TUMOURS IN RATS. *(E.)* Hilfrich, J. (Hannover Med. Coll., West Germany). *Br J Cancer* 28(1):46-54, 1973.

After the implantation of ovarian tissue into the spleen of gonadectomized female Sprague-Dawley rats (splenic ovary), luteomata and later benign granulosa or granulosa-theca cell tumors develop. Treatment of these rats with 7,12 dimethylbenz(a)anthracene (DMBA), given i.v. 2 mg/kg body wt weekly, total dosage 40 mg/kg, immediately and especially 25 wk after implantation of ovarian tissue into the spleen, led to malignant, partially metastasizing granulosa, and in one case theca cell tumors, 16-46 wk after beginning the carcinogen treatment. No malignant neoplastic growth was seen when diethylnitrosamine (DEN), 20 mg/kg once weekly for life, was injected s.c. immediately or 25 wk after implanting ovarian tissue. Since the normal, non-implanted rat ovary was not affected by DMBA treatment the malignant transformation of splenic ovaries in the respective experimental groups may be related to the increased stimulation by pituitary gonadotrophins and formation of luteoma or beginning granulosa and theca cell proliferations. The similarity between the chemical structure of the polycyclic hydrocarbon and steroid hormones and the similarity of their biological effects may interfere with the metabolism of luteinized cells.

2495 THE EFFECT OF URETHAN ON THE INCORPORATION OF THYMIDINE-^{3}H INTO DNA AND THE ACTIVITIES OF SOME ENZYMES REQUIRED FOR DNA BIOSYNTHESIS IN RAT REGENERATING LIVER. *(E.)* Hwang, K. M. (Yale U. Sch. Med., New Haven, Conn.), S. A. Murphree and A. C. Sartorelli. *Cancer Res* 33(9):2149-2155, 1973.

Urethan, a hepatocarcinogen in adult regenerating liver, was administered to rats at various intervals after partial hepatectomy to determine the effect of this drug on (a) the pattern of DNA replication and (b) the induction by partial hepatectomy of some enzymes required for DNA synthesis. A single i.p. dose of urethan, 1 mg/g of body weight, was injected into rats at either 0.5, 12, or 24 hr after surgery. When urethan was given 0.5 hr after partial hepatectomy, the initiation of DNA synthesis was delayed by 6 hr, followed by greater uptake of thymidine-^{3}H into nuclear DNA than into that of untreated regenerating liver. When urethan was given 12 hr after partial hepatectomy, the first wave of DNA synthesis was suppressed by 50 to 60%; the second cycle of DNA replication was essentially unaffected. Administration of urethan 24 hr postsurgery resulted in maximum inhibition at 14 hr after treatment, the incorporation of thymidine-^{3}H during the second wave of DNA replication occurred within a shorter period of time than that of the control. The induction of certain enzymes linked to DNA replication during liver regeneration was affected by the urethan in a similar manner. Ribonucleoside diphosphate reductase and thymidine kinase were sensitive to urethan treatment, while DNA polymerase activity was insensitive. Other enzymes not tightly coupled to DNA synthesis in regenerating liver, such as adenine and hypoxanthine-guanine phosphoribosyltransferases, were not affected by the carcinogen. The results indicate that urethan selectively inhibits the induction by partial hepatectomy of enzymes necessary for DNA synthesis.

2496 INHIBITION OF CARCINOGENESIS IN THE LIVERS OF RATS TREATED WITH DIETHYLNITROSAMINE BY INTRAPERITONEAL PRETREATMENT WITH HOMOLOGOUS THYMUS. *(Ger.)* Weissberg, M. (Inst. Cancer Res., U. Vienna, Austria). *Exp Pathol (Jena)* 8:58-71, 1973.

Injections of homologous thymus, suspended in physiological saline, were administered i.p. three times at weekly intervals to female Sprague-Dawley rats. Starting one week after the last injection, diethylnitrosamine (DENA) was administered in their drinking water until they had ingested 100 or 200 mg/kg. Controls were rats given the same doses of DENA without thymus pretreatment. Of 30 controls given 200 mg/kg DENA, 10 developed carcinomas, whereas only 3 of the 33 rats pretreated with thymus had tumors. Hepatocellular carcinomas developed in 3 controls given 100 mg/kg DENA but in none of thymus-pretreated rats given this dose of DENA. Examination of the livers at various intervals after DENA treatment showed that liver degeneration and foci of atrophy were more common and extensive in controls than in rats pretreated with thymus; their absence was striking in those given the lower dose of DENA. Precancerous changes, characterized by increases in glycogen storage and RNA and protein synthesis, were also more common in controls. Two types of hepatocellular carcinomas were found: small cell carcinomas which infiltrated the sinusoids and invaded the portal system and polyploid, polymorphocellular carcinomas which were well supplied with blood vessels. Biliary tract carcinomas, associated with periportal cirrhosis, occurred in 2 cases.

2497 HOST-MEDIATED *IN VIVO-IN VITRO* ASSAY FOR CHEMICAL CARCINOGENESIS. *(E.)* DiPaolo, J. A. (Natl. Cancer Inst., Bethesda, Md.), R. L. Nelson, P. J. Donovan and C. H. Evans. *Arch Pathol* 95(6):380-385, 1973.

A host-mediated *in vivo-in vitro* combination bioassay for identification of potential mammalian cell chemical carcinogens is described. The procedure involves intraperitoneal injection of drug into pregnant hamsters followed 48 to 72 hr later by excision, mincing and culturing of the embryos. Secondary cultures are then evaluated according to conventional *in vitro* methods for the presence of transformed cells and chromosome changes. Transformation is verified by *in vivo* tumor production following inoculation of morphologically changed cells into x-irradiated weanling hamsters. All 12 chemical carcinogens tested resulted in morphologically transformed cultures after plating the secondary cultures on irradiated feeder layers or after several *in vitro* passages. None of the five noncarcinogens tested resulted in transformation. Cell lines derived from tumors induced in hamsters by s.c. inoculation of chemically transformed secondary cell lines mimicked the original transformed lines in morphologic and growth characteristics. Transformed cell lines often showed an increased number of telocentric chromosomes in the 16 to 19 group range and a loss of one of the small metacentrics from pair number 20. This *in vivo-in vitro* method had the advantage of reducing false negatives which might occur *in vitro* because of lack of metabolic activation.

2498 AGE-ADJUSTED MORTALITY RATES FOR LUNG CANCER PLOTTED AGAINST NATIONAL CIGARETTE CONSUMPTION. *(E.)* Clemmesen, J. (Danish Cancer Registry, Copenhagen) and A. Nielsen. *Acta Pathol Microbiol Scand* 81(1):95-96, 1973.

Mortality rates for lung cancer among men in various countries in 1950 were plotted against national figures for per capita cigarette consumption 20 yr earlier. Under these conditions, the correlation between the two values was R = .89. When mortality rates were aged-adjusted to a European standard population, the value for R was .92.

2499 RELATIVE DNA DAMAGE INDUCED IN CULTURED HUMAN SKIN FIBROBLASTS BY EXPOSURE TO THE PRECARCINOGEN 2-ACETYLAMINOFLUORENE, THE PROXIMATE CARCINOGEN *N*-HYDROXY-2-ACETYLAMINOFLUORENE, AND THE ULTIMATE CARCINOGEN *N*-ACETOXY-2-ACETYLAMINOFLUORENE. *(E.)* Laishes, B. A. (U. British Columbia, Vancouver, Canada) and H. F. Stich. *Can J Biochem* 51(7):990-994, 1973.

The relative DNA damage induced in human skin fibroblasts by exposure to 2-acetylaminofluorene (AAF), *N*-hydroxy-2-acetylaminofluorene (*N*-hydroxy-AAF), and *N*-acetoxy-2-acetylaminofluorene (*N*-acetoxy-AAF) was demonstrated by measuring the sedimentation of DNA through alkaline sucrose gradients. DNA damage was induced by *N*-acetoxy-AAF at concentrations as low as 2×10^{-5} M. The precarcinogen AAF and the proximate carcinogen *N*-hydroxy-AFF did not induce measureable DNA damage. The extent of damage increased with the applied concentration of *N*-acetoxy-AFF and the carcinogen 4-nitroquinoline-1-oxide. Measurement of DNA damage induced by 30 min exposure to 4×10^{-7} M 4-nitroquinoline-1-oxide showed that the technique used in this study is more sensitive than those reported previously.

2500 A METHOD FOR MEASURING DNA DAMAGE AND REPAIR IN THE LIVER *IN VIVO*. *(E.)* Cox, R. (Temple U. Sch. Med., Philadelphia, Pa.), I. Damjanov, S. E. Abanobi and D. S. Sarma. *Cancer Res* 33(9):2114-2121, 1973.

Alkaline sucrose gradients were used to study the induction of single-strand breaks in and repair of white male Wistar rat liver DNA *in vivo*. Liver DNA was labeled by injecting thymidine-methyl-^{3}H during regeneration after partial hepatectomy. DNA was isolated from the organ in such a way as to minimize the induction of breaks due to handling or enzymatic activity. Different methods of preparing high-molecular-weight liver DNA were compared. Best results were obtained by squashing the liver in an ethylene-diaminetetraacetic acid-sodium chloride buffer. Using this squash technique and alkaline sucrose gradients, high-molecular-weight DNA was regularly obtained. This DNA behaved like single-stranded DNA on hydroxyapatite columns and contained no detectable contamination of protein or RNA. It was demonstrated by the method described that hepatic DNA damage was produced within 4 hr by the i.p. administration of methyl methanesulfonate at a dose of 120 mg/kg and that the rat can repair such hepatic DNA damage *in vivo* within 48 hr.

2501 ABNORMAL CHOLESTEROL UPTAKE, STORAGE, AND SYNTHESIS IN THE LIVERS OF 2-ACETYLAMINOFLUORENE-FED RATS. *(E.)* Horton, B. J. (U. Wisconsin, Madison), J. D. Horton and H. C. Pitot. *Cancer Res* 33(6):1301-1305, 1973.

Livers from male Sprague-Dawley rats were tested for rate of cholesterol synthesis and uptake of a p.o. dose of cholesterol-^{3}H after the rats were fed 0.06% 2-acetylaminofluorene (AFF) for 1 or 2 wk. For 2 days prior to the assay, all rats also received 5% cholesterol in the diet. In the controls, the high-cholesterol diet caused almost a total inhibition of cholesterol synthesis, but after AAF treatment the levels of cholesterol synthesis were significantly higher, with considerable variation between individual rats. Uptake of cholesterol-^{3}H given p.o. was markedly lower in the AAF group and was also variable, but there was no correlation with sterol synthesis, indicating that blockage of cholesterol uptake is not the critical factor in loss of control. Liver cholesterol levels in the AAF-fed rats were significantly lower than those in the controls. There was also a highly significant inverse correlation between liver cholesterol levels and sterol synthesis. Therefore, the lack of regulation of cholesterol synthesis induced by AAF seems to be a result of a defect in intracellular binding or storage of cholesterol rather than defective uptake. The loss of this storage mechanism may be a vital characteristic of the carcinogenic process.

2502 EFFECTS OF ANTIFERTILITY DRUG ENOVID IN FIVE STRAINS OF MICE, WITH PARTICULAR REGARD TO CARCINOGENESIS. *(E.)* Heston, W. E. (Natl. Cancer Inst., Bethesda, Md.), G. Vlahakis and B. Desmukes. *J Natl Cancer Inst* 51(1):209-224, 1973.

The antifertility drug, Enovid (norethynodrel and mestranol, 98.5:1.5), was tested for possible carcinogenicity in female mice of five specially selected strains: BALB/c, C3H, C3HfB, A, and C57BL. The drug was fed three dose levels: 5 μg Enovid/g food that did not prevent reproduction, 10 μg/g that prevented some females from reproducing, and 20 μg/g that prevented all females from reproducing. The results emphasized that inbred strains of mice differed in their response to Enovid. Enovid treatment reduced the gain in weight in all strains. It had no effect on lifespan in 1 strain, but in 2 strains lifespan was lengthened and in 2 shortened because of the effect on tumors or other lesions. Cervical and vaginal lesions showing invasion of the epithelium into the stroma with varying degrees of progression were observed but limited, with few exceptions, to the BALB/c females. They occurred in control and treated females of this strain but with a higher incidence and possibly further progression in the group treated with the highest dose of Enovid. None of these lesions were observed grossly as tumors, all being found on histologic examination, and none had extended to organs and tissues beyond the vaginal wall or showed any evidence of having metastasized. Enovid treatment of these dose levels did not increase the occurrence of ovarian tumors, even in the highly susceptible strains C3H and C3Hfb, or of mammary gland tumors in any strain. In fact, in the highly susceptible strains C3H mammary tumors were somewhat inhibited in the treated females. In strain C3HfB there was some inhibition of hepatomas by the treatment, probably related to the reduction in growth rate, and in BALB/c some inhibition of adrenocortical adenomas. Chromophobe adenomas of the hypophysis were significantly increased in old C57BL females treated with the highest dose of Enovid. Many of these were active in stimulating the mammary glands of these old females and caused the females to have to be necropsied.

2503 ENZYMATIC N-ACETYLATION OF CARCINOGENIC AROMATIC AMINES BY LIVER CYTOSOL OF SPECIES DISPLAYING DIFFERENT ORGAN SUSCEPTIBILITIES. *(E.)* Lower, G. M. Jr. (U. Wisconsin Med. Sch., Madison) and G. T. Bryan. *Biochem Pharmacol* 22(13):1581-1588, 1973.

One of the major routes for the metabolism of carcinogenic arylamines involves the transfer of the acetyl moiety of acetyl-CoA to the arylamines, as catalyzed by N-acetyl-transferase enzyme systems present in the soluble portion of mammalian liver (liver cytosol). The present report is a comparative examination *in vitro* of the ability of various species to carry out the N-acetylation of 2-aminofluorene, 4-aminobiphenyl and 2-aminonaphthalene in an attempt to clarify the possible role of N-acetylation as a determinant of organ susceptibility to these carcinogens. Liver cytosol, containing the N-acetyl-transferase system free of competing microsomal N-hydroxylation enzymes, was obtained in the form of supernatants from high speed centrifugation of liver homogenates. Assays were carried out by incubation of arylamine substrate with ^{14}C-labeled acetyl-CoA and liver cytosol, followed by benzene extraction of the product and measurement by liquid scintillation technique. In hamsters, guinea pigs, mice and rats, 2-aminofluorene was the superior substrate, followed in order by 4-aminobiphenyl and 2-aminonaphthalene. Highest enzyme activity was present in liver cytosol from the hamster, followed in order by cytosols from the guinea pig, mouse and rat. In contrast, dog liver cytosol was inactive. Administration of carcinogenic arylamines to dogs was shown in previous work to result in the formation of only urinary bladder tumors, while administration of carcinogenic arylacetamides resulted in the formation of both urinary bladder tumors and hepatomas. These considerations suggest that N-acetylation is not required for carcinogenesis of the urinary bladder and that expression of urinary bladder or liver carcinogenicity by arylamines and arylacetamides in a given experimental system may depend on the quantitative ability of the species being studied to utilize pathways for the interconversion of arylamine and arylacetamide derivatives.

2504 EFFECT OF ACETANILIDE AND p-HYDROXYACETANILIDE ON THE CARCINOGENICITY OF N-2-FLUORENYLACETAMIDE AND N-HYDROXY-N-2-FLUORENYLACETAMIDE IN MICE, HAMSTERS, AND FEMALE RATS.

(E.) Weisburger, J. H. (Natl. Cancer Inst., Bethesda, Md.) E. K. Weisburger, R. M. Madison, M. L. Wenk, and D. S. Klein. *J Natl Cancer Inst* 51(1):235-240, 1973.

Simultaneous dietary administration of acetanilide (AA) or p-hydroxyacetanilide (p-OHAA) with the carcinogen N-2-fluorenylacetamide (FAA) inhibited mammary tumor formation in female Sprague-Dawley-derived rats. p-OHAA failed to inhibit the overall mammary tumor incidence from N-hydroxy-2-fluorenylacetamide (N-OHFAA) although fewer animals had multiple tumors. Non-neoplastic liver lesions were present in rats fed either FAA or N-OHFAA; this lesion was completely inhibited in the groups receiving AA or p-OHAA supplements. In male NIH general purpose mice on similar regimens, AA was more effective than p-OHAA in reducing the liver cancer incidence from FAA. Neither AA nor p-OHAA altered the yield of bladder tumors induced by FAA in mice. p-OHAA decreased the bladder but not the liver tumor incidence caused by N-OHFAA in male mice. Either AA or p-OHAA reduced the number of liver tumors in female mice fed FAA, and p-OHAA had a similar action with N-OHFAA. Either AA or p-OHAA inhibited liver tumors in male and to a lesser extent, female Syrian golden hamsters fed FAA, and p-OHAA did likewise with N-OHFAA. The incidence of gastric papillomas and carcinomas from N-OHFAA was not markedly decreased by p-OHAA.

2505 SYNERGISTIC EFFECT OF FERRIC OXIDE ON DIMETHYLNITROSAMINE CARCINOGENESIS IN THE SYRIAN GOLDEN HAMSTER.

(E.) Stenback, F. (U. Nebraska Med. Ctr., Omaha), A. Ferrero, R. Montesano and P. Shubik. *Z Krebsforsch* 79(1):31-38, 1973.

The carcinogenic effect of dimethylnitrosamine (DMN) injected s.c. in Syrian golden hamsters as well as the synergistic effect of intratracheal instillations of ferric oxide were studied. DMN alone induced cavernous hemangiomas, hemangioendotheliomas, hemangioendotheliosarcomas, as well as one hepatoma of the liver and one papilloma of the forestomach, but no tumors of the respiratory system. When ferric oxide was given in addition to DMN, liver tumors of vacular and biliary origin were seen, in addition to tumors of the nasal cavity, neuroepithelial tumors and one adenoma. It was concluded that s.c. injected DMN is a powerful hepatic carcinogen for the Syrian golden hamster, producing liver tumors of vascular, bile duct as well as liver cell origin. The lack of tumorigenic effect in the respiratory system was considered to be due to the lack of metabolism of DMN in the lung.

2506 PATTERNS OF DAMAGE AND REPAIR OF LIVER DNA INDUCED BY CARCINOGENIC METHYLATING AGENTS *IN VIVO*.

(E.) Damjanov, I. (Temple U. Sch. Med., Philadelphia, Pa.), R. Cox, D. S. R. Sarma and E. Farber. *Cancer Res* 33(9):2122-2128, 1973.

Single-strand breaks induced by methylating agents in liver DNA of white male Wistar rats and their rejoining were studied *in vivo* by the use of alkaline sucrose gradients. DNA breaks were induced by the i.p. injection of single doses of dimethylnitrosamine (DMN), methylazoxymethanol acetate (MAM), N-methyl-N-nitrosourea (MNU), and N-methyl-N-nitrosourethan (MNUT) dissolved or diluted with 0.9% NaCl solution. The degree of damage was dependent on the dose of the substance given. DMN and MAM are comparably efficient in inducing marked single-strand breaks of liver DNA in small doses. MNU, although noncarcinogenic for liver, causes single-strand breaks of liver DNA, while N-methyl-N-nitrosourethan causes less damage even at toxic doses. The repair of the damage to DNA induced by MNU was complete in the first week while that induced by DMN and MAM was still not complete within 14 days after administration of the compound. Thus, the repair of the damage induced by hepatocarcinogens (DMN and MAM) seemed to be slower than that with the methylating agents not carcinogenic for the liver (MNU and methyl methanesulfonate).

2507 THE TOXICOLOGY OF DIELDRIN (HEOD). I. LONG-TERM ORAL TOXICITY STUDIES IN MICE.

(E.) Walker, A. I. T. (Tunstall Lab., Sittingbourne, Kent, England), E. Thorpe and D. E. Stevenson. *Food Cosmet Toxicol* 11(3):415-432, 1972.

In five yr long-term oral toxicity experiments, dieldrin (purity > 99%), p,p'-DDT (purity > 99.5%), and a combination of these two compounds were fed, at various dietary concentrations, to CF1 mice bred and maintained under specific-pathogen-free conditions. The interaction between the method of diet sterilization and the development of liver changes, and the behavior of the liver lesion after restoration to the basal diet were also studied. In a positive control group fed 600 ppm 4-amino-2,3-dimethylazobenzene (ADAB), 61% of the males developed sarcomas while the females showed hepatic-cell tumors together with areas of fibroplasia and bile-duct proliferation. No signs of ill-health were associated with dieldrin at levels of 10 ppm or below or with DDT at levels of 100 ppm or less. Mice fed dieldrin showed liver enlargement 9 months after treatment with 10 ppm, 19 months after 5 ppm, and 23 months after 2.5 ppm. In all 3 groups, liver enlargement was associated with a decrease in survival rate. At intakes of 1.25 and 1 ppm dieldrin, no enlargement was found and survival was not affected. With DDT, liver enlargement was detected in the 100-ppm group after 16 months and morbidity was increased. In the 50-ppm group, enlargement occurred after 24 months, but morbidity was not affected. The method of diet sterilization did not influence the development of liver lesions. Two types of liver lesions were found in all groups: 1) a simple nodular growth of liver parenchymal cells, and 2) areas of papilliform and adenoid growth of tumor cells, sometimes accompanied by metastases to the lungs. Often the two types of lesions were contiguous. An increased incidence of liver lesions was noted in the treated groups. The incidence of other tumors was increased in ADAB-treated mice but not in the dieldrin or DDT groups.

2508 THE TOXICOLOGY OF DIELDRIN (HEOD). II. COMPARATIVE LONG-TERM ORAL TOXICITY STUDIES IN MICE WITH DIELDRIN, DDT, PHENOBARBITONE, β-BHC AND γ-BHC. *(E.)* Thorpe, E. (Tunstall Lab, Sittingbourne, Kent, England) and A. I. Walker. *Food Cosmet Toxicol* 11(3):433-442, 1973.

A 2 yr oral toxicity study is described in which dieldrin (purity > 99%), p,p' DDT (purity > 99.5%), β-hexachlorocyclohexane (β-BHC, purity > 99%), γ-hexachlorocyclohexane (γ-BHC, purity > 99.5%), and phenobarbital (purity not < 98%) were fed to CF1 mice at different concentrations. The development of liver changes induced by these compounds known to produce hepatic microsomal-enzyme induction was studied. Liver enlargement was detected after 50 wk in males and females given dieldrin (10 ppm) or γ-BHC (400 ppm) and in females fed DDT (100 ppm) or β-HBC (200 ppm), after 60 wk in males given β-BHC (200 ppm) and after 68 wk, in males given DDT (100 ppm) and in both sexes given phenobarbital (500 ppm). Liver lesions observed in all groups were classified as hyperplastic foci, type (a) a simple nodular growth of parenchymal cells and type (b) with areas of papilliform and adenoid growth of tumor cells often associated with lung metastases. The problem of the classification and diagnosis of type (a) and (b) growths is examined, as well as the biological significance of the induction of liver tumors and the importance of liver cell hyperplasia.

2509 FILAMENTOUS FORMS OF ENVELOPED A PARTICLES IN CELL CULTURES FROM CHEMICALLY INDUCED RAT HEPATOMAS. *(E.)* Orenstein, J. M. (Coll. Physicians Surg., Columbia U., New York, N.Y.) and I. B. Weinstein. *Cancer Res* 33(9):1998-2004, 1973.

A small number of filamentous or cylindrical forms of enveloped A particles were observed in cell cultures established from either a primary or a transplanted rat hepatoma induced *in vivo* by aromatic amine carcinogens. These same cells produced typical budding particles, developed A particles, C particles, and intracisternal A particles. When these cultures were treated with 5-bromo-2-deoxyuridine (BUdR) and/or dimethyl sulfoxide (DMSO) a striking increase in the total number and percentage of filamentous forms was observed. The length of the filamentous forms was also markedly enhanced by drug treatment, and filaments as long as 1.85 μm were observed. The atypical forms were usually seen budding singly from the cell membrane. Occasionally, however, clusters of these forms were seen associated with the plasma membrane of cells in cultures treated with BUdR and/or DMSO. Our findings suggest that BUdR and DMSO, while increasing total virus production in hepatoma cultures, can induce marked aberrations in virus assembly and maturation. It is not clear whether the increase in filamentous forms is due to a general toxic effect of BUdR and DMSO on the cell, an alteration in the expression of genetic information specifically related to virus maturation, or a direct toxic effect on certain regions of the cell surface. The occasional occurrence of clusters of filamentous forms at specific regions of the cell surface lends support to the notion that local cell surface factors favor their formation.

2510 *N*-OXIDATION OF CERTAIN AROMATIC AMINES, ACETAMIDES, AND NITRO COMPOUNDS BY MONKEYS AND DOGS. *(E.)* Radomski, J. L. (U. Miami Sch. Med., Fla.), G. M. Conzelman, Jr., A. A. Rey and E. Brill. *J Natl Cancer Inst* 50(4):989-995, 1973.

To test the hypothesis than *N*-oxidized metabolites of aromatic amines are essential to the carcinogenic process dogs and rhesus monkeys were injected with agents including 4-aminobiphenyl and 2-naphthylamine and their urine was examined by gas chromatography for nonacetylated *N*-oxidation products. *N*-hydroxylation occurs in monkeys given 2-naphthylamine and results in the excretion of *N*-hydroxy-2-naphthylamine in the urine as it does in the dog. Monkeys and dogs given 4-aminobiphenyl excreted *N*-hydroxy-4-aminobiphenyl. Excretion of acetylated *N*-hydroxy metabolites occurred in the monkeys, but not in dogs given these amines. Dogs given 2-acetylaminonaphthalene, a noncarcinogen, excreted *N*-hydroxy-2-acetylaminonaphthalene, but no *N*-hydroxy-2-naphthylamine or 2-nitrosonaphthalene. These observations are consistent with the hypothesis that the nonacetylated *N*-hydroxy metabolites of the aromatic amines are the active urinary carcinogens. The relatively weak bladder carcinogen, 4-nitrobiphenyl, was metabolized to small amounts of *N*-hydroxy-4-aminobiphenyl and 4-nitrosobiphenyl in both monkeys and dogs. Comparable *N*-oxidation products were found in the urine of monkeys, but not in that of dogs given 2-nitronaphthalene.

2511 METABOLISM OF NITROSAMINES *IN VIVO*. II. ON THE METHYLATION OF NUCLEIC ACIDS BY DI-n-ALKYL-NITROSAMINES *IN VIVO*, CAUSED BY β-OXIDATION: THE INCREASED FORMATION OF 7-METHYL-GUANINE AFTER APPLICATION OF β-HYDROXYPROPYL-PROPYL-NITROSAMINE COMPARED TO THAT AFTER APPLICATION OF DI-n-PROPYL-NITROSAMINE. *(E.)* Kruger, F. W. (Inst. Exp. Toxicology Chemotherapy, Heidelberg, Germany). *Z Krebsforsch* 79 (2):90-97, 1973.

The syntheses of 1-(^{14}C)- and 2-(^{14}C)-β-hydroxypropyl-propyl-nitrosamine (HHPN) were described. Male Sprague-Dawley rats were inoculated i.p. with 438 mg, 2000 Ci/kg of each compound. Formation of labeled 7-methyl-guanine in the RNA and DNA of liver occurred only when the 1-(^{14}C)-labeled compound was injected. The amount of (^{14}C)-labeled 7-methyl-guanine found was 50 times higher than that found after administration of 1-(^{14}C)-di-n-propyl-nitrosamine. These data support the hypothesis that di-n-alkyl-nitrosamines with more than two carbons per alkyl group are degraded to methylating agents by β-hydroxylation. In contrast to results obtained in experiments using di-n-alkyl-nitrosamines not hydroxylated in the β-position, no labeled alkylated base other than 7-methyl-guanine was found in the present work.

2512 BASAL PROLACTIN BLOOD LEVELS IN THREE STRAINS OF RAT WITH DIFFERING INCIDENCE OF 7,12-DIMETHYLBENZ(a)ANTHRACENE INDUCED MAMMARY TUMOURS.

(E.) Boyns, A. R. (Welsh Natl. Sch. Med., Cardiff), R. Buchan, E. N. Cole, A. P. M. Forrest and K. Griffiths. *Eur J Cancer* 9(3):169-171, 1973.

Plasma prolactin in strains of rats with differing incidence of tumor induction was measured in the diestrous phase of the estrous cycle to determine if a relationship exists between circulating prolactin levels and tumor incidence. Two strains of Sprague-Dawley rats and one strain of Long-Evans rats were examined. At 50-days-old, the three groups of rats were given 5 mg of 7,12-dimethylbenz(a)-anthracene by injection into a dorsal tail vein. At 250 days of age, a post-mortem examination showed that there was a much higher incidence of tumors in one of the Sprague-Dawley strains than in the other two rat strains. The concentration of prolactin in the serum of this high-incidence Sprague-Dawley strain was significantly higher than that in the other groups. The data suggest that the high incidence of tumors in the one rat strain and the low incidence in the other two may be related to different concentrations of circulating pituitary prolactin.

2513 DEUTERIUM ISOTOPE EFFECT ON THE CARCINOGENICITY OF DIMETHYLNITROSAMINE IN RAT LIVER.

(E.) Keefer, L. K. (U. Nebraska Med. Ctr., Omaha), W. Lijinsky and H. Garcia. *J Natl Cancer Inst* 51(1): 299-302, 1973.

The effect of complete deuteration of dimethylnitrosamine on its carcinogenicity in the liver of rats was examined. Liver tumor incidence was 26% in male MRC rats receiving 5 ppm dimethylnitrosamine in their drinking water for 30 wk. A significantly lower liver tumor incidence of 3% was observed in a second group of rats receiving fully deuterated dimethylnitrosamine under identical conditions. The data also indicate the great carcinogenic potency of dimethylnitrosamine. A total average dose of only 15 mg - approximately one drop - sufficed to produce liver tumors in 8 of the 30 animals receiving this compound.

2514 MULTICARCINOGENICITY OF N-NITROSOBUTYLUREA IN MICE AND RATS AS DEMONSTRATED BY HOST CONDITIONING.

(E.) Yokoro, K. (Res. Inst., Nuclear Med. Biol., Hiroshima U., Japan), S. Takizawa, Y. Kawamura, M. Nakano and A. Kawase. *Gann* 64(2):193-196, 1973.

Continuous oral administration of N-nitrosobutylurea in drinking water to rats induces neoplastic lesions in lymphoreticular tissue, mammary gland, brain, kidney, gastrointestinal tract and soft tissue; for mice, the carcinogen is prominently leukemogenic under similar conditions. To test the effect of physiological state of the host on the neoplastic response to the carcinogen, the authors investigated the effects of thymectomy, ovariectomy and injection of CCl_4 on the nature of the tumors produced by subsequent administration of N-nitrosobutylurea. Female mice subjected to thymectomy and/or ovariectomy were operated on at 4 wk of age, and 1 mg carcinogen per day, dissolved in drinking water, was given for 180 days. Male mice given CCl_4 were injected s.c. with 0.1 ml compound, 50 % in sesame oil, at 7-10 wk of age, and a single dose of 10 or 20 mg carcinogen was given by intragastric intubation 24 hr after injection of CCl_4. Female W/Fu rats subjected to ovariectomy were subgrouped, operated on at 6, 12, 22, or 33 wk of age and subsequently given 5.0 mg carcinogen per day for 180 days. Intact mice given carcinogen all developed thymic lymphoma, while mice previously thymectomized showed only 13.3 % leukemia and simultaneously showed 80 % multiple lung adenomas and 66.6 % multiple mammary adenocarcinomas and adenocanthomas. N-nitrosobutylurea-induced leukemogenesis inhibited by thymectomy was restored by ovariectomy, suggesting that stimulation of the reproductive system might be mediated by the ovary or by the anterior pituitary-ovary axis. Ovariectomy caused similar results in rats as shown by the finding that while administration of N-nitrosobutylurea elicited mammary tumors but no leukemia in intact rats, removal of the ovary before or during carcinogen treatment brought about the development of various types of leukemia and the abolition of development of mammary tumors. Mice given single doses of carcinogen or of CCl_4 alone showed no tumorigenesis, while the combined treatment, 24 hr apart, elicited hepatomas in 30 % of the mice. The authors conclude that evaluation of the carcinogenic potential of an agent should be determined by careful study in the test system of various physiological states.

2515 SUPPRESSED ACTIVITY OF T-CELL IN MICE TREATED WITH 7,12-DIMETHYLBENZ(a)-ANTHRACENE.

(E.) Yamashita, U., (Osaka U. Med. Sch., Japan), Y. Matsuoka and M. Kitagawa. *Gann* 64(3): 317-320, 1973.

The effect of 7,12-dimethylbenz(a)anthracene (DMBA) on B (bone marrow-derived) or T (thymus-derived) cell lineage was examined using hapten-carrier conjugate as the antigen, in an effort to determine the cellular mechanism of DMBA-induced immunosuppression. A group of ddO albino mice, 6-wk old, was given 1 mg of DMBA in 0.2 ml of olive oil by s.c. injection and 7 days later immunized i.p. with 100 μg of 2,4-dinitrophenyl(DNP)-Taka-amylase-A. Control mice received 0.2 ml of olive oil and the antigen. The anti-DNP response of DMBA-treated mice was almost completely suppressed, whereas the control mice responded distinctly and the titers kept increasing during a 6-wk period. When DMBA was administered simultaneously with the antigen, much less suppressive effect was observed on the anti-DNP response. The effect of DMBA on B-cells and on T-cells was also assessed separately by estimating B-cell activity as hapten-primed antibody forming cell precursors (AFCP) and T-cell activity as carrier-primed helper cells in the system of adoptive transfer of these cells into 600 R X-irradiated recipients. The development of both cells was markedly depressed by the DMBA treatment. It seems probable that DMBA may reveal its immuno-suppressive effect in a bimodal way; one is relatively short-acting and directed to the B-cell lineage and the other is long-lasting and directed to T-cell lineage.

2516 1-NITROSO-5,6-DIHYDROURACIL: INDUCTION OF LIVER CELL CARCINOMAS AND KIDNEY ADENOMAS IN THE RAT.

(E.) Mirvish, S. S. (U. Nebraska Med. Ctr., Omaha) and H. Garcia. *Z Krebsforsch* 79(4):304-308, 1973.

1-Nitroso-5,6-dihydrouracil (NO-DHU) was synthesized by nitrosation of 5,6-dihydrouracil (DHU) and its carcinogenic effect on MRC Wistar rats was studied. The LD_{50} was 850 mg/kg by i.p. injection. Long-term administration of 450 mg/liter citrate buffer as drinking water induced liver cell carcinomas in 98% of the rats in 21-34 wk. Also, 19% of the rats showed kidney adenomas. The organ specificity might be connected with the fact that DHU is a normal metabolite related to uracil. NO-DHU could have a selective affinity for nucleic acids or for a liver enzyme involved in DHU or uracil metabolism. The kinetics of nitrosation of DHU showed a rather low rate constant of $1.0\ M^{-2}\ min^{-1}$ at 25C.

2517 FUNCTIONAL CHARACTERISTICS OF MAST CELLS ASSOCIATED WITH RAT MAMMARY TUMORS INDUCED BY 7,12-DIMETHYLBENZ[a]ANTHRACENE.

(E.) Combs, J. W. (Hershey Med. Ctr., Pa.) and D. M. Purnell. *J Natl Cancer Inst* 50(4):1003-1011, 1973.

The functional status of the mast cells associated with 30 rat mammary tumors induced by 7,12-dimethylbenz[a]anthracene (DMBA) was assessed by histochemical, autoradiographic, and quantitative histologic techniques. Specifically, the alcian blue-safranin (AB-S) reaction and radiosulfate ($^{35}S-O_4$) uptake were used to assess mast cell heparin, the phenylpropionylnaphthol-AS (PPNAS) reaction was used to study mast cell chymase, ^{3}H-TDR uptake was utilized to evaluate mast cell proliferation, and an estimate of relative tumor volume was obtained from overall tumor dimensions. The density of mast cells (mast cells/high-power field) was threefold higher in adenomas than in normal breast tissue, whereas mast cell density in cystadenomas and fibroadenomas was similar to that in normal mammary tissue. However, the mast cells in the stroma of adenomas were a mosaic of the four functional stages defined by the differential staining of heparin and its percursors by the AB-S reaction. Furthermore, the density of tumor mast cells demonstrable by the AB-S reaction was usually less than the density of tumor mast cells positive with the PPNAS reaction for mast cell chymase, but both AB-S positive and PPNAS-positive mast cells incorporated $^{35}S-O_4$ to a similar degree. These findings indicate that some mast cells associated with DMBA-induced rat mammary tumors were actively engaged in heparin synthesis and secretion. The pattern of heparin metabolism in the tumor mast cell population was independent of tumor volume and the density of mast cells in individual tumors, and tumor mast cell proliferative activity was minimal. The implications of these findings in terms of the function of mast cells in these tumors and the possibility of control of the functional status of the mast cell population by proliferating neoplastic mammary epithelium are discussed.

2518 HEPATOCARCINOGENESIS AND THE APPEARANCE OF SERUM ALPHA-FETOPROTEIN IN MICE TREATED WITH EXTRACTS OF BARLEY GRAIN INFECTED WITH *FUSARIUM SPOROTRICHIOIDES*.

(E.) Akhmeteli, M. A. (Gamalija Inst. Epidemiol. Microbiol., Moscow, USSR), A. B. Linnik and K. S. Cernov. *Bull W.H.O.* 47(5):663-664, 1972.

Sera from 49 F_1 hybrid mice (C57 x CBA), including 38 treated with *F. sporotrichioides* extract for 1 yr, were examined for alpha-fetoprotein (AFP) by a modified immunoautoradiographic method. The results show a significant association between the presence of hepatomas and AFP positivity: of 21 AFP-positive mice, 9 had hepatomas while of 28 AFP-negative mice, only 1 had a hepatoma. There was no significant difference in the incidence of hepatomas in extract-treated mice and controls. However, the data run counter to studies suggesting that AFP does not appear in hepatocarcinogenesis due to mycotoxins.

2519 SUPPRESSION OF DMBA-TUMOUR INDUCTION IN RAT MAMMARY GLANDS BY PREGNANCY OR CHLOROTRIANISENE.

(E.) Kellen, J. A. (Banting Inst., U. Toronto, Canada). *Z Krebsforsch* 79(2):75-77, 1973.

Mammary tumor induction in Sprague-Dawley rats by 7,12-dimethylbenz(a)anthracene (DMBA) during pregnancy with subsequest weaning and the effect of tri-p-anisylchlorethylene (TACE), a synthetic estrogen, were investigated. Pregnancy within five days of a single 5 mg dose of DMBA protects against the induction of mammary tumor completely; the same effect was observed after treatment with TACE, 10 mg s.c. weekly. The influence of TACE on the mitotic index and maturity of mammary epithelial cells on which DMBA acts, seem to protect from tumorigenesis provided the timing of the hormonal influence is optimal.

2520 AN ANIMAL MODEL FOR THE EVALUATION OF DRUG-INDUCED CHROMOSOME DAMAGE.

(E.) Amarose, A. P. (Pritzker Sch. Med., U. Chicago, Ill.), C. R. Schuster and T. P. Muller. *Oncology* 27(6):550-562, 1973.

Attempts to study cytogenetic effects of drugs of dependence in the human are difficult because of problems such as incomplete drug histories, multiple drug use, non-sterile administration of drugs and poor health of drug abusers. Therefore, an experimental animal system using the rabbit was established to ascertain whether a relationship exists between *in vivo* administration of drugs of abuse and chromosome damage.

Rabbits were injected i.v. with *d*-methamphetamine-HCl or with LSD-25, using single large doses in some tests, multiple smaller doses in others. A positive control was established with mitomycin C, and sterile saline was used for negative controls. Peripheral blood cultures were used for scoring chromosome complements and classifying aberrations. Chromosome complements studied before and after injection showed no association between damage and LSD or methamphetamine. Temperature in the LSD-treated animals was elevated compared to saline controls and was similar to that observed in humans. Although acute heat insult has long been known to cause chromosome damage in plants and animals, the hyperthermia observed in rabbits following LSD injection was not associated with significant chromosome damage.

2521 STUDIES OF THE TUMORIGENIC EFFECT IN FEEDING OF NITROSAMINO ACIDS AND OF LOW DOSES OF AMINES AND NITRITE TO RATS. *(E.)* Garcia, H. (U. Nebraska Med. Ctr., Omaha) and W. Lijinsky. *Z Krebsforsch* 79(3):141-144, 1973.

Three nitrosamino acids (nitrosoproline, nitrosohydroxyproline and nitrosopipecolic acid) were not tumorigenic by chronic administration at 0.015% in drinking water to MRC rats for 75 wk. Similar administration for 75 wk of mixtures of sodium nitrite (0.05%) with each of five secondary amines (0.025%), pyrrolidine, piperidine, piperazine, morpholine and heptamethyleneimine was not tumorigenic; neither was feeding of piperidine (0.1%) with sodium nitrite (0.2%). Survival in all groups, including controls, was very similar, showing no evidence of toxicity from any of the treatments. In the tests of low doses of morpholine and nitrite, which are closer to those to which man might be expected to be exposed, no liver tumors were found. It is deduced that formation of nitrosamines *in vivo* from the free amines and nitrite in these conditions was insufficient to give rise to tumors.

2522 TUMOR REGRESSION MEDIATED BY *MYCOBACTERIUM BOVIS* (STRAIN BCG). EFFECTS OF ISONICOTINIC ACID HYDRAZIDE, CORTISONE ACETATE, AND ANTITHYMOCYTE SERUM. *(E.)* Chung, E. B. (Natl. Cancer Inst., Bethesda, Maryland), B. Zbar and H. J. Rapp. *J Natl Cancer Inst* 51(1):241-250, 1973.

A methylcholanthrene-induced murine sarcoma, which grows progressively when grafted intradermally into syngeneic mice, failed to grow when injected intradermally in admixture with Bacillus Calmette-Guerin (BCG). The effect of BCG on tumor suppression depended on the number of BCG organisms injected: 10^7 colony-forming units (CFU) suppressed the growth of 5×10^5 tumor cells in 100% of mice, 10^6 CFU suppressed the growth of 5×10^5 tumor cells in 50% of mice. Elimination of the ability of BCG organisms to multiply by heat treatment *in vitro* or isonicotinic acid hydrazide treatment *in vivo* did not influence the local tumor suppressive effect of BCG or interfere with the development of delayed hypersensitivity to purified protein derivative (PPD) to tuberculin. Cortisone acetate and antimouse thymocyte serum treatment abolished BCG-mediated tumor regression and temporarily impaired delayed hypersensitivity to PPD. The data suggest that immunologically committed small lymphocytes are essential for BCG-mediated tumor killing and that the time required for completion of BCG-mediated tumor killing is >7 days. There is no convincing *in vivo* evidence that macrophages are necessary for BCG-mediated tumor regression.

2523 GLUCURONYLTRANSFERASE ACTIVITY TOWARDS DIETHYLSTILBESTROL IN RAT AND HAMSTER: EFFECT OF ACTIVATORS. *(E,)* Gabaldon, M. (Fac. Med., Valencia, Spain) and T. Lacomba. *Europ J Cancer* 8(3):275-279, 1972.

Livers were removed from rats and hamsters and liver material was incubated with diethylstilbestrol (DES) and various activators of glucuronyltransferase activity toward DES. In the rat liver, the reaction activity was negligible unless uridine diphosphate glucuronic acid (UDPGA) was added to reaction mixtures. In the presence of UDPGA only adenosine triphosphate (ATP) plus uridine diphosphate-N-acetyl-glucosamine (UDPAG) increased glucuronyltransferase activity; addition of Triton abolished the increase produced by ATP + UGPAG. In hamster liver, all activators except ethylene diamine tetraacetic acid (EDTA) increased glucuronyltransferase activity in the presence of UDPGA, while in its absence there were no significant variations. In hamster liver, the activation of glucuronyltransferase by Triton and ATP + UDPAG was additive. Glucuronyltransferase activity toward DES was higher in hamster liver than in rat liver, regardless of activation conditions. In related experiments, breakdown of UDPGA in rat was studied to determine whether the destruction of UDPGA by pyrophosphatase is a rate limiting factor of glucuronidation. Activity in rat did not increase in cases where UDPGA breakdown was not complete, which suggested that the rate limiting factor is the low content of enzyme.

2524 PHYSICAL BASIS OF CHEMICAL CARCINOGENESIS BY *N*-2-FLUORENYLACETAMIDE DERIVATIVES AND ANALOGS. *(E.)* Fuchs, R. (Biophys. Group, Louis Pasteur U., Strasbourg, France) and M. Daune. *FEBS Letters* 34(2):295-298, 1973.

Analogs and derivatives of the carcinogen *N*-2-fluorenylacetamide (AAF), after metabolic activation, are known to induce tumors in many different tissues. The use of synthetic analogs of these metabolites provided a unique opportunity to study *in vitro* the relationship between carcinogenic activity and physical modification of DNA. Calf thymus DNA was reacted *in vitro* with *N*-acetoxy-*N*-2-acetylamino-7-fluorofluorene (AAAFF), an ultimate metabolite of the strong carcinogen *N*-2-acetylamino-7-fluorofluorene (AAFF), and with *N*-acetoxy-*N*-2-acetylamino-7-iodofluorene (AAAIF), a possible metabolite of the non-carcinogen *N*-2-acetylamino-7-iodofluorene (AAIF), as well as with other analogs. The methods which were applied to the reaction products were: circular dichroism (CD) to determine the amounts of carcinogen bound; melting curves to reflect relative effects of different percentages of modified bases on melting temperatures of different products; and kinetic tests showing changes in optical density at 251 nm during incubation of the different products in borate-buffered formaldehyde (HCHO). The presence of hypochromicity, cooperative melting and CD at 305 nm in products containing noncarcinogenic as well carcinogenic compounds showed that these physical parameters do not relate directly to carcinogen properties. The appearance of optical activity and hypochromism in the absorption band of the fluorene derivatives, which was considered as evidence of intercalation, indicated only that the aromatic chromophore experiences the internal field of the double helix. More indicative of intercalation was the change of melting temperature with relative amount of modified bases, since in this case a strong linear decrease was found only with DNA modified by carcinogens like AAAF and AAAFF, not with DNA modified by AAIF. Kinetic data for unwinding induced by HCHO permitted calculation of the number of open base plates introduced by a single fluorene derivative bound to DNA and showed that the iodo-derivatives induces only a weak destabilization (1-2 base plates/bound residue) whereas the carcinogenic derivatives induce much larger loops (12-13 base plates/bound residue). The number of disorganized nucleotide pairs in the neighborhood of a binding site can thus be considered as a physical measurement of the carcinogenic activity.

2525 DOSE-RESPONSE STUDIES WITH A PURE TUMOR-PROMOTING AGENT, PHORBOL MYRISTATE ACETATE.

(E.) Van Duuren, B. L. (New York U. Med. Ctr., New York City), A. Sivak, A. Segal, I. Seidman and C. Katz. *Cancer Res* 33(9):2166-2172, 1973.

The dose-response relationship of the mouse skin tumor-promoting agent, phorbol myristate acetate (PMA), was determined over a dose range of 0.02 to 25 μg/application. 7,12-Dimethylbenz[α]anthracene was used as the initiating agent, administered one month prior to application of PMA. The effect of frequency of application of PMA was also examined by three times weekly or once weekly treatments. Administration of PMA three times weekly gave a maximal tumor response with all of the mice at risk in each of the three highest dosage groups, 25, 5, and 2.5 μg, developing papillomas. At the lower dose, 0.5 μg, the tumor incidence was 72% of the population at risk, and no tumors were observed at the two lowest doses, 0.1 and 0.02 μg. The time to 50% incidence of animals with tumors was 7.5 to 8 wk for each of the three maximal response groups, indicating that the rate of appearance of tumor-bearing mice was similar in each of these groups. However, if the total number of papillomas is compared, a dose-response relationship is clearly apparent. It was found that 2.5 μg of PMA given three times weekly is an optimal dose to obtain 100% of the mice with papillomas, although the total numbers of papillomas and cancers are significantly fewer than at the two higher doses of tumor promoter. At low doses of PMA (0.02 and 0.1 μg/ml) an inflammatory response was observed without subsequent tumor induction, even after treatment for 1 yr at these doses. Further, papillomas often appeared in areas adjacent to skin that exhibited no notable pathological changes. Thus, while tumor promotion is accompanied by epidermal hyperplasia and inflammation, these manifestations are in all likelihood not related to the mode of action of PMA. A new abbreviated method for the preparation of pure PMA is described.

2526 BIOCHEMICAL STUDIES ON ENVIRONMENTAL CARCINOGENS: EVIDENCE OF A NEW METABOLIC PATHWAY IN THE RAT.

(E.) Laham, S. (Dept. Natl. Hlth. Welfare, Ottawa, Canada). *Ann Occup Hyg* 15(2-4):203-208, 1973.

The metabolism of the carcinogen 4,4'-dinitrobiphenyl (4,4'-DNB) has been studied in male guinea pigs and Sprague-Dawley rats. Urine collected from animals receiving daily i.p. injections of 4,4'-DNB for four days and from controls was processed and studied by various physical methods in order to identify all metabolites. The metabolism of 4,4'-DNB differed markedly between the two species. Whereas guinea pigs could rapidly detoxify 4,4'-DNB and immediately acetylate the resulting amino derivatives, rats could not. Also, rats excreted large quantities of 3-chloro-4-amino-4'-nitrobiphenyl (CPAPNB) and 3-chloro-4-acetamido-4'-nitrobiphenyl (CPACPNB), two rat carcinogens which were totally absent from guinea pig urine. The biosynthesis of these chloroderivatives was investigated in rats and guinea pigs by injecting 4,4'-DNB daily for four days. Twenty-four hr after the first injection of 4,4'-DNB each animal received an injection of 0.5 ml radioactive chlorine (Na^{36}Cl). Labeled CPAPNB and CPACPNB appeared only in the urine of treated rats. Purification and identification of intermediary metabolites suggested that halogenation occurred with 4-amino-4'-nitrobiphenyl, and 4-acetylamino-4'-nitrobiphenyl, two biotransformation products of 4,4'-DNB, rather than with 4,4'-DNB itself. It was thus proposed that halogenation may be a new and unique metabolic pathway in the Sprague-Dawley rat for a number of molecules. This pathway is either absent in the guinea-pig or blocked by an inhibitor.

2527 THE EFFECTS OF IMMUNE DEPRESSION ON CHEMICAL CARCINOGENESIS IN THE RAT. II. ANTILYMPHOCYTE SERUM.

(E.) Bolton, P. M. (Dept. Surg., U. Queensland, Brisbane, Australia). *Oncology* 27(6):520-524, 1973.

The effect of immunosuppression by a goat anti-rat anti-lymphocyte serum (ALS) upon the 7,12-dimethyl benz-(α)-anthracene (DMBA)-induced mammary tumor in young Buffalo rats is reported. Before use, the potency of the antiserum was confirmed by tests for skin homograft survival time. One group of animals received 1.5 ml goat antiserum i.m. daily, commencing 2 days prior to the first dose of carcinogen and continuing for 30 days; a control group received normal goat serum by an identical regimen. No significant effect was shown, however, upon either induction rate or tumor behavior. It was suggested that immunosuppression has less effect upon chemically induced tumors than upon the more highly immunogenetic virus-induced tumors.

2528 COMPARATIVE STUDIES OF NEOPLASTIC RESPONSE TO A SINGLE DOSE OF NITROSO COMPOUNDS. 2. THE EFFECT OF N-DIBUTYLNITROSAMINE IN THE SYRIAN GOLDEN HAMSTER. *(E.)* Althoff, J. (Eppley Inst. Res. Cancer Allied Dis., U. Nebraska Med. Ctr., Omaha), P. Pour, A. Cardesa and U. Mohr. *Z Krebsforsch* 79(2): 85-89, 1973.

Papillary tumors of the trachea occurred in 3-month-old male and female Syrian golden hamsters after single increasing doses of dibutyniltrosamine (DBN) administered either by intraperitoneal, subcutaneous or intragastric route. The incidence of these tumors was found to be dose related without regard to route of application. A single dose (150-1600 mg/kg) of DBN did not lead to the development of neoplasms in the urinary bladder, as was found in previous work after chronic application of the compound.

2529 MALIGNANT UTERINE MESOTHELIOMAS IN SQUIRREL MONKEYS FOLLOWING DIETHYLSTILBESTROL ADMINISTRATION. *(E.)* McClure, H. M. (Yerkes Reg. Primate Res. Ctr., Emory U., Atlanta, Ga.) and C. E. Graham. *Lab Anim Sci* 23(4):493-498, 1973.

Ten adult female squirrel monkeys (*Saimiri sciureus*) were implanted s.c. on the medial aspect of the thigh or the upper back with four 60 mg pellets of diethylstilbestrol. Animals were killed at 5, 9, 11, and 14 months after initiation of treatment, and the abdominal and thoracic organs were examined grossly. Malignant uterine mesotheliomas occurred in seven of the ten animals. The other three animals showed early proliferative lesions of the uterine serosa. Two animals had extra-genital lesions involving the adrenals, spleen and mesentery. The short latency of tumor induction under these experimental conditions is of interest, as malignant lesions were observed in one animal only 5 months after implantation of DES, and extensive lesions in animals treated for 11 and 14 months. The findings in this preliminary study indicate that DES or a metabolite is carcinogenic in the squirrel monkey.

2530 PATTERNS OF INCORPORATION OF THYMIDINE IN THE FEMALE GENITAL AND OTHER ORGANS OF MICE, TREATED WITH 20-METHYLCHOLANTHRENE, ESTRADIOL AND OVARIECTOMY. *(E.)* Klavins, J. V. (Dept. Path., Long Island Jewish Hillside Med. Ctr., New York, N.Y.) and Z. Wessely. *Oncology* 27(6):502-519, 1973.

The pattern of incorporation of ^{3}H-thymidine with the morphological appearance of various histologically demonstrable changes was studied in the female genital organs of C3H virgin mice treated with 20-methylcholanthrene, estrogen (estradiol-benzoate) and ovariectomy. The drugs were impregnated on a cotton thread and inserted through the vagina onto the external os of the cervix and the right uterine horn. In dysplasia, pseudoephitheliomatous hyperplasia and normal epithelium, only the nuclei of the basal-cell layer contain ^{3}H-thymidine when animals are sacrificed 3 hr after the administration of the nucleotide. On the other hand, in intraepithelial carcinoma and squamous-cell carcinoma, the incorporation is diffuse throughout the tumor. The pattern of incorporation of ^{3}H-thymidine is suggested as a criterion for distinguishing dysplasias from intraepithelial carcinomas in situations where cell morphologies might appear similar.

2531 THE EFFECTS OF IMMUNE DEPRESSION ON CHEMICAL CARCINOGENESIS IN THE RAT. III. CYCLOPHOSPHAMIDE. *(E.)* Bolton, P. M. (Dept. Surg., U. Queensland, Brisbane, Australia). *Oncology* 27(6): 525-532, 1973.

The effect of the immunosuppressive drug cyclophosphamide upon the incidence and growth characteristics of rat mammary tumor induced by 7,12-dimethylbenz(α)anthracene was studied. Cyclophosphamide was administered to Buffalo rats in eight biweekly i.m. injections at dose levels of 10, 15, 20, 25 and 30 mg/kg body weight. Where cyclophosphamide was administered six days before or concurrently with the carcinogen, there was a decreased incidence and an increased latency of induced tumors. However, when the cyclophosphamide was commenced six days after the first dose of carcinogen there was an increased incidence of tumors and a decreased latency. While the exact explanation of this phenomenon is uncertain, it may be that cyclophosphamide exerts a maximal cytotoxic effect at the time of tumor induction or very early in its growth; when given after the tumor is established, the immunosuppressive effect on the host may outweigh any direct cytotoxic effect upon the tumor in this system.

2532 INFLUENCE OF HYPOPHYSECTOMY ON THE BIOCHEMICAL EFFECTS AND METABOLISM OF AFLATOXIN B_1 IN RATS. *(E.)* Friedman, L. (Division Toxicology, Food and Drug Administration, Washington, D.C.) and L. Yin. *J Natl Cancer Inst* 51(2):479-487, 1973.

Since hypophysectomy prevents induction of liver tumors by aflatoxin in rats, and since aflatoxin inhibits RNA synthesis in the liver, the present study was undertaken to determine the effects of hypophysectomy on the inhibition of RNA synthesis in rat liver *in vivo* by aflatoxin B_1 and the metabolism of aflatoxin B_1 by liver *in vitro* and *in vivo*. RNA synthesis, as measured by the incorporation of ^{14}C-orotic acid into the nucleic acid fraction of liver, was inhibited 46 and 65% by 0.33 and 0.50 mg aflatoxin/kg body wt, resp., in intact rats. Hypophysectomy resulted in the obliteration of these inhibitory effects. The metabolism of aflatoxin B_1 by 900 X g supernatants of liver homogenates, as measured by fluorescent-metabolite production *in vitro*, was altered by the hypophysectomy. The production of aflatoxin M_1 and one unidentified metabolite (X_3) was not changed, but the metabolite X_2 (produced primarily by the soluble, nonmicrosomal fraction of liver) was increased and metabolite X_1 (produced by the microsomal fraction) markedly decreased. Hypophysectomy depressed the aminopyrine demethylase activity of these same liver preparations. Similar changes occurred when SKF 525-A (5 x 10^{-5}M) was added to the incubation system, except that aflatoxin M_1 production was also slightly decreased. The shifts in metabolite concentrations that accompanied hypophysectomy were apparently the consequence of a selective decrease in one or more of the enzymes comprising the microsomal mixed-function oxidase systems. Since at least one of the discerned metabolites of aflatoxin (X_1) was decreased in amount by hypophysectomy, the postulate of Goodall and Butler that the formation of proximate carcinogen of aflatoxin (not necessarily X_1, whose activity is unknown) is depressed by this treatment is reasonable. Aflatoxin B_1 administered to rats i.p. was cleared and/or metabolized by livers of intact rats faster than by those of hypophysectomized rats.

2533 PREMALIGNANT DNA SYNTHESIS IN HAMSTER POUCH. *(E.)* Toto, P. D. (Loyola U. Dental Sch., Chicago, Ill.) and C. Swatske. *Oral Surg* 36(3):393-396, 1973.

Previous work showed that 9,10-dimethyl-1,2-benzanthracene (DMBA) can induce cancer early in pouches of prenatal and neonatal hamsters, suggesting that rapidly growing tissues are more susceptible than slower growing tissues. Since the growth of cancer is related to DNA synthesis, the process can be followed by monitoring DNA synthesis autoradiographically with the use of ^{3}H-thymidine. Eighteen dark-eared albino hamsters, 30 days old, were treated with 0.5% DMBA in mineral oil. The right buccal pouch was painted twice each wk. Each wk, for a period of 9 wk, two animals were killed one hr after injection of tritiated thymidine. A labeling index of labeled basal cells was determined each wk, comparing the DMBA-treated with the untreated control pouches. The number of labeled cells in the control pouches was 106/1,000 cells, whereas the number was 329/1,000 cells in the DMBA-treated pouches. DMBA significantly increased the number of cells entering DNA synthesis.

2534 DIFFERENT ACTION OF CARCINOGENIC AND NON-CARCINOGENIC SUBSTANCES ON THE PROLIFERATIVE ACTIVITY OF ADRENAL CORTEX CELLS. *(E.)* Danz, M. (Inst. Path., Friedrich-Schiller U., Jena, German Democratic Republic), E. Amlacher, H. Urban and F. Bolek. *Exp Pathol* 8(2):122-127, 1973.

Since liver regeneration after partial hepatectomy of the rat is accompanied by proliferative activity of the adrenal cortex cells, and since carcinogens inhibit liver regeneration, it was expected that carcinogens might also inhibit growth of adrenal cortex cells. Previous work showed, however, that certain carcinogens, namely, diethylnitrosamine (DENA) and 2-acetyl-aminofluorene (AAT), stimulated adrenal cortex cells in partially hepatectomized rats. To determine whether the observed stimulating effect depended on the partial hepatectomy, the present tests were carried out in the absence of surgery. It was found that p.o. administration of 30% LD_{50} doses of DENA, AAT or dimethylnitrosamine in ethanol-sunflower oil caused, after 48 hr, a significant increase in mitotic index of adrenal cortical cells when compared with the effect of administration of other carcinogens (3,4-benzopyrene or 7,12-dimethylbenz(a)anthracene), of non-carcinogens (allylalcohol, trinitroso-trimethylene-triamine or CCl_4) or of a positive control (ethanol alone). It was clear that the stimulating action of certain carcinogens on mitotic activity of adrenal cortex cells was not dependent on partial hepatectomy. However, the results could not be explained.

2535 COCARCINOGENIC AGENTS IN TOBACCO CARCINOGENESIS. *(E.)* Van Duuren, B. L. (New York U. Med. Ctr., New York City), C. Katz and B. M. Goldschmidt. *J Natl Cancer Inst* 51(2):703-705, 1973.

Since not all cocarcinogens are tumor-promoting agents and vice versa and since exposure of mouse skin or human lung to cigarette smoke condensate (CSC) or smoke, resp., gives simultaneous exposure to a variety of carcinogens and cocarcinogens, the use of a mouse skin cocarcinogenesis test was considered to be more meaningful than tumor promotion tests in the assay of known CSC components. Five cigarette smoke components were tested for cocarcinogenic activity; they were repeatedly applied to mouse skin together with the carcinogen benzo[a]pyrene for 52 wk. A dose of benzo[a]pyrene of 5 μg per application was used because it yielded few tumors when applied without cocarcinogen. In control groups given the test compounds without benzo[a]pyrene, no skin tumors were observed. Pronounced cocarcinogenic activity was shown by catechol and the noncarcinogenic aromatic hydrocarbons pyrene, benzo[e]pyrene, and benzo[g,h,i]perylene. Phenol had a small inhibitory effect on benzo[a]pyrene tumorigenesis. The findings have immediate practical implications in attempts to develop a less hazardous cigarette and to reduce air pollution.

2536 EFFECTS OF METALS ON VIRAL INFECTIONS IN MICE. *(E.)* Gainer, J. H. (Natl. Inst. Environmental Health Sci., Res. Triangle Park, N.C.). *Environ Health Perspect* (4):98-99, 1973.

2537 EFFECT OF DIMETHYLBENZYLDESMETHYLRIFAMPICIN (DMB) ON CHEMICALLY INDUCED MAMMARY TUMOURS IN RATS. *(E.)* Joss, U. R. (Lab. Chem. Biodynamics, U. California, Berkeley), A. M. Hughes and M. Calvin. *Nature [New Biol]* 242(116):88-90, 1973.

2538 EFFECT OF ERYTHROPOIETIC SERUM ON DNA SYNTHESIS IN THE SKIN AND SPLEEN OF MICE DURING CHEMICAL CARCINOGENESIS. *(E.)* Finogenova, M. A. (Acad. Med. Sci., Moscow, USSR). *Bull Exp Biol Med* 74(9):1087-1088, 1973.

2539 PATHOLOGY OF LIVER NECROSIS AND REGENERATION AFTER ADMINISTRATION OF DIMETHYLNITROSAMINE IN MASSIVE DOSES. *(E.)* Kuster, G. G. R. (Mayo Clin., Mayo Fdn., Rochester, Minn.), J. E. Woods and J. Ludwig. *J Surg Oncol* 5(1):9-16, 1973.

2540 AGAROSE-GEL ELECTROPHORESIS OF SOLUBLE PROTEINS FROM BRONCHIAL MUCOSA AND BRONCHOGENIC CARCINOMA. *(E.)* Louis, C. J. (Dept. Path., U. Melbourne, Victoria, Australia), J. M. Blunck and L. M. Richmond. *Oncology* 27(4):324-335, 1973.

2541 MICROBIAL FORMATION OF NITROSAMINES *IN VITRO*. *(E.)* Ayanaba, A. (Dept. Agronomy, Cornell U., Ithaca, N.Y.) and M. Alexander. *Appl Microbiol* 25(6):862-868, 1973.

2542 ELECTRON PARAMAGNETIC RESONANCE STUDY OF IODINE-INDUCED RADICALS OF BENZO[*a*]PYRENE AND OTHER POLYCYCLIC HYDROCARBONS. *(E.)* Caspary, W. (U.S. Army Med. Res. Inst. Infectious Dis., Frederick, Md.), B. Cohen, S. Lesko and P. O. P. Ts'o. *Biochemistry* 12(14):2649-2656, 1973.

2543 METABOLISM OF DIMETHYLNITROSAMINE BY AMPHIBIANS AND FISH *IN VITRO*. *(E.)* Montesano, R. (Internatl. Agency Res. Cancer, Lyon, France), A. J. Ingram and P. N. Magee. *Experientia* 29(5):599-601, 1973.

2544 CONVERSION OF STERIGMATOCYSTIN TO AFLATOXIN B_1 BY *ASPERGILLUS PARASITICUS*. *(E.)* Hsieh, D. P. H. (Dept. Environmental Toxicology, U. California, Davis), M. T. Lin and R. C. Yao. *Biochem Piophys Res Commun* 52(3):992-997, 1973.

2545 IMPURITIES IN SACCHARIN AND BLADDER CANCER. *(E.)* Hicks, R. M. (Middlesex Hosp. Med. Sch., London, England), J. St. J. Wakefield and J. Chowaniec. *Nature* 243(5407):424, 1973.

2546 ANTIBIOTICS AND LEUKAEMIA. *(E.)* Rosner, F. (Queens Hosp. Ctr., Jamaica, N.Y.) and H. Grünwald. *Lancet* (7815):1319, 1973.

2547 INFLUENCE OF AFLATOXIN B_1 AND AFLATOXIN B_2 ON RAT LIVER LYSOSOMAL ACID DEOXYRIBONUCLEASE. *(E.)* Pitout, M. J. (Natl. Res. Inst. Nutritional Dis., Pretoria, South Africa) and J. C. Schabort. *Biochem Pharmacol* 22(14):1801-1805, 1973.

2548 THE ROLE OF CYTOCHROME P-450 IN *N*-HYDROXYLATION OF 2-ACETYLAMINOFLUORENE. *(E.)* Thorgeirsson, S. S. (Natl. Inst. Allergy, Infectious Dis., Bethesda, Md.), D. J. Jollow, H. A. Sasame, I. Green and J. R. Mitchell. *Mol Pharmacol* 9(3):398-404, 1973.

2549 INDUCTION OF DRUG METABOLISM. VII. DIFFERENCES IN P-420 HEMOPROTEINS FROM UNTREATED AND 3-METHYLCHOLANTHRENE-TREATED RATS. *(E.)* Shoeman, D. W. (U. Minnesota Med. Sch., Minneapolis), F. M. Vane and G. J. Mannering. *Mol Pharmacol* 9(3):372-382, 1973.

2550 REACTIONS OF ESTERS OF *N*-HYDROXY-2-ACETAMIDOPHENANTHRENE WITH CELLULAR NUCLEOPHILES AND THE FORMATION OF FREE RADICALS UPON DECOMPOSITION OF *N*-ACETOXY-*N*-ARYLACETAMIDES. *(E.)* Scribner, J. D. (Pacific Northwest Res. Fdn., Seattle, Wash.) and N. K. Naimy. *Cancer Res* 33(6):1159-1164, 1973.

2551 ENZYME ACTIVITIES IN REGRESSING ESTRONE-INDUCED MAMMARY TUMORS OF THE RAT. *(E.)* Cutts, J. H. (Dept. Anatomy, U. Missouri, Columbia). *Cancer Res* 33(6):1235-1237, 1973.

2552 ASSAY OF CARCINOGENS. *(E.)* Anonymous. *JAMA* 224(11):1522-1523, 1973.

2553 HANDLING CARCINOGENS. *(E.)* Groenen, P. J. (Central Inst. Nutrition, Food Res. TNO, Zeist, Netherlands). *Science* 180(4086):542, 1973.

2554 ENDOREDUPLICATION IN CULTURED MAMMALIAN CELLS TREATED WITH 4-NITROQUINOLINE 1-OXIDE. *(E.)* Sutou, S. (Nomura Res. Inst., Kamakura, Kanagawa, Japan). *Mutat Res* 18(2):171-178, 1973.

2555 MUTAGENICITY OF DIMETHYLNITROSAMINE IN THE METABOLIC PROCESS BY RAT LIVER MICROSOMES. *(E.)* Nakajima, T. (Natl. Inst. Hygienic Sci., Tokyo, Japan) and S. Iwahara. *Mutat Res* 18(2):121-127, 1973.

2556 CHRONIC THYROIDITIS IN BUFFALO-STRAIN RATS INGESTING 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE OR N-2-FLUORENYLDIACETAMIDE. *(E.)* Reuber, M. D. (U. Maryland Sch. Med., Baltimore) and E. L. Glover. *Pathol Microbiol* 39(5):383-388, 1973.

2557 TESTS ON THE PRODUCTION OF TUMOURS IN ANIMAL EXPERIMENT WITH POLYCYCLIC AROMATIC HYDROCARBONS. *(Ger.)* Pott, F. (Med. Inst. Air Hygiene, Silicosis Res., U. Düsseldorf, West Germany), A. Brockhaus and F. Huth. *Zentralbl Bakteriol Hyg* 157 (1):34-43, 1973.

2558 ASSESSMENT OF THE ACTION OF ASBESTOS UNDER INDUSTRIAL ENVIRONMENT CONDITIONS. (RECOMMENDATIONS OF THE ASBESTOSIS SUBCOMMITTEE AFFILIATED WITH THE PERMANENT COMMITTEE AND INTERNATIONAL ASSOCIATION FOR OCCUPATIONAL DISEASES). *(Rus.)* Krelli, L. (USA), G. Eyer, K. Amoudrue, G. Gibbs, S. Holmes, E. Occella and R. Du Tois. *Gig Sanit* (2):76-79, 1973.

2559 THE MECHANISM OF ANTICARCINOGENIC ACTION OF 2,6-DI-TERT-BUTYL-4-METHYLPHENOL (IONOL), AN INHIBITOR OF FREE-RADICAL PROCESSES. *(Rus.)* Emanuel, N. M. (Inst. Chem. Phys., USSR Acad. Sci., Moscow), A. N. Saprin, T. S. Shulyakovskaya, L. N. Kozlova, and T. V. Bunto. *Dokl Akad Nauk SSSR* 209(6):1449-1451, 1973.

2560 AN EXPERIMENTAL MODEL FOR CANCER OF THE COLON AND RECTUM. INTESTINAL CARCINOMA INDUCED IN THE RAT BY 1,2-DIMETHYLHYDRAZINE. *(E.)* Martin, M. S. (Lab. Exp. Med., Dijon, France), F. Martin, H. Bastien, E. Justrabo, M. Bordes and B. Viry. *Digestion* 8:22-34, 1973.

2561 SMOKING AND CANCER. *(E.)* Peto, R. (Radcliffe Infirm., Oxford, England) and J. Mathews. *Lancet* (7812):1126, 1973.

2562 *IN VITRO* DEMONSTRATION OF TUMOR-SPECIFIC COMMON ANTIGENS AND EMBRYONAL ANTIGENS IN MURINE FIBROSARCOMAS INDUCED BY 7,12-DIMETHYLBENZ(α)ANTHRACENE. *(E.)* Ménard, S. (Natl. Inst. Study, Cure Tumors, Milan, Italy), M. I. Colnaghi and G. D. Porta. *Cancer Res* 33(3):478-481, 1973.

2563 HEART RHABDOMYOSARCOMA DEVELOPED IN A RAT UPON ORTHO-TOLUIDINE TREATMENT. *(Rus.)* Pliss, G. B. (Sci. Res. Inst. Oncol. USSR Acad. Med. Sci.) and N. I. Volfson. *Vopr Onkol* 19(3):98-99, 1973.

2564 THE CARCINOGEN-BINDING PROTEIN OF NORMAL AND TUMOR CELLS. *(Rus.)* Gelshtein, V. I. (Moscow, USSR), G. A. Bannikov and T. A. Chipysheva. *Vopr Onkol* 19(4):96-97, 1973.

2565 FEATURES OF THE CARCINOGENIC ACTION OF DIBUTYLNITROSOAMINE. *(Rus.)* Vlasov, N. N. (N. N. Petrov Sci. Res. Inst. Onkol. Leningrad, USSR), F. K. Dzhioyev and G. B. Pliss. *Vopr Onkol* 19(4):55-59, 1973.

2566 LIVER CANCER IN THE RAINBOW TROUT INDUCED BY AFLATOXINS. *(Ger.)* Wunder, W. (Friedrich-Alexander U. Inst. Zool. Erlangen-Nürnberg, W. Germany). *Z Lebensm Unters-Forsch* 150(4):250-255, 1973.

2567 FURTHER OBSERVATION ON THE EFFECTS OF 3-METHYLCHOLANTHRENE AND PHENOBARBITAL ON CARBON TETRACHLORIDE HEPATOTOXICITY. *(E.)* Pani, P. (U. Turin, Torino, Italy), M. V. Torrielli, L. Gabriel and E. Gravela. *Exp Mol Pathol* 19(1):15-22, 1973.

2568 EXPERIMENTAL HEPATOPATHIES AND MAMMARY GLAND CARCINOMA IN RATS. *(E.)* Al'pert, A. É. (Saratov Med. Inst., USSR), A. V. Arkhangel'skii, A. M. Lunts and N. P. Panina. *Bull Exp Biol Med* 74(10):1299-1301, 1972.

2569 MYCOTOXINS. INTERNATIONAL AFLATOXIN CHECK SAMPLE PROGRAM: 1972 STUDY. *(E.)* Coon, F. B. (WARF Inst., Madison, Wis.), F. J. Baur and L. R. L. Symmes. *J Am Osteopath Assoc* 56(2):322-327, 1973.

2570 TOXICITY OF METABOLITES PRODUCED BY THE "ALTERNARIA." *(E.)* Pero, R. W. (Natl. Insts. Health, Res. Triangle Park, N.C.), H. Posner, M. Blois, D. Harvan and J. W. Spalding. *Environ Health Perspect* (4):87-94, 1973.

2571 NICOTINE STOMATITIS AND ITS RELATION TO CARCINOMA OF THE HARD PALATE IN REVERSE SMOKERS OF CHUTTAS. *(E.)* Ramulu, C. (Andhra Med. Coll., Visakhapatnam, India), M. V. Raju, G. Venkatarathanam and C. R. Reddy. *J Dent Res* 52(4):711-718, 1973.

2572 PIGMENTED TUMORS IN RATS INDUCED BY INTRODUCTION OF PLATINUM AND CELLOPHANE FILMS INTO THE CHAMBER OF THE EYE. *(E.)* Evgen'eva, T. P. (Acad. Sci., USSR, Moscow). *Bull Exp Biol Med* 74(10):1296-1298, 1972.

2573 ULTRASTRUCTURE OF PULMONARY ADENOMAS INDUCED BY INTRATRACHEAL INSTILLATION OF DIETHYLNITROSAMINE IN SYRIAN GOLDEN HAMSTERS. *(E.)* Straks, W. (State U., Utrecht, Netherlands) and V. J. Feron. *Europ J Cancer* 9(5):359-362, 1973.

2574 THE EFFECT OF ERYTHROPOIETIC SERUM ON DNA SYNTHESIS IN THE SKIN AND SPLEEN OF MICE DURING CHEMICAL CARCINOGENESIS. *(Rus.)* Finogenova, M. A. (USSR Acad. Med. Sci., Moscow). *Biull Eksp Biol Med* 74(9):90-92, 1972.

2575 RECENT CASES OF ETHMOID AND MAXILLARY TUMORS OCCURRING AMONG WOOD WORKERS. *(Fr.)* Fombeur, J. -P. (Saint-Michel Hosp., Paris, France). *Arch Mal Prof* 33(9):453-455, 1972.

2576 EXPERIMENTAL STUDIES ON TUMORS OF THE URINARY SYSTEM OF RATS INDUCED BY CHEMICAL CARCINOGENS. *(E.)* Nobuyuki, I. (Nara Med. Coll., Kashihara, Japan). *Acta Path Jap* 23(1):87-109, 1973.

2577 CHEMICALLY-INDUCED CANCER OF THE UTERINE CERVIX IN RATS. ULTRASTRUCTURE OF THE EARLY STAGES. ROLE OF CASTRATION. *(Fr.)* Laffargue, P. (Lab. Path. Anat., Marseille, France) and J. F. Boyer. *Bull Cancer* 59(3):315-330, 1972.

2578 MAMMALIAN CELLS FOR CARCINOGEN IDENTIFICATION. *(E.)* Evans, C. H. (Natl. Cancer Inst., Bethesda, Md.) and J. A. DiPaolo. *JAMA* 225(6):631-632, 1973.

2579 THE EFFECT OF DIMETHYLNITROSAMINE ON EMBRYONIC TISSUE OF MOUSE KIDNEY IN AN ORGAN CULTURE. *(Rus.)* Bogovsky, S. P. (USSR Acad. Med. Sci., Moscow) and Yu. D. Sorokina. *Biull Eksp Biol Med* 74(2):75-77, 1973.

2580 ARSENATE-INDUCED CARCINOGENESIS AND TUMOR DOUBLING TIME. *(Fr.)* Chahinian, P. (Lariboisière Hosp., Paris, France). *Nouv Presse Med* 2(11): 720, 1973.

2581 CARCINOGENESIS ACROSS THE PLACENTA. *(E.)* Anonymous. *Br Med J* 3(5874):250, 1973.

2582 THE EFFECT OF ADULT THYMECTOMY AND ADULT SPLENECTOMY ON THE PRODUCTION OF LEUKEMIA AND STOMACH NEOPLASMS IN MICE BY *N*-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL] ACETAMIDE. *(E.)* Cohen, S. M. (U. Wisconsin Med. Sch., Madison), D. B. Headley and G. T. Bryan. *Cancer Res* 33(4):637-640, 1973.

See also:

- * (Rev): 2405, 2410, 2427, 2431, 2436, 2447, 2454, 2465, 2467, 2468, 2470, 2475
- * (Phys): 2538, 2596
- * (Immun): 2699, 2705, 2735, 2761, 2782
- * (Epid-Biom): 2915, 2920, 2923, 2928

2583 FORMATION OF A CARCINOGEN OF NATURAL ORIGIN IN THE ETIOLOGY OF ULTRAVIOLET LIGHT-INDUCED CARCINOGENESIS.

(E.) Black, H. S. (Baylor Coll. Med., Houston, Texas) and D. R. Douglas. *Cancer Res* 33(9): 2094-2096, 1973.

The time course of cholesterol α-oxide formation in the skin of hairless mice receiving chronic suberythemic levels of UV radiation was determined. The concentration of this carcinogen increased and reached a peak level at 10 wk, after which squamous cell carcinomas appeared. The facts that an increase in formation of this carcinogen precedes the appearance of tumors and that this increase is apparently related to the dose of UV radiation received suggest that this photoproduct, of natural origin, is involved in the etiology of UV-induced carcinogenesis.

2584 EFFECT OF ^{60}CO-γ-RAYS ON THE INFRARED SPECTRA OF DNA.

(E.) Karolyi, G. (Med. U., Debrecen, Hungary), J. Skapinyecz, B. Somogyi and S. Damjanovich. *Acta Biochim Biophys Acad Sci Hung* 7(2):179-185, 1972.

The effect of ^{60}Co-γ-irradiation on high power polymer calf thymus DNA solutions was determined by infrared spectroscopy between 800-1700 cm^{-1}. Irradiation ($\sim 10^4$ kR) caused only a minor alteration in infrared spectra, mainly due to damage of different bases (e.g., adenine-thymine, guanine-cytosine). Higher irradiation doses ($\sim 10^5$ kR) increased spectral changes at wavenumbers characteristic of certain bases. However, spectral parts characterizing the number of base pairs remained practically unchanged after doses that produced degradation detectable by viscosity measurements. The viscosity determinations suggested that strand breaks are more significant in radio-denaturation of DNA than direct damage of bases. Experimental results obtained in the presence of sodium dodecylsulfate pointed to the importance of H bonds in the formation of infrared absorption of nucleic acids. On the other hand, infrared and viscosity measurements refer to a reversibility of postirradiation H bond breaks.

2585 INDUCTION OF TUMORS INVOLVING BONE IN BEAGLES FED TOXIC LEVELS OF STRONTIUM 90.

(E.) Pool, R. R. (Radiobiol. Lab., U. California, Davis), R. J. Romer Williams and M. Goldman. *Am J Roentgenol Radium Ther Nucl Med* 118(4):900-908, 1973.

Beagles were observed during and after the feeding of Sr^{90} for the first 1.5 yr of life. The distribution of the sites at which tumors involving bone appear to rise and the possible radiobiologic significance of the interaction of the local dose rate with the quality of cell population at risk were determined. Bone tumors developed in 11 of 19 dogs receiving about 20,000 mrad/day, 9 of 47 receiving about 7,000 mrad/day, and 2 of 56 receiving about 2,000 mrad/day. No tumors developed in 251 dogs fed Sr^{90} at lower levels or in 160 control dogs. Tumors appeared first in dogs given the highest level of Sr^{90} and were mainly osteosarcomas of long bones; next in dogs given intermediate levels and mainly osteosarcomas of the head; last in dogs given lowest levels and were fibrosarcomas of the head. The risk for tumor production may be a function of rate and degree of cellular repair which, in turn, is dependent on the severity of prior radiation injury.

2586 THYROID CARCINOMA AND RADIATION.

(E.) DeGroot, L. (Dept. Med., U. Chicago, Ill.) and E. Paloyan. *Jama* 225(5):487-491, 1973.

The records of 50 patients with thyroid carcinoma were studied. Of these patients, 20 had received prior neck x-ray treatment. These patients developed tumors an average of 20 years after x-ray exposure, and were nearly all under 35 years of age. Clinical findings were similar to those of the nonexposed carcinoma patients, but the tumors were less invasive and never undifferentiated. One patient in the x-ray group succumbed to the illness. Two thirds of these patients who had a history of known x-ray exposure and who showed clinical thyroid abnormalities had cancer.

2587 INFLUENCE OF OESTROGENIC HORMONES ON CARCINOGENESIS AND TOXICITY OF RADIOSTRONTIUM.

(E.) Nilsson, A. (Res. Inst. Natl. Defence, Sundbyberg, Sweden) and C. Rönnbäck. *Acta Radio* [*Ther*] *(Stockh)* 12(3):209-228, 1973.

The influence of estrogen on the skeleton of ^{90}Sr-treated mice was studied. In one experiment male and female CBA mice were treated with various doses of estrogen s.c. on days, 0, 30 and 60 and with various doses of ^{90}Sr i.p. on days 7 and 67. The diagnosis of bone tumors in this experiment was based entirely on roentgenologic and post mortem findings. In another experiment histologic changes were observed in male mice treated with estrogen alone, ^{90}Sr alone, or estrogen + ^{90}Sr and the different tumors which were induced were classified. The dose of estrogen was in some cases logarithmically decreasing on days 0, 30 and 60, and in other cases a uniform dose of 0.25 mg was administered on each of these days. Among the males given both estrogen and ^{90}Sr there was a mean number of 4.2 bone tumors/mouse with a mean induction time of 194.1 ± 2.6 days as compared with 2.3 bone tumors/mouse and mean induction time of 316.1 ± 1.1 days among those given only ^{90}Sr. Similarly, the female mice treated with estrogen + ^{90}Sr developed 4.1 bone tumors/mouse with an induction time of 251.0 ± 4.1 days and those given ^{90}Sr alone developed 2.0 bone tumors/mouse with an induction time of 379.3 ± 9.7 days. No bone tumors were found in the mice treated with estrogen alone. Osteoclastic and mixed osteosarcomas were more frequent in combination treatment groups than in groups given ^{90}Sr alone.

2588 THE ROLE OF SURGERY AND ^{131}I THERAPY FOR HYPERTHYROIDISM IN THE ETIOLOGY OF THYROID CARCINOMA.

(E.) Ashkar, F. S. (U. Miami Sch. Med., Fla.), A. Maile, Jr., W. M. Smoak III and M. B. Weinstein. *South Med J* 66(9):1014-1016, 1973.

The occurrence of thyroid carcinoma following ^{131}I therapy for thyrotoxicosis is unusual; only nine cases are reported to date in the literature. This study reports four patients who developed thyroid carcinoma following surgical treatment for thyrotoxicosis in a series of 48 patients. It was suggested that chronic stimulation with endogenous thyrotropin (TSH) and long-acting thyroid stimulator (LATS), acting upon the thyroid remnant left after surgery, may have some etiologic role in this situation. Since ^{131}I therapy for hyperthyroidism has been found to cause an impairment of the reproductive integrity of the thyroid cell, resulting in a low incidence of thyroid carcinoma, it is recommended that all surgically treated hyperthyroid patients be placed on thyroid hormone replacement to turn off TSH and to reduce the stimulation to the thyroid remnant.

2589 ACTIVATION OF LYMPHOID LEUCOSIS IN CHICKS BY IRRADIATION. *(E.)* Lageröf, B. (Dept. Pathol., Karolinska Sjukhuset, Sweden) and I. Mares. *Acta Path Microbiol Scand* [A] 81(8):569-576, 1973.

Two strains of commercially bred chicks (White Leghorn and Babcock), and one strain of "leucosis-free" chicks (White Leghorn-SPAFAS) were treated with whole-body irradiation. This treatment enhanced the incidence of lymphoid leucosis in the commercially bred animals; with the highest dose of irradiation employed (400 rad X 4), 20% of the Babcock chicks developed lymphoid leucosis after a latent period of 161-357 days. One of 12 Babcock chicks developed lymphoid leucosis following a dose of only 150 rad X 4. None of the irradiated SPAFAS chicks which were kept in strict isolation during the whole experimental period developed leucosis. The disease was transmissible to other non-irradiated chicks with cell-free material. The whole-body irradiation is considered to have a complex effect with possible activation of latent avian tumor virus. One of nine irradiated SPAFAS chicks kept in contact with "leucosis-carrying" chicks developed lymphoid leucosis. Low levels of viral antibodies against RSV (RAV-2) were repeatedly detected, but virus isolation experiments were negative. Cell free homogenate of liver tissue from this chick was inoculated i.v. into Babcock and SPAFAS White Leghorn chicks, and none of these chicks developed lymphoid leucosis.

2590 CELL KINETIC MODEL OF TUMOUR GROWTH AND REGRESSION FOR A RHABDOMYOSARCOMA IN THE RAT: UNDISTURBED GROWTH AND RADIATION RESPONSE TO LARGE SINGLE DOSES. *(E.)* Curtis, S. B. (Radiobiol. Inst. TNO, Rijswijk, Netherlands), G. W. Barendsen and A. F. Hermens. *Eur J Cancer* 9(2):81-87, 1973.

2591 EFFECT OF PREIRRADIATION OF THE TUMOR BED ON THE RELATIVE VASCULAR SPACE OF MOUSE GASTRIC ADENOCARCINOMA 328 AND MAMMARY ADENOCARCINOMA CA755. *(E.)* Jirtle, R. (U. Wisconsin Med. Sch., Madison) and K. H. Clifton. *Cancer Res* 33(4):764-768, 1973.

2592 MALIGNANT MELANOMA AND LATITUDE. *(E.)* Anonymous. *Med J Aust* 1(13):624, 1973.

2593 DEFENSE OF CUTANEOUS CELLS AGAINST UV IRRADIATION. II. RESTRICTED PHOTOMEDIATED BINDING OF TRIMETHYL PSORALEN TO PIGMENTED SKIN *IN VIVO. (E.)* Carter, D. M. (Yale U. Sch. Med., New Haven, Conn.), B. V. Jegasothy and E. S. Condit. *J Invest Dermatol* 60(5):274-277, 1973.

2594 DEFENSE OF CUTANIOUS CELLS AGAINST UV IRRADIATION. I. PHOTOMEDIATED BINDING OF TRIMETHYL PSORALEN TO DNA OF MELANOMA CELLS IN CULTURE. *(E.)* Carter, D. M. (Yale U. Sch. Med., New Haven, Conn.), M. deL. V. McMacken and E. S. Condit. *J Invest Dermatol* 60(5):270-273, 1973.

2595 ULTRAVIOLET-INDUCED MUTATIONS TO ASPARAGINE INDEPENDENCE IN JENSEN SARCOMA CELLS *IN VITRO. (E.)* Vernick, D. (Dept. Physiol., Cell Biol., U. Kansas, Lawrence) and J. Morrow. *Mutat Res* 18(2):225-231, 1973.

2596 INDUCTION OF CHROMOSOMAL ANOMALIES BY UV RADIATIONS AND ETHYLENIMINE IN MAMMALIAN CELLS DEPENDING ON THEIR REPAIR ACTIVITY. *(Rus.)* Dubinin, N. I. (Inst. Gen. Genetics, Moscow, USSR), G. I. Goroshkina, L. L. Matusevich and G. D. Zasukhina. *Dokl Akad Nauk SSSR* 210(2):464-467, 1973.

2597 THE ROLE OF DIRECT RADIATION IN THE MECHANISM OF MAMMARY GLAND TUMOR DEVELOPMENT. *(Rus.)* Moskalev, Yu. I. (Inst. Biophys. USSR Pub. Hlth. Min., Moscow). *Biull Eksp Biol Med* (5):68-70, 1973.

2598 STIMULATION OF CLONOGENIC GROWTH OF TUMOUR CELLS AND METASTASES IN THE LUNGS BY LOCAL X-RADIATION. *(E.)* Van den Brenk, H. A. S. (St. Thomas Hosp., London, England), W. M. Burch, C. Orton and C. Sharpington. *Br J Cancer* 27(4):291-306, 1973.

2599 DISTRIBUTIONS OF CELL VOLUME AND DNA CONTENT OF RHABDOMYOSARCOMA CELLS GROWING *IN VITRO* AND *IN VIVO* AFTER IRRADIATION. *(E.)* Kal, H. B. (Radiobiol. Inst. TNO, Rijswijk, Netherlands). *Eur J Cancer* 9(1):77-79, 1973.

2600 INTESTINAL POLYPOSIS INDUCED BY DIFFERENT KINDS OF IONIZING RADIATION. *(Rus.)* Lebedeva, G. A. (No affiliation). *Vopr Onkol* 19(4):47-51, 1973.

2601 THE ENHANCEMENT OF THE RADIATION LETHAL EFFECT UPON EHRLICH ASCITES TUMOUR CELLS BY AMINOHYDROXYTHYMINE HYDRAZINE. *(E.)* Mustea, I. (Oncological Inst., Cluj, Romania). *Int J Radiat Biol* 23(4):409-413, 1973.

2602 THE DYNAMICS OF ALTERATIONS OCCURRING IN THE OVARIES, HYPOPHYSIS, AND ADRENALS DURING THE LATENT PERIOD OF RADIATION-INDUCED CARCINOGENESIS. *(Rus.)* Pavlenkomiknaylov, Yu. N. (Biophys. Inst. Min. Pub. Hlth., USSR). *Vopr Onkol* 18(10):87, 1972.

2603 THE MUTAGENIC EFFECT OF URANIUM TESTED HUMAN LEUKOCYTES AND MOUSE FIBROBLASTS IN TISSUE CULTURE *IN VITRO*. *(Rus.)* Malenchenko, A. F. (Inst. Nuclear Energetics, BSSR Acad. Sci., Minsk, USSR) and N. A. Barkun. *Dokl Akad Nauk BSSR* 17(4):375-377, 1973.

2604 THE RELATIONSHIP OF INTRA-UTERINE RADIATION TO SUBSEQUENT MORTALITY AND DEVELOPMENT OF LEUKEMIA IN CHILDREN. A PROSPECTIVE STUDY. *(E.)* Diamond, E. L. (Johns Hopkins U. Sch. Hygiene, Publ. Hlth., Baltimore, Md.), H. Schmerler and A. M. Lilienfeld. *Am J Epidemiol* 97(5):283-313, 1973.

2605 SOME LATE EFFECTS OF RADIATION IN THE BONE MARROW STEM CELLS OF THE MOUSE. *(E.)* Proukakis, C. (Med. Coll. St. Bartholomew's Hosp., London, England), J. E. Coggle and P. J. Lindop. *Radiol Clin Biol* 42(1):24-29, 1973.

2606 MORPHOLOGICAL MODIFICATIONS OF RAT GLIAL TUMOR CELLS AFTER X-IRRADIATION. *(E.)* Heilporn, V. (Lab. Cytology, Embryology, Free U. Brussels, Rhode St. Genèse, Belgium), A. Lievens, S. Limbosch, F. Zampetti-Bosseler and G. Steinert. *Radiat Res* 54(2):252-260, 1973.

2607 EXPOSURE-RESPONSE CURVE FOR RADIATION-INDUCED LUNG TUMORS IN THE MOUSE. *(E.)* Yuhas, J. M. (Oak Ridge Natl. Lab., Tenn.) and A. E. Walker. *Radiat Res* 54(2):261-273, 1973.

2608 DOSE-RATE EFFECTS IN SYNCHRONOUS MAMMALIAN CELLS IN CULTURE. *(E.)* Bedford, J. S. (Vanderbilt U. Hosp., Nashville, Tenn.) and J. B. Mitchell. *Radiat Res* 54(2):316-327, 1973.

2609 CHROMOSOMAL INTERCHANGES INDUCED BY RADIATION IN SPERMATOGONIAL CELLS AND LEUKOCYTES OF MOUSE AND CHINESE HAMSTER. *(E.)* Brewen, J. G. (Oak Ridge Natl. Lab., Oak Ridge, Tenn.) and R. J. Preston. *Nature [New Biol]* 244(134):111-113, 1973.

2610 CARCINOMA OF THE THYROID IN A PATIENT TREATED WITH RADIOIODINE FOR HYPERTHYROIDISM. *(E.)* Reeve, T. S. (Royal North Shore Hosp. Sydney, Australia), I. B. Hales, K. V. Smith and N. A. Fowler. *Med J Aust* 1(20):993-996, 1973.

2611 RADIATION-INDUCED ADSORPTION OF ^{3}H-THYMIDINE ON LEUKOMA CELLS. *(Ger.)* Schmitt, G. (Ruhr-U. Clin., Essen, West Germany), H. Kaufmann and E. Löhr. *Radiol Clin Biol* 42(1):40-45, 1973.

See also:

* (Rev): 2401, 2403, 2408, 2436, 2452
* (Immun): 2755

2612 MOLECULAR PROBES IN STUDIES OF THE RELATIONSHIP OF VIRUSES TO HUMAN NEOPLASIA.

(E.) Bowen, J. M. (M. D. Anderson Hosp. Tumor Inst., Houston Texas), P. T. Allen, J. L. East, K. Maruyama, W. A. Newton, Jr., J. Georgiades, E. S. Priori and L. Dmochowski. *Am J Clin Pathol* 60(1):88-99, 1973.

In a series of studies based on the animal leukemia-sarcoma virus model system, cultures derived from human fibrosarcoma desmoides and from giant cell tumor of bone were inoculated with fresh bone marrow aspirates from patients with acute lymphocytic leukemia and investigated with the use of biochemical probes. Foci of morphologically altered cells appeared in these cultures several tissue culture passages after inoculation. Cell lines were derived from the altered cells and were characterized biologically and biochemically. The cells exhibited growth properties and tumorigenicity characteristic of malignant cells in immunosuppressed mice. The cells were also found to release particles (buoyant density 1.15 to 1.17 Gm. per ml.) containing reverse transcriptase and high molecular weight RNA. The data obtained suggest that genetic information similar to that of animal RNA tumor viruses was present in the cells and was activated during transformation. The results add further evidence implicating RNA tumor viruses in the etiology of some types of human neoplasia, such as leukemia, giant cell tumor, and fibrosarcoma desmoides.

2613 FAST-LABELED HIGH MOLECULAR WEIGHT RIBONUCLEIC ACID (RNA) AND VIRUS PRODUCTION IN AVIAN MYELOBLASTOSIS VIRUS-INFECTED CHICKEN FIBROBLASTS.

(Fr.) Imbenotte, J. (Gastave-Roussy Inst. Villejuif, France) and C. Verger. *C R Acad Sci* [*D*] *(Paris)* 276(6):1049-1052, 1973.

Fast-labeled polysomal RNA (messenger RNA) sucrose gradient sedimentation profiles from *in vitro* cultivated brown Leghorn Carr strain chicken embryo fibroblasts were studied after treatment with avian myeloblastosis virus (AMV)-infected leukemic chicken plasma or with plasma supernates obtained upon centrifugation at 55,000 g for 60 min at 4 C. Control fibroblast cultures were maintained in an Eagle's media containing 15% of foal serum. In experimental cultures, the foal serum was substituted with leukemic plasma or with supernatant. For fibroblasts treated with either leukemic plasma or supernate, an augmentation of RNA production, as revealed by uptake of ^{3}H-uridine in RNA fractions from polysome preparations, occurred between the 3rd and 7th day. For those fibroblasts treated with the leukemic plasma, however, the RNA which was isolated was 18S, and virions, as revealed by electron microscopic examination, did not appear in abundant amounts until the 5th day. For those fibroblasts treated with supernate, the RNA had a sedimentation coefficient greater than 30 and virus was revealed in large amounts after only 2 days. The presence of this heavy 30S fraction was attributed to viral genome replication or to maturation forms cosedimentating with the cellular polysomes. Experiments attempting to identify the particles responsible for viral infection in the 55,000 g leukemic cell incubation media supernates are in progress.

2614 CHARACTERIZATION OF VIRUSES OBTAINED AFTER CELL FUSION OR TRANSFECTION OF CHICKEN CELLS WITH DNA FROM VIROGENIC MAMMALIAN ROUS SARCOMA CELLS.

(E.) Hlozanek, I. (Czechoslovak Acad. Sci., Prague) and J. Svoboda. *J Gen Virol* 17(1):55-59, 1972.

Previous work showed that transfection of chicken embryo fibroblasts with DNA from virogenic mammalian cells transformed by Rous sarcoma virus gave rise to infective, transforming, tumor-inducing virus. Studies were carried out to determine whether the viruses recovered after transfection belonged to subgroups which were the same as, or different from, those of the viruses used initially for transformation of the cell lines from which the DNA transfecting agents were obtained. The initial viruses were varied as to subgroup and the final viruses were tested for subgroup by serum neutralization, virus interference and host range tests. It was found that the final virus obtained after transfection with DNA from rat cells transformed with Prague strain of Rous sarcoma virus had the properties of the subgroup C avian sarcoma viruses to which the initial Prague strain belongs. Similarly, final virus obtained after transfection with DNA from Chinese hamster cells transformed by Schmidt-Ruppin strain of Rous sarcoma virus had the properties of the subgroup D viruses to which the initial Schmidt-Ruppin strain belongs. Parallel results were obtained in experiments involving fusion instead of transfection. The results point to the specificity of the transfection system and preclude the possibility that DNA from virogenic cells only activates the pre-existing endogenous virus genomes present in chicken cells. However, the possibility is still not excluded that genetic recombination with those genomes occurs when DNA is transferred to chicken cells.

2615 CROSS-SPECIES TRANSMISSION OF MAMMALIAN RNA TUMOR VIRUSES.

(E.) Maruyama, K. (M.D. Anderson Hosp. Tumor Inst., Houston, Tex.) and L. Dmochowski. *Tex Med* 69(8):65-75, 1973.

Type C mouse Friend leukemia virus (MFLV) and type C feline sarcoma virus of Theilen strain were adapted to grow in the Wistar rat. This was accomplished by i.p. inoculation of the MFLV and by s.c. injection of kitten tumor tissue into newborn rats. The rat Friend leukemia virus (RFLV) thus produced had increased oncogenicity in rats, as demonstrated by a shortened latent period for production of tumors following neonatal inoculation, but decreased immunogenicity, as shown by lack of mixed hemagglutination (MHA) activity of rat anti-RFLV sera compared with rat anti-MFLV sera when tested against cultured mouse or rat Friend tumor cells. The rat Friend tumor (RFT) cells carried antigens common to mouse virus, as shown by the observation that sera of rats which rejected grafts of RFT neutralized MFLV and the finding that rats inoculated when newborn with MFLV almost all died with tumors when challenged with RFT at the 7th day. Untreated control rats showed no deaths on challenge. After several passages of RFT in rats, the RFLV failed to induce leukemia in newborn mice, providing further evidence of the adaptation of the virus to rats. Results of studies carried out in the feline versus rat system were generally similar to those of the mouse versus rat system. Thus, inoculation of extracts of feline virus-induced rat tumors containing virus particles into newborn kittens failed to induce tumors, while inoculation into newborn rats produced a 28% incidence. Also, the latent period for induction of tumors in rats was shortened. Generally, however, the adapted rat tumors were virus-nonproducing. MHA tests with goat or rabbit antisera to feline leukemia virus failed to show virus antigens at the surfaces of the rat cells. However, immunofluorescence studies showed such antigens in the cytoplasm. Furthermore, some tumor-bearing rats developed antibodies which precipitated ether-soluble components of the virus particles.

2616 CYCLIC AMP-AMPLIFIED REPLICATION OF RNA TUMOUR VIRUS-LIKE PARTICLES IN CHINESE HAMSTER OVARY CELLS. *(E.)* Tihon, C. (St. Louis U. Sch. Med., Mo.) and M. Green. *Nature* [*New Biol*] 244(138):227-231, 1973.

The effects of cyclic adenosyl monophosphate (cyclic AMP) on the characteristics of transformed cells were examined. Treatment of Chinese hamster ovary cells (CHO-K1) with 10^{-3} M dibutyryl cyclic AMP plus 1.5 x 10^{-5} M testosterone proprionate altered the morphology of the cells from randomly oriented, epithelial-like to parallel-arrayed fibroblasts. This treatment also led to the formation of large quantities of particles that contain the 70S RNA and RNA-directed DNA polymerase activity characteristic of RNA tumor viruses. These results suggested that exposure to cyclic AMP enhanced formation of large quantities of an endogenous hamster leukemia or sarcoma virus. This possibility was strengthened by the large increase in agglutinability by concanavalin A of CHO-K1 cells which had been treated with 10^{-3} M dibutyryl cyclic AMP plus 1.5 x 10^{-5} M testosterone for 48 hr. The mechanism by which the addition of dibutyryl cyclic AMP plus testosterone causes a large stimulation in the expression of this presumed endogenous hamster virus is unknown. The cyclic AMP enhanced virus production in the CHO-K1 system may well be a further example of the expression of an endogenous genome in a mammalian cell.

2617 CHARACTERIZATION AND SEPARATION OF VIRAL DNA POLYMERASE IN MOUSE MILK. *(E.)* Howk, R. S. (Meloy Lab., Springfield, Va.), L. A. Rye, L. A. Killeen, E. M. Scolnick and W. P. Parks. *Proc Natl Acad Sci USA* 70(7):2117-2121, 1973.

Previous demonstration of an RNA-directed DNA polymerase activity in mouse milk from a high mammary tumor virus (MTV) strain, RIII, provided a potential measure of viral activity in milk. Recent experiments of the authors showed that polymerase activity in mouse milk was partially and inconsistently inhibited by antisera against the type-C viral enzyme. Since one explanation of this variability was that the milk actually contained a mixture of type-B and type-C viral enzymes, they undertook in the present study to identify several viral enzymes in milk of RIII mice. Two were found, using fractionation on phosphocellulose and Sephadex G-100 columns. One was an RNA-directed DNA polymerase of type-C viruses; this enzyme preferred manganese to magnesium in activity measurements using poly(rA)·oligo(dT) as synthetic substrate, was inhibited by specific sera, and had an apparent MW of 70,000. Milk from BALB/c and NIH Swiss mice contained a vast predominance of this C-type enzyme. The other DNA polymerase from RIII mouse milk preferred magnesium to manganese, was not inhibited by type C antipolymerase serum, and appeared of larger molecular wt than the type-C polymerase. Its presence in milk from RIII mice and absence from milk of mice with low content of MTV correlates with the relative degree of type-B virus expression in these mice. Because of certain morphological similarities between murine MTV and the Mason-Pfizer monkey virus (MP-MV) isolated from a spontaneous breast tumor of a rhesus monkey, the authors also compared the MP-MV RNA-directed DNA polymerase with a gibbon type-C viral enzyme. The MTV and MP-MV polymerases both preferred magnesium as a divalent cation and eluted ahead of the type-C enzymes on chromatography, while the murine type-C enzyme and the gibbon enzyme were similar in cation preferences and in size. Thus, the MP-MV and gibbon type-C viral enzymes share similarities, resp., with the mouse type-B and mouse type-C viral enzymes. The apparent relationship between MTV and MP indicate the importance of obtaining evidence for additional similarities.

2618 INHIBITORY EFFECT OF CHICK INTERFERON AND INTERFERON INDUCER (rI:rC) ON THE INDUCTION OF CELLULAR DNA SYNTHESIS AND TRANSFORMATION OF CHICK EMBRYO FIBROBLASTS INFECTED IN CULTURE WITH ROUS SARCOMA VIRUS. *(E.)* Kara, J. (Inst., Exp. Biol. Genetics, Czechoslovak Acad. Sci, Prague). *Folia Biol (Praka)* 19(3):194-202, 1973.

Chick embryo fibroblasts (CEF) preincubated for 24 hr in tissue culture with chick interferon and subsequently infected with Rous sarcoma virus (RSV), strain Schmidt-Ruppin, exhibit a significant resistance to the induction of cellular DNA synthesis after infection and a concomitant resistance to the transformation by RSV. The induction of uridine kinase activity and cellular DNA polymerase activity after RSV-infection is inhibited in CEF cultures pretreated with chick interferon. When RSV preparation purified by zonal centrifugation in sucrose density gradient was used for infection of CEF cultures, cellular DNA synthesis was activated and the inhibitory effect of interferon on the induction of cellular DNA synthesis and cell transformation was observed. Preincubation of CEF cultures for 4 hr in the presence of polyriboinosinic: polyribocytidylic acid (rI:rC, 20 μg/ml.) resulted in complete inhibition of the induction of cellular DNA synthesis and 95% inhibition of cell transformation after infection of rI:rC pretreated cultures with RSF. The purified RSV, heat-inactivated for 5 min at 100 C, does not induce DNA synthesis in CEF cultures. These results suggest that induction of cellular DNA synthesis and cell transformation are virus-specific functions, very probably mediated by virus-specific proteins. The biosynthesis of the viral proteins seems to be inhibited in cells pretreated with interferon and/or with rI:rC.

2619 CHROMOSOMAL ABERRATIONS OF A LYMPHOBLASTOID CELL LINE BY EXPERIMENTAL INFECTION WITH EPSTEIN-BARR VIRUS. *(E.)* Amano, K. (Fukushima Med. Coll., Japan), S. Shigeta, I. Awano and Y. Hinuma. *Gann* 64(3):309-311, 1973.

The lethal infection of cells with Epstein-Barr (EB) virus was examined by karyological studies. The C-6 cell line derived from a single colony in a soft agar plate inoculated with cells of the human lymphoblastoid NC-37 line, which is known to possess the EB viral genomes but not the viral antigens and particles, was used as target cells. Infective EB virus was prepared from the culture fluid of the P3HR-1 Burkitt lymphoma cell line. NC-37 cells without viral infection showed two peaks at 46 and 48 in chromosomal number distributions. Chromosome number in the majority (75.5%) of C-6 cells was 46. This finding supports the hypothesis that the C-6 line originated from a single cell and was clonal in nature. Chromosomal aberrations, such as secondary constriction, translocation, and deletion, were observed in all groups of chromosomes. These aberrations were found consistently at 2,4, and 6 hr after infection. The frequency of cells with definite constriction of chromosomes was low, but it became higher in the later stages of infection. The rate of chromosomal aberrations correlated roughly to the virus-dose inoculated. These observations indicate that the pattern of chromosomal alterations in cells infected with EB virus is primarily determined by the nature of the host cells and not by the genetic specificity of the EB virus.

2620 MURINE RETICULUM CELL NEOPLASMS TYPE B (HODGKIN'S-LIKE LESIONS) INDUCED IN BALB/c MICE WITH FIELD ISOLATES OF MURINE LEUKEMIA VIRUS. *(E.)* Ebbesen, P. (Inst. Med. Microbiol., U. Copenhagen, Denmark), R. Rask-Nielsen, J. W. Hartley and W. P. Rowe. *Eur J Cancer* 9(3):173-179, 1973.

In vitro and *in vivo* characteristics of field isolates of mouse leukemia virus inducing reticulosarcoma type B were examined. Twenty-four per cent of specific pathogen-free BALB/c female control mice and 9% of control males developed lymphoid neoplasias; 3/4 of the neoplasms were reticulosarcomas type B (Hodgkin's-like lesions). Without change in the proportion of reticulosarcomas, the tumor incidence was tripled by infecting newborn BALB/c mice with 0.05 ml five times concentrated subcellular extracts of reticulosarcomas, doubled by injecting 10^5 XC-units of either of two leukemia-type viruses recently isolated from BALB/c mice; and tripled by grafting 10^4 cells from leukemia virus-infected syngeneic secondary embryonic fibroblast cultures. Intrathymic inoculation gave the same result as s.c. administration. A four log reduction in virus dose given s.c. only lowered the tumor incidence slightly. The mean latent period was 13 months following inoculation of cell-free virus preparations and 8 months after injection of infected tissue culture fibroblasts. Adult mice receiving virus did not show enhanced tumor incidence. The preferential development in virus-treated mice, whether inoculated s.c. or in the thymus, of the tumor type most common in untreated animals gives indirect evidence for the etiologic role of C-type virus for spontaneous tumors of the reticuloendothelial system. From the nearly identical effect on incidence and survival time of giving newborn BALB/c mice 10^5 or 10^1 plaque forming units of B-tropic virus, it is assumed that no threshold dose exists for new-borns, whereas the results indicate a state of resistance in adults.

2621 VIRUS-LIKE TUBULAR CYTOPLASMIC INCLUSIONS IN EPITHELIAL TUMORS. ELECTRON MICROSCOPIC STUDIES. *(E.)* Maciejewski, W. (Warsaw Med. Sch., Poland), J. Dabrowski, S. Jablonska and K. Jakubowicz. *Dermatologica* 146(3):141-148, 1973.

Electron microscopic studies were made in 11 cases of benign epithelial tumors (common warts, seborrheic warts, and papilloma) and malignant ones (Bowen's disease and basal cell epithelioma). The age of the patients ranged from 20 to 60 yr. Characteristic tubular inclusions were detected chiefly in the cytoplasm of vascular endothelial cells in six cases (common warts, papilloma, Bowen's disease, and basal cell epithelioma). Morphologically, they resembled paramyxoviruses and were exactly similar to those described in collagen diseases, chiefly in lupus erythematosus. The relation of the virus-like tubular inclusions to the virus of Gross remains obscure. It is possible that the former develop in consequence of an already established epithelial proliferation without being a causative factor. Their presence both in benign proliferation lesions and in those with certain atypical histological features, the inconstancy of this presence, and a lacking correlation between it and the character and duration of epithelial tumors do not speak in favor of the pathogenic significance.

2622 UTILIZATION OF MAMMALIAN 70S RNA BY A PURIFIED REVERSE TRANSCRIPTASE FROM HUMAN MYELOCYTIC LEUKEMIC CELLS. *(E.)* Bhattacharyya, J. (Natl. Cancer Inst., Bethesda, Md.), M. Xuma, M. Reitz, P. S. Sarin and R. C. Gallo. *Biochem Biophys Res Commun* 54(1):324-334, 1973.

A DNA polymerase activity present in the 60,000 x g pellet of human acute myelocytic leukemic blood cells catalyzes an endogenous RNA-dependent (RNase sensitive) DNA synthesis. After purification, this enzyme transcribes heteropolymeric regions of 70S RNA from various RNA tumor viruses but prefers mammalian over avian viral RNA. DNA transcribed from the viral RNA is, like that from RNA tumor virus reverse transcriptase, hydrogen bonded and covalently attached to the RNA template-primer. The DNA transcript, after alkaline hydrolysis, hybridizes specifically to the template-primer RNA. This activity is not decreased in the presence of DNA polymerases from normal cells, suggesting that the inability of the latter polymerases to catalyze transcription of viral RNA is not due to the presence of inhibitor(s). This DNA polymerase is easily distinguished from a terminal addition deoxynucleotidyl transferase and has previously been shown to be immunologically closely related to primate type-C virus reverse transcriptase. Present results indicate that this enzyme is detectable in a limited number of patients with acute leukemia. It is, however, too early to estimate its distribution or whether it is present only in certain stages of the disease.

2623 ELECTRON MICROSCOPE STUDIES OF MOUSE MAMMARY TUMOR VIRUS (MTV) WITH SPECIAL CONSIDERATION OF THE MECHANISM INVOLVED IN EXTRUSION OF A-PARTICLES FROM THE CELL.

(Ger.) Rühland, D. (Dept. Med. Ultrastructural Res., U. Münster, Germany), E. Fasske, R. Fetting and H. Themann. *Z Krebsforsch* 79(4):309-318, 1973.

Electron microscope studies were made of mammary tumors which developed spontaneously in up to 15% of female NMRI mice. A-particles were found only within the cell, while B-particles were present both in cytoplasmic vacuoles and in extracellular spaces. In actively secreting cells it was demonstrated that A-particles were extruded from the cells and matured into B-particles. Many A-particles formed in the Golgi apparatus. Protein secretion vacuoles developed nearby and migrated into these clumps of A-particles. A-particles then adhered to the membranes of protein-secretion vacuoles, and the secretion vacuoles, together with A-particles, migrated to the cell surface. Drops of protein secretion with A-particles adhering to their surrounding membrane were extruded into the glandular lumen by reverse pinocytosis. Here A-particles matured into B-particles by budding on the cell membrane. It is likely that there is a direct relation between A- and B-particles because: (1) both particles are always found together in spontaneous mammary tumors in mice, (2) A-particles and the inner part of immature B-particles are morphologically identical; (3) the stereochemical nature of A- and B-particles is similar under the electron microscopy and (4) A-particles are extruded from inside the cytoplasm into the glandular lumen in actively secreting cells.

2624 PREFERENTIAL INTEGRATION OF SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID INTO A PARTICULAR SIZE CLASS OF CV-1 CELL DEOXYRIBONUCLEIC ACID.

(E.) Waldeck, W. (German Cancer Res. Ctr., Heidelberg), K. Kammer and G. Sauer. *Virology* 54(2):452-464, 1973.

Permissive CV-1 cells were productively infected with low multiplicities (0.5 PFU/cell) of plaque-purified Simian virus 40 (SV40). The DNA was radioactively labeled throughout the period of infection (48 hr) and then separated into a supernatant and pellet fraction by a selective extraction method. Covalent linkage of SV40 DNA sequences with host DNA sequences was shown to occur preferentially in newly replicated, small linear, double-stranded DNA molecules that can be isolated from the supernatant. These molecules have an average length of 1.5-2 times the contour length of SV40 DNA. Covalent linkage of SV40 DNA sequences with larger host DNA sequences which can be pelleted during selective extraction has also been demonstrated to occur, although to a much lesser extent. The viral-host DNA sequences are discussed as a possible means for the study of the properties of the site of integration of tumor virus genes in mammalian DNA.

2625 IDENTIFICATION OF A CRITICAL PERIOD DURING THE S PHASE FOR ACTIVATION OF THE EPSTEIN-BARR VIRUS BY 5-IODODEOXYURIDINE.

(E.) Hampar, B. (Natl. Cancer Inst., Bethesda, Md.), J. G. Derge, L. M. Martos, M. A. Tagamets, S.-Y. Chang and M. Chakrabarty. *Nature* [*New Biol*] 244(137):214-217, 1973.

Epstein-Barr (EB) virus activation by the thymidine analogue 5-iododeoxyuridine (IdU, 20 μg/ml) was studied in synchronized EB-3 (producer) and Raji (nonproducer) human lymphoblastoid cell lines. The findings localize the critical period for EB virus activation by IdU to DNA synthesized at approximately 60 min into the cells' S phase. Although definite evidence is lacking, this critical period appears to coincide with the period of DNA synthesis where spontaneous virus activation occurs. Since virus activation requires only short-term treatment with the drug, and is followed rapidly by antigen synthesis which does not persist due to cell death, an inheritable mutation is unlikely.

2626 THE POSSIBILITY TO OVERCOME SPECIES AND AGE RESISTANCE IN THE INDUCTION OF A NEOPLASTIC PROCESS BY AN AVIAN TUMOUR VIRUS.

(E.) Zhdanov, V. M. (USSR Acad. Med. Sci., Moscow), G. -K. S. Chepulis, V. Ya. Shevlyaghin, N. P. Mazurenko, J. G. Kiselieva and J. N. Zueva. *Rev Roum Virol* 10(2):161-168, 1973.

C57BL and BaLB/c mice with an induced immunological tolerance to normal chick embryo tissue antigens became susceptible to the Schmidt-Ruppin strain of Rous Sarcoma virus (RSV) at the age of 1.5 to 4 months. Tumors of sarcoma type developed in the animals 1.5 to 3.5 months after their infection with RSV. The sarcomas were transplantable to normal mice and produced RSV that was regularly isolated from chickens. RSV group-specific antigen was regularly revealed in the tumors by complement fixation tests for avian leukosis virus. The experiments showed the possibility to overcome both the species and the age resistance barriers to the avian tumour virus.

2627 HEMATOPOIETIC COLONY-FORMING UNITS IN MICE INFECTED BY THE POLYCYTHEMIA-INDUCING FRIEND VIRUS. III. PATTERN OF BLOOD RECOVERY IN IRRADIATED MICE GRAFTED WITH NORMAL OR INFECTED SPLEEN CELLS.

(E.) Wendling, F. (Natl. Inst. Hlth. Med. Res., Orsay, France), P. E. Tambourin and P. Jullien. *J Natl Cancer Inst* 51(1):179-189, 1973.

The ability of spleen colony-forming units (CFU) from leukemic donors to differentiate into mature progeny was studied for 28 days after spleen cells were grafted in isogenic X-irradiated recipients, and compared with the progeny produced by the same amount of normal spleen CFU. The repopulation assays indicated that CFU derived from Friend leukemic spleens were able to provide reticulocytes, leukocytes, and platelets in the peripheral blood. However, these

CFU had little ability to produce thrombocyte progeny, and erythrocyte production was lowered 7.5-fold. The CFU in leukemic mice spleens were less efficient than CFU from normal spleens in protecting irradiated mice from acute hematopoietic death. The low therapeutic efficiency of leukemic spleen CFU is probably connected with the deficit in blood platelets.

2628 RAUSCHER VIRUS INFECTION, ERYTHROCYTE CLEARANCE STUDIES, AND AUTOIMMUNE PHENOMENA.

(E.) Cox, K. O. (Sch. Med., Perth, Australia) and D. Keast. *J Nat Cancer Inst* 50(4):941-946, 1973.

BALB/c mice infected with high doses of Rauscher virus (RV) developed the typical prelymphoid leukemic disease characterized by hemolytic anemia and thrombocytopenia. Survival times of ^{51}Cr-labeled erythrocytes from normal syngeneic mice were significantly shorter in RV-infected mice than in uninfected controls. *In vitro* tests showed that serum factors were not responsible for the shortened erythrocyte survival times. Spleen lymphocytes, but not peripheral blood lymphocytes, from RV-infected mice were stimulated into DNA synthesis by erythrocytes from both RV-infected and uninfected mice. Apparently spleen lymphocytes from RV-infected mice made an immune response against erythrocytes from both uninfected and RV-infected mice. The spleen lymphocytes did not respond to erythrocytes from sheep and, compared with spleen lymphocytes from normal mice, showed a decreased response to phytohemagglutinin (PHA). However, this decrease was counterbalanced by the increase in PHA-responsive cells in the peripheral blood.

2629 SYNCYTIAL GIANT CELL FOCUS ASSAY FOR VIRUSES DERIVED FROM FELINE LEUKEMIA AND A SIMIAN SARCOMA.

(E.) Rangan, S. R. S. (Hazelton Lab., Inc., Vienna, Va.), P. J. Ueberhorst and M. C. Wong. *Proc Soc Exp Biol Med* 142(4):1077-1082, 1973.

A syncytial giant cell (SGC) focus assay has been developed which permits the determination of the results of titrations of feline leukemia and simian sarcoma virus preparations in a period of 1 wk. A canine tumor cell line, M.132-1, and a rat tumor cell line, XC, were used as initiator of infection and detector of the test viruses, resp. Test virus adsorption was carried out in suspension in 13 x 100 mm sterile, screw-capped plastic tubes. Various concentrations of freshly trypsinized M.132-1 cells in 0.1 ml growth medium and 0.1 ml of virus dilutions were mixed and incubated at 37 C for 1 hr. Identical samples of M.132-1 cells incubated with 0.1 ml Hanks balanced salt solution were used as controls. Between 96 and 120 hr, supernatant fluid from each culture was replaced with 5 ml 10% fetal bovine serum supplemented Eagle's minimum essential medium containing 1.5 x 10^6 freshly trypsinized XC cells. Following 48 hr of incubation, the cultures were washed, fixed, stained, and the plates scanned at a magnification of about 35 with an inverted microscope. A direct correlation between virus dilution and the number of SGC foci was repeatedly obtained. SGC focus formation was prevented when the test virus was reacted with specific antisera.

2630 VIRUSES AND RENAL CARCINOMA OF *RANA PIPIENS*. XIV. TEMPERATURE-SENSITIVE MUTANTS OF FROG VIRUS 3 WITH DEFECTIVE ENCAPSIDATION.

(E.) Purifoy, D. (St. Jude Children's Res. Hosp., Memphis, Tenn.), R. F. Naegele and A. Granoff. *Virology* 54(2):525-535, 1973.

The temperature-sensitive defects of six mutants of frog virus 3 were identified. Although each of the mutants synthesized comparable amounts of virus-specific DNA at permissive (25 C) and nonpermissive (30 C) temperatures, viral DNA was not encapsidated at 30 C. When infected cells were shifted to permissive temperature, the viral DNA made at 30 C was encapsidated. Encapsidation required DNA and protein synthesis at 25 C, and the 30 C DNA could act as template for the required RNA. The assembled virions containing 30 C DNA were infectious. Thus, the DNA synthesized at nonpermissive temperature is fully functional and the temperature-sensitive defects involve only encapsidation.

2631 PRIMARY MALIGNANT MELANOMA IN A SMALLPOX VACCINATION SCAR: REPORT OF A CASE AND REVIEW OF THE LITERATURE.

(E.) DeOreo, G. (Eisenhower Med. Ctr., Palm Desert, Ca.). *Int J Dermatol* 12(4):217-219, 1973.

The fourteenth case of primary malignant melanoma in a smallpox vaccination scar is reported. In this case, the patient is a 47-year-old Caucasian woman, who had been vaccinated for smallpox on her arm when she was six years old. There were no abnormal physical findings other than a scaling, black nodule (0.6 cm diameter) within the vaccination scar (1.5 cm diameter). There were no satellite lesions and no enlarged nodes in the axilla. The probability of a chance occurrence of a melanoma in an area of skin equal to the size of a vaccination was calculated at 1/1,000,000,000. A Communicable Disease Center report, however, that lists all the complications which resulted following vaccination of 1,650,000 people, lists one case of a skin cancer in the vaccination site; this malignancy was a melanoma. This occurrence constitutes a rate exceeding 600 times that which could be anticipated. Presumptive evidence, therefore, suggests a possible causal relationship between vaccination and melanomas.

2632 MOLECULAR EVIDENCE FOR A VIRAL ETIOLOGY OF HUMAN LEUKEMIAS, LYMPHOMAS, AND SARCOMAS.

(E.) Hehlmann, R. (Coll. Physicians Surgeons, Columbia U., New York, N. Y.), W. Baxt, D. Kufe and S. Spiegelman. *Am J Clin Pathol* 60(1): 65-79, 1973.

The etiologic significance of earlier findings in human leukemia, lymphoma, and sarcoma of RNA homologous to that of the Rauscher leukemia virus (RLV) was examined. The data obtained indicate that at least a portion of the viral-related RNA detected previously is in the form of a 70S-RNA template physically associated with a reverse transcriptase, two of the diagnostic features of the RNA tumor viruses. Further, the DNA synthesized by the leukemic reverse transcriptase on its own endogenous template is related in sequence to RLV-RNA. This last finding completes the logic of earlier experiments in which the DNA synthesized on RLV-RNA was used as a probe to find the viral-rated information in leukemic cells. Finally, the data show that a reverse transcriptase and 70S RNA are associated with a particle possessing the exact density of oncogenic RNA viruses. The data show that human leukemic cells contain a 70S RNA encapsulated with RNA-instructed DNA polymerase in a particle possessing the density characteristic of RNA viruses. Further, the DNA synthesized by the human RNA-enzyme complex hybridizes specifically with the RNA of a murine leukemogenic agent. Similar data are now available with human mammary cancers. Thus, for both leukemias and adenocarcinomas of the breast, four diagnostic features of the corresponding animal tumor viruses are exhibited by particles found in these analogous human neoplasias. The accumulating evidence for the involvement of RNA tumor viruses in at least some forms of human cancer is becoming more convincing.

2633 LOADING OF THE RETICULOENDOTHELIAL SYSTEM: EFFECT ON THE INDUCTION, DEVELOPMENT AND CHEMOTHERAPY OF RAUSCHER VIRUS LEUKEMIA.

(E.) Buchman, V. M. (Inst. Exp. Clin. Oncol., Acad. Med. Sci. USSR, Moscow), R. M. Radzichovskaya, G. J. Svet-Moldavsky, M. D. Kozlova, S. E. Mikaelian and V. J. Levin. *Biomedicine* 19(8):335-339, 1973.

Based on the supposition that one of the main functions of the reticuloendothelial system (RES) is protection of the organism from infectious and toxic agents and that loading of the system with colloidal solutions inhibits the function of the RES cells, experiments were conducted to test the effect of treating adult BALB/c mice with colloidal gold or carbon on the susceptibility of the mice to Rauscher leukemia virus (RLV) and on the chemotherapeutic action of methotrexate on established Rauscher leukemia. Two i.p. injections of colloidal gold solution (CGS) given 24 and 1 hr prior to injection of RLV resulted in significantly greater susceptibility to the virus as shown by acceleration of the increase of spleen wt and increase in number of subcapsular leukemic colonies in the spleen. Injection of CGS also increased the toxicity of large doses of methotrexate as shown by mortality rate. Injection of CGS or colloidal carbon solution rendered small doses of methotrexate therapeutically effective against established Rauscher leukemia, where the methotrexate alone was not effective. It is concluded that the RES has a "barrier" function which protects stem and other progenitor cells of hemopoietic tissues and tumor cells from different injurious agents such as oncogenic viruses and antitumor chemotherapeutic drugs.

2634 PARTICLES RELEASED FROM ARGININE DEPRIVED HUMAN LEUKAEMIC CELLS.

(E.) Kotler, M. (Hebrew U. -Hadassah Med. Sch., Jerusalem, Israel), E. Weinberg, O. Haspel, U. Olshevsky and Y. Becker. *Nature* [*New Biol*] 244(137):197-200, 1973.

Arginine deprivation of Rous sarcoma virus (RSV) transformed cells was previously demonstrated to induce the synthesis and release of C type virions. When this approach was applied to leukemia cells which were obtained from patients with acute lymphoblastic leukemia and myeloblastic leukemia and which were labeled with ^{3}H-uridine, particles were released which were labeled with ^{3}H-uridine, banded at a density of 1.17 g/ml, and contained RNase sensitive DNA polymerase activity which transcribed from 70S RNA molecules and formed 70S RNA:DNA hybrid molecules. These particles thus resemble oncorna viruses. The mechanism of action of arginine deprivation is being studied.

2635 DEAE-DEXTRAN ENHANCEMENT OF *IN VIVO* INFECTION WITH MURINE LEUKEMIA VIRUS.

(E.) Ebbesen, P. (Inst. Med. Microbiol., U. Copenhagen, Denmark). *Arch Gesamte Virusforsch* 40(3-4):307-310, 1973.

The applicability of diethylaminoethyl (DEAE)-dextran as an *in vivo* enhancer of infection with murine leukemia virus was examined by measuring the tissue content of leukemia virus 3 wk after simultaneous inoculation of virus and DEAE-dextran. Two-month old inbred C3H male mice were inoculated s.c. with 10^6 XC units of Rauscher or B_1 murine leukemia virus suspended in 2 ml Minimum Essential Medium (Eagle) pH 7.2 (MEM). The mice received 25 μg DEAE-dextran i.p. in 1 ml MEM simultaneously with the virus inoculation. Controls received Rauscher virus s.c. only, B_1 virus s.c. only, DEAE-dextran i.p. only or no treatment at all. The mice were sacrificed 2 wk after infection and two pools of spleen, thymus, and serum from five animals in each experimental group were collected, frozen, and stored at -70 C before analysis. It was found that DEAE-dextran enhanced the amount of virus by 2 to 140 times. The virus titre was found to be one to two log higher in spleens than in thymus and serum whether the infection was accompanied by DEAE-dextran inoculation or not.

2636 RELATIONSHIP BETWEEN NUCLEAR POCKETS IN BOVINE PERIPHERAL BLOOD LYMPHOCYTES AND C-TYPE VIRUS PARTICLES IN CULTURES OF THESE CELLS.

(E.) Weber, A. (Col. Veterinary Med., U. Minn., St. Paul), R. H. Hammer and C. Jessen. *J Natl Cancer Inst* 51(1):81-88, 1973.

Quantitative hematologic and ultrastructural investigations of freshly drawn and 72-hour cultured blood lymphocytes were made on 50 clinically normal adult Holstein-Freisian (1-12 yr old) bulls to examine the relationship between lymphocyte nuclear pockets (LNP's) and the presence of C-type virus particles. Average peripheral LNP values for the 50 bulls were 0.75± 0.12 (SE). Statistical evaluation of the LNP virus relationship for 10 high-LNP and 10 low-LNP animals showed a probability of <0.001 that these two groups represented 1 population. Analysis of this relationship for individual animals in the pooled sample indicated a correlation of 0.565. The probability that the pooled sample came from a population in which the actual coefficient of correlation was zero was 0.009. These results suggest that lymphocyte populations with increased LNP incidences can produce C-type virus particles.

2637 EVIDENCE OF BOVINE C-TYPE (LEUKEMIA) VIRUS IN DAIRY CATTLE. *(E.)* Olson, C. (Dept. Vet. Sci., U. Wisconsin, Madison), H. E. Hoss, J. M. Miller and L. E. Baumgartener. *J Am Vet Med Assoc* 163(4):355-357, 1973.

Approximately 1,000 dairy cattle in 11 herds were tested for precipitating antibodies against bovine C-type (leukemia) virus; 222 reactors were detected. Five herds in which lymphosarcoma had not occurred during the past 13 years (or more) had a lower percentage of reactors (2 to 16%) than did 6 herds with histories of lymphosarcoma during the past ten years (24 to 42%). Virus was demonstrated in 100 of 117 reactors, including 36 cattle from the five herds without histories of recent lymphosarcoma. The leukemia virus of cattle may not have originated in cattle. The natural hosts of the leukemia viruses of chickens, mice, and cats do not produce antibodies against the internal antigens of their virus. It is assumed that the internal nucleoid of these viruses does not differ enough from the host for it to recognize the virus as foreign and to react by producing antibodies. Since antibody to an internal antigen of bovine leukemia virus can be demonstrated in cattle, the virus may eventually be found to come from some other animals and infect cattle.

2638 HUMAN POLYOMAVIRUS INFECTION IN RENAL ALLOGRAFT RECIPIENTS. *(E.)* Coleman, D. V. (St. Mary's Hosp., London, England), S. D. Gardner and A. M. Field. *Br Med J* 3(5876):371-375, 1973.

Viruses morphologically resembling members of the polyoma virus group have been recovered recently from man. The first of these, named B.K. virus, was isolated in tissue culture from the urine of a renal transplant patient. For further evidence of infection with human polyomaviruses, particularly B.K. virus, cytological and virological studies were undertaken on 75 patients with functioning renal allografts. Ten patients (13.5%) were excreting polyomavirus. Virus particles were seen in the electron microscope in urine samples from eight patients. B.K. polyomavirus was isolated from four patients. Infection with a different polyomavirus was probable in one patient. Virus isolation was most readily achieved when large numbers of intact virus particles were seen in the urine. Patients who were excreting large amounts of polyomavirus shed numerous inclusion-bearing cells which could be detected by cytology. A serological study showed that the prevalence of B.K. antibody was similar to that found in the general population and 38% of this series of transplant patients had evidence of active infection with B.K. virus.

2639 DNA POLYMERASES IN RNA TUMOR VIRUSES AND POSSIBLE "PROTOVIRUSES". *(E.)* Mizutani, S. (McArdle Lab. Cancer Res., U. Wisconsin, Madison). *Am J Clin Pathol* 60(1):19-24, 1973.

Experiments were conducted to determine whether the DNA polymerase of the chicken endogenous RNA-directed DNA polymerase activity is related to the avian RNA tumor virus DNA polymerase or to other DNA polymerases in normal chicken embryos. A 3-4S DNA polymerase was partially purified from a fraction with endogenous RNA-directed DNA polymerase activity isolated from normal chicken embryos. The activity of this 3-4S DNA polymerase was not neutralized either by an antibody prepared against DNA polymerase purified from avian myeloblastosis virus or by an antibody prepared against a 10S DNA polymerase purified from the soluble fraction of chicken embryos. After removal of ribonuclease activity, the 3-4S DNA polymerase could utilize avian RNA tumor virus RNA as a template primer. The response of this 3-4S DNA polymerase to synthetic template primers was very similar to that of DNA polymerase from avian sarcoma virus and different from that of soluble chicken DNA polymerase. The existence of a chicken DNA polymerase which can utilize RNA as well as DNA as a template primer for DNA synthesis suggests that this type of DNA polymerase could play a role in information transfer from RNA to DNA in normal cells, as suggested by the "protovirus hypothesis."

2640 ROLES OF CALCIUM, SERUM, PLASMA, AND FOLIC ACID IN THE CONTROL OF PROLIFERATION OF NORMAL AND ROUS SARCOMA VIRUS-INFECTED CHICKEN FIBROBLASTS. *(E.)* Balk, S. D. (Natl. Res. Counc. Canada, Ottawa, Ontario), J. F. Whitfield, T. Youdale and A. C. Braun. *Proc Natl Acad Sci USA* 70(3):675-679, 1973.

In a culture medium of pH 7.4 and a folic acid concentration of 100 µg/liter that contains 5% heat-inactivated chicken plasma rather than serum, the rate of proliferation of normal chicken fibroblasts is determined by the concentration of calcium. Proliferation, rapid when the calcium concentration is physiological, decreases when the calcium concentration is reduced. At a very low calcium concentration, in this culture medium, normal fibroblasts are maintained without proliferation, whereas those infected with Rous sarcoma virus proliferate rapidly. This proliferative inactivity of normal fibroblasts does not involve contact-inhibition, since the effect is observed at low, as well as higher, culture densities. When a physiological amount of calcium is added to cultures of normal fibroblasts that have been maintained in very low calcium-plasma medium for three days, labeled thymidine uptake and protein synthesis are strongly stimulated, and cell division follows. The use of heat inactivated chicken serum, instead of plasma, in this medium appears to strongly sensitize normal fibroblasts to the mitogenic action of calcium. In a plasma-containing culture medium of physiological calcium concentration and a folate concentration of 5 µg/liter neither normal nor Rous sarcoma virus-infected fibroblasts proliferate to an appreciable extent. The use of serum, however, instead of plasma results in rapid proliferation of both normal and infected cells, as does increase in the folate concentration of the plasma-containing medium to 100 µg/liter. The fact that while normal fibroblasts are maintained without proliferation in low calcium-plasma medium, Rous sarcoma virus-infected fibroblasts proliferate rapidly, indicates that the effect of calcium is regulatory rather than permissive. These results suggest that the proliferation of normal fibroblasts is initiated by a cellular function involving calcium, and that the autonomous proliferation of the neoplastic fibroblasts results either from increased calcium uptake or from an alteration or a bypass of that function. The results also suggest that serum contains a mitogenic factor(s) not present in plasma, possibly a "wound hormone" for fibroblasts.

2641 STUDIES OF FBJ OSTEOSARCOMA VIRUS IN TISSUE CULTURE. I. BIOLOGIC CHARACTERISTICS OF THE "C"-TYPE VIRUSES. *(E.)* Levy, J. A. (Natl. Cancer Inst., Bethesda, Md.), J. W. Hartley, W. P. Rowe and R. J. Huebner. *J Natl Cancer Inst* 51(2):525-539, 1973.

The FBJ osteosarcoma virus (FBJ-MSV) is the only known naturally occurring murine sarcoma virus (MSV). It is unique among strains of MSV in producing primarily osteosarcomas in mice since other MSV's have emerged only after tissue culture and animal passages. It appeared that study of its biology might suggest new approaches to research on other naturally occurring animal sarcoma viruses. A nonpathogenic virus, FBJ-MLV (murine leukemia virus), was found to be associated at high titer with the MSV and could be detected by limiting dilution passage. This MSV/MLV complex was similar to the RNA members of the murine oncogenic virus group, having typical "C"-type morphology, group-specific antigen, density of 1.16 g/cm^3, and chloroform sensitivity, and could be classified in the *N*-tropic Gross-AKR subgroup. FBJ-MSV induced foci of cell alteration in rat and mouse tissue-culture cells which differed morphologically and temporally from those produced by other MSV's. The foci formed in mouse embryo cells had the unusual capacity to propagate independently. The non-focus-forming MLV did not produce leukemia or any other pathological disease after inoculation into hamsters, rats, or several strains of mice. Apparently, indigenous MLV is common to the CF-1 mice in which the FBJ-MSV originated, where it appears to be a nonpathogenic MLV or, possibly, a nonpathogenic mutant of FBJ-MSV.

2642 A NEW GROUP OF ONCOGENIC VIRUSES: RETICULOENDOTHELIOSIS, CHICK SYNCYTIAL, DUCK INFECTIOUS ANEMIA, AND SPLEEN NECROSIS VIRUSES. *(E.)* Purchase, H. G. (Reg. Poultry Res. Lab., U.S. Dept. Agriculture, East Lansing, Mich.), C. Ludford, K. Nazerian and H. W. Cox. *J Natl Cancer Inst* 51(2): 489-499, 1973.

This study was carried out to determine immunological relationships between reticuloendotheliosis virus (REV) isolated from turkeys, duck infectious anemia virus (DIAV) and spleen necrosis virus (SNV) isolated from ducks, and chick syncytial virus (CSV) isolated from chickens. In the fluorescent antibody test, involving secondary coverslip cultures of virus-infected chicken and duck embryo fibroblasts and immune chicken sera, the viruses were serologically indistinguishable. The viruses that did not usually kill their host namely, CSV, DIAV, and REV, produced resistance in ducks to subsequent challenge exposure to *Plasmodium* sp. In neutralization tests, neutralization was stronger in the homologous than in the heterologous system, thus showing some antigenic differences. These viruses were, therefore, members of a group of viruses named the reticuloendotheliosis group. The viruses of this groups differed morphologically from those of the leukosis/sarcoma group and they did not produce the latter's group-specific antigen. The SNV and some strains of REV were highly pathogenic for ducks and chickens, producing reticuloendotheliosis of the viscera, together with proliferative and infiltrative lymphoid nerve lesions in chickens. The DIAV and CSV were less pathogenic. Viruses of the reticuloendotheliosis group were generally poorly transmitted horizontally but more readily so in ducks than in chickens. In these experiments, flocks of turkeys, ducks, and geese, but not chickens, evidenced infection with viruses of this group.

2643 GROWTH CHARACTERISTICS OF BURKITT SOMATIC CELL HYBRIDS *IN VITRO*. *(E.)* Glaser, R. (Hershey Med. Ctr. Pennsylvania State U., Hershey), B. Decker, R. Farrugia, T. Shows and F. Rapp. *Cancer Res* 33(9):2026-2029, 1973.

The properties of Burkitt somatic cell hybrids (D98/HR-1) are compared to the two parental cell lines. The hybrid cells synthesize both the A and B forms of glucose 6-phosphate dehydrogenase, which were originally derived from the two fused human cells. The A form is from D 98 cells and the B form was supplied by the P3J-HR-1 cells. The formation of a heteropolymer of glucose 6-phosphate dehydrogenase in the hybrid proves that a hybrid was obtained. Selection of hybrid cells with lower numbers of chromosomes has taken place after several cell passages. There was some evidence that cells containing a higher number of Epstein-Barr virus (EBV) genome equivalents as measured by DNA-cRNA hybridization were being preferentially selected. If selection is partially based on rate of growth, it may be that those cells containing higher amounts of EBV DNA grow faster than those cells with less virus genome equivalents. The properties of the hybrid and parental cell lines differ in their ability to grow in soft agar, but all showed little dependence on serum concentration. One possible explanation for the results obtained in these studies may be that the EBV genome, once having become associated with the D98 cell genome, enhanced the attributes described and may be responsible for the changes in the properties of the resulting hybrid cells.

2644 MUSCLE DIFFERENTIATION IN CELL CULTURE. EFFECTS OF NUCLEOSIDE INHIBITORS AND ROUS SARCOMA VIRUS. *(E.)* Easton, T. G. (Rockefeller U., New York, N.Y.) and E. Reich. *J Biol Chem* 247(20): 6420-6431, 1972.

Some biochemical aspects of the differentiation of chick embryonic skeletal muscle cells in cell culture were studied. After 40-44 hr of culture in a medium that supports cell fusion, the normal onset of cell fusion was prevented by exchanging the medium for one low in Ca^{++} (80μM). After a further 24 hr, high Ca^{++} (880μM) medium was restored. Cell fusion proceeded, followed by synthesis of creatine kinase and myokinase. In cultures treated with 5-flourouridine before cell fusion was allowed to proceed, fusion and synthesis of creatine kinase and myokinase proceeded as in control cultures. In cultures treated for 8 hr with 5-bromotubercidin, cell fusion and synthesis of creatine kinase and myokinase lagged about 16 hr behind control cultures. Continuing synthesis of ribosomal RNA did not appear to be necessary for expression of differentiated muscle properties, i.e. cell fusion and enzyme synthesis. After cultures were fed high Ca^{++} medium there must have been synthesis of one or more messenger type RNA's before differentiated properties were expressed. Also it seemed that synthesis and translation to protein of the messenger RNA's needed for the expression of differentiated muscle properties did not require concomitant synthesis of ribosomal RNA. Cultures of differentiating muscle cells infected with the RNA tumor virus Schmidt-Ruppin RSV were also studied. Forming myotubes, or multinucleated muscle fibers, became progressively vacuolated and were destroyed by 72 hr postinfection. Cell fusion and synthesis of creatinine kinase and myokinase in myotubes continued for two or three days after infection, however. The degenerative response of myotubes to infection was contrasted with the response of fibroblasts; fibroblasts assume abnormal growth pattern after virus infection, while myotubes, which cannot grow or divide, simply die.

2645 "EARLY" VIRUS-SPECIFIC RNA MAY CONTAIN INFORMATION NECESSARY FOR CHROMOSOME REPLICATION AND MITOSIS INDUCED BY SIMIAN VIRUS 40. *(E.)*

May, E. (Inst. Sci. Res. Cancer, Villejuif, France), P. May and R. Weil. *Proc Natl Acad Sci USA* 70(6):1654-1658, 1973.

Studies were conducted on the mode of synthesis and molecular and biological properties of "early" SV40 RNA (i.e., virus-specific RNA synthesized and processed during abortive infection). The system used was SV40-infected confluent mouse kidney cultures in which intranuclear T-antigen is synthesized followed by replication of mouse-cell chromatin and mitosis without production of viral progeny DNA or capsid protein. Cultures of infected MK cells were labeled with ^{3}H-uridine at various times following infection and hybridized with SV40 DNA. It was thus possible to measure the amount of radioactive, hybridizable, virus-specific RNA in each sample. On parallel cultures the appearance of SV40-specific T-antigen was studied by the immunofluorescence reaction and the time course of SV40-induced cellular (chromosomal) DNA synthesis was studied by the use of autoradiography. The results suggest that the initial increase in the amount of early SV40 RNA measured, which precedes by 2-3 hr the appearance of T-antigen, is the expression of the asynchronous onset of the synthesis of early SV40 RNA and that the subsequent rapid decrease may reflect the switch-off of transcription, linked to SV40-induced replication of mouse cell chromatin. The experimental results reveal a striking temporal and quantitative relation between early SV40 RNA and subsequent events of the abortive infection. This possibility is indirectly supported by the observation that treatment of confluent MK cultures with a mouse-specific interferon before (but not after) infection depresses by as much as 70% the synthesis of early SV40 RNA and results in a comparable reduction in the number of T-antigen-positive nuclei and in the number of cells participating in SV40-induced cellular DNA synthesis and mitosis.

2646 TWO TYPES OF TARGET CELLS FOR TRANSFORMATION WITH AVIAN MYELOCYTOMATOSIS VIRUS. *(E.)*

Graf, T. (Dept. Microbiol. Immunol., Duke U. Med. Ctr. Durham, N.C.). *Virology* 54(2):398-413, 1973.

A strain, MCV-B, was isolated from avian myelocytomatosis virus MC29, by clone purification and found to belong to subgroup B of avian leukosis-sarcoma viruses. It contained a transforming virus defective for reproduction and an excess of a nontransforming virus. MCV-B retained the capacity of MC29 to transform hematopoietic chicken tissues at a high efficiency. It was also capable of reducing two types of foci and agar suspension colonies in cell cultures derived from 11-day-old chicken embryos. These alterations were shown to be due to a morphological transformation of two different types of target cells designated *s* and *l*. Several lines of evidence indicate that a single agent is able to transform both types of target cells. Pure cultures of *s*-type cells were obtained by cloning of uninfected chicken cells. They were highly susceptible to transformation with Rous sarcoma virus (RSV) but resistant to transformation with avian myeloblastosis virus (AMV) and myeloblastosis associated virus (MAV). Efforts to clone-purify uninfected *l*-type cells have failed so far. It is suggested that this type of cell is derived from the hematopoietic system and is the *in vitro* target cell for transformation with AMV.

2647 REPLICATION OF THE RESIDENT NON-DEFECTIVE SV40 IN TRANSFORMED HUMAN CELLS SUPERINFECTED WITH DNA FROM A TEMPERATURE-SENSITIVE SV40 MUTANT.

(E.) Dubbs, D. R. (Baylor Coll. Med., Houston, Texas) and S. Kit. *J Gen Virol* 19(1):103-111, 1973.

Cells of a strain of human embryonic lung cells transformed by SV40 and producing infectious SV40 (WI38 Val3A cells) spontaneously produced infectious virus which was temperature resistant (*tr*), i.e. replicated in monkey kidney cells at either 38.7 C or 33.5 C. Superinfection of WI38 Val3A cells with DNA from a temperature-sensitive (*ts*) SV40 mutant at the permissive temperature (33.5 C) did not enhance rescue of the resident *tr* SV40 genome. In related experiments, the competition between replicating superinfecting *ts* SV40 and endogenous *tr* SV40 was studied. It was determined that failure of superinfecting *ts* SV40 to enhance replication of resident *tr* SV40 was not due merely to a competitive advantage of *ts* SV40 over *tr* SV40. 1-β-D-arabinofuranosylcytosine treatment of WI38 Val3A cells did not enhance formation of the endogenous *tr* SV40. The experiments suggested that the resident SV40 is determined primarily by cellular rather than by superinfecting SV40 gene functions.

2648 THE POLYPEPTIDES OF ADENOVIRUS TYPE 12.

(E.) Russell, W. C. (Natl. Inst. Med. Res., London, England) and J. J. Skehel. *J Gen Virol* 20(2): 195-209, 1973.

Adenovirus type 12, in contrast to type 5, can induce tumors in baby hamsters. In attempts to obtain information on the factors responsible for the oncogenic properties of the type 12 virus, a comparative study was made of the structural polypeptides present in the two different types of virus and of the polypeptides synthesized in human embryonic kidney cells infected by the two types. The polypeptides were detected by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Synthesis of polypeptides in infected cells was followed by labeling with (^{35}S)-methionine and by autoradiography of polyacrylamide gels after electrophoresis of extracts of the infected cells. Six polypeptides were found in purified adenovirus type 12. One of these, the Y polypeptide, was not seen in type 5 virus and appeared to be associated with the core components rather than with the capsid. Incomplete type 12 virus particles lacked the core polypeptides, but were found to contain three other polypeptides. The nature and functions of the Y polypeptide of the complete virus and of the three additional polypeptides of the incomplete virus were not known. Structural polypeptides seen in the complete virus were also found in the extracts; in addition, there were at least eight other polypeptides in the extracts, which appeared to be specific for the infected cells. One of them, of about 55,000 molecular wt, was synthesized early in infection even in the presence of an inhibitor of DNA synthesis and was thus presumed to be related to the adenovirus type 12 T antigen. Phosphorylation of polypeptides was noted in the infected cells, and in the purified virus both fiber and core-1 polypeptides appeared to be phosphorylated. The finding of phosphorylated polypeptides with type 12 virus as well as with type 5 infection suggests that this pattern of phosphorylation may be characteristic of adenovirus infection. The relation of the various findings to the oncogenic potential of the type 12 virus is still a matter for speculation.

2649 HIGH MOLECULAR WEIGHT RNA IN A MURINE LEUKAEMIA VIRUS HELPER-INDEPENDENT STRAIN OF MOLONEY SARCOMA VIRUS.

(E.) Vidrine, J. G. (Pfizer Inc., Maywood, N.J.), K. R. Harewood, L. M. Bulfone, C. R. Higdon and S. A. Mayyasi. *J Gen Virol* 20(2): 239-242, 1973.

Certain cell types infected with and containing a rescuable murine sarcoma virus (MSV) genome in the absence of detectable murine leukemia virus (MLV) are referred to as sarcoma positive, leukemia negative (S+L-), as also is the noninfectious type C virus which is produced by such cells. S+L- virus, when isolated from clone C-243-3 of 3T3 Moloney MSV-transformed cells, was found previously to contain low molecular weight RNA (4, 18 and 28S species) and also low levels of RNA-dependent DNA polymerase (RDDP). In the present work an S+L- virus isolated from another clone of 3Y3 cells, namely, clone D-56, was found to contain high molecular weight RNA (60 to 70S species) and to possess 50- to 10-fold more DDP than that possessed by virus from the C-243-3 clone. It was concluded that factors other than absence of high molecular weight RNA or adequate levels of RDDP are responsible for the lack of infectivity of S+L- strain of MSV.

2650 SINGLE-STRANDED REGIONS IN REPLICATING DNA OF ADENOVIRUS TYPE 2.

(E.) Robin, J. (U. Hosp. Ctr., U. Sherbrooke, Quebec, Canada), D. Bourgaux-Ramoisy and P. Bourgaux. *J Gen Virol* 20(2):233-237, 1973.

A new species of DNA, designated DNA-R, was isolated from KB cells 24 hr after infection with adenovirus type 2 (ad. 2). It was separated from regular, mature DNA by fractionation on benzoylated-naphthoylated-DEAE (BND) cellulose. It was observed that the DNA-R was labeled preferentially after short pulses of radioactive thymidine, that the label could be chased from DNA-R into mature DNA and that the DNA-R could be hybridized with DNA extracted from ad. 2 virus, but not KB cell DNA. Furthermore, DNA-R had the same density as ad. 2 DNA in cesium chloride solution and sedimented in neutral sucrose solution as expected for linear duplexes, being one of two times the size of ad. 2 DNA, and it was eluted from BND-cellulose in a fashion characteristic of partly single-stranded or replicating DNA. These observations indicated that DNA-R represented linear molecules of ad. 2 in the act of replication. In the present work, information was reported on the effect on the replicating DNA and on the mature DNA of a nuclease from *Neurospora crassa* conidia, which was highly specific for single-stranded nucleic acids. Using ^{3}H-thymidine-labeling and studying the preparations by sedimentation in linear sucrose gradients both before and after nuclease treatment, it was found that the enzyme, while exerting no detectable effect on mature virus DNA, altered the properties of DNA-R in at least two respects. First, nuclease-treated DNA-R was recovered in the mature DNA fraction after chromatography on BND-cellulose. Secondly, a change in the sedimentation properties indicative of fragmentation of the DNA-R molecules was observed. The results were consistent with the assumption that DNA-R represents molecules of ad. 2 DNA replicating unidirectionally.

2651 INHERITANCE OF SUSCEPTIBILITY TO FRIEND MOUSE LEUKEMIA VIRUS. XI. GENETIC CONTROL OF LATE SPLENOMEGALY.

(E.) Odaka, T. (Inst. Med. Sci. Tokyo, Japan). *Int J Cancer* 12(1):124-128, 1973.

Genetic control of splenomegaly which occurs at a late stage of Friend leukemia virus infection was studied in hybrid mice between DDD-Fv^r and C57BL/6. It was demonstrated that this late splenomegaly was mainly controlled by a single autosomal locus with the dominant allele for resistance. This locus may be identical with or genetically linked with the locus which controls the level of virus multiplication at an early stage of infection. Considered together with previous findings, splenomegaly induced by Friend leukemia virus in mice is classified into two types, early and late, both being controlled by two separate gene loci, and scarcely affected by additional gene loci.

2652 LATENT INFECTIONS INDUCED BY HERPES SIMPLEX VIRUSES. *(E.)* Stevens, J. G. (Reed Neurol. Res. Ctr., U. California, Los Angeles) and M. L. Cook. *Cancer Res* 33(6):1399-1401, 1973.

Mice inoculated intradermally in the rear footpad and rabbits inoculated on the cornea with either herpes simplex virus type 1 or type 2 develop chronic latent infections following an acute episode. The infection is restricted to sensory neurons as determined from the ability of dissected nerves to replicate infectious virus on RK_{13} cell monolayers. The latent infection persists for at least four months on mice, and rabbits experience recurrent eye infections. Attempts to reactivate either infectious virus or clinical evidence of disease in mice have been unsuccessful. Morphological and immunological evidence suggests that latent virus is selectively harbored in the neurons and is not present in surrounding tissues. All evidence is consistent with the "static state" hypothesis for recurrent herpes simplex infection in man. According to this hypothesis, virus is maintained in "virogenic" cells, with complete viral replication reversibly interrupted at an early state in the cycle.

2653 SIMIAN HERPESVIRUSES. *(E.)* Deinhardt, F. (Rush-Presbyterian-St. Luke's U. Illinois Med. Ctr., Chicago), L. A. Falk and L. G. Wolfe. *Cancer Res* 33(6):1424-26, 1973.

Two oncogenic simian herpesviruses, *H. saimiri* (HVS) and *H. ateles* (HVA), are discussed and compared and some of the current, pertinent literature is reviewed. The natural hosts of the two viruses, squirrel and spider monkeys, resp., are infected early in life and become lifelong carriers. Experimentally, both HVS and HVA induce fatal malignant lymphoproliferative disease in several nonhuman primate genera other than their natural hosts. Neither virus nor virus proteins can be demonstrated in carrier hosts or in virus infected lymphoma-bearing animals; however, cultivation of such cells *in vitro* results in rapid expression of viral antigens and infectious virus. Studies of permanent cell lines established from lymphomas or from abnormal peripheral lymphocytes from virus-infected animals have indicated that the disease is multiclonal, that these lines always produce a small amount of virus, and that degenerating producer cells contain virus antigens which are expressed in a fashion very similar to that of Epstein-Barr virus (EBV) in lines established from infectious mononucleosis and Burkitt's lymphoma patients. Recent serological studies suggest a cross-reactivity between HVS and HVA. In addition, HVS-infected cells contain at least two and possibly three different antigens which may be analogous to the early and late antigens of EBV. Virus expression *in vivo* and *in vitro* and antibody responses of infected hosts are almost identical for HVS and EBV and probably also for HVA.

2654 NUCLEIC ACID PRECURSOR UPTAKE PATTERNS IN SHOPE VIRUS INDUCED PAPILLOMA AND Vx-2 CARCINOMA. *(E.)* Rao, P. R. (Dept. Zool., Osmania U., Warangal-I, Andhra Pradesh, India). *Indian J Med Res* 61(6):833-837, 1973.

It would appear that for a meaningful chemotherapy only those antimetabolites should be used whose normal counterparts are extensively used by the tumor but not by normal cells. A simple chromatographic system was adopted in this study to measure quantitatively the amount of precursor taken up by tumor cells when incubated in a nutrient medium containing at physiological concentration all the nucleic acid precursors. Slices of young and vigorously growing tissue from Shope virus induced rabbit papilloma and Vx-2 tumor were hashed and incubated in TC 199 medium containing specific nucleic acid precursors. After incubation the medium was analyzed on a Dowex anion exchange resin. An increase in the concentration of deoxyuridine in the medium was observed. No such increase was noted with normal or embryonic rabbit skin. The uptake of thymine was high in both tumor tissues, and Vx-2 cells further acquired the ability of taking up uracil from the medium whereas normal cells did not utilize these precursors from the medium. Deoxycytidine and thymidine were, however, taken up in comparable amounts in both normal and tumor tissues. This study demonstrates, therefore, that the use of deoxyuridine or uridine metabolites for chemotherapy of tumors which release or do not use these precursors is of no value.

2655 ONCOGENIC POTENTIAL OF MURINE C-TYPE RNA VIRUS PASSAGED DIRECTLY FROM NATURALLY OCCURRING TUMORS OF THE BALB/cCr MOUSE. *(E.)* Peters, R. L. (Microbiol. Assoc., Walkersville, Md.), G. J. Spahn, L. S. Rabstein, G. J. Kelloff and R. J. Heubner. *J Natl Cancer Inst* 51(2):621-630, 1973.

Tumors of various origins, namely, lymphoreticular (LR), mammary, sarcomatous, and benign, appear spontaneously in BALB/cCr mice. Group specific (gs) antigen found in spleen and tumor tissues of these tumor-bearing mice related to C-type virus and suggested an etiologic role for the virus. This information led to a study of the oncogenic potential of murine C-type RNA virus passaged directly from the various types of naturally occurring tumors of the BALB/cCr mouse. A total of 1954 BALB/cCr mice were inoculated at birth with 141 viral preparations and observed up to 24 months. Of the different tumors, 51% yielded virus. Significant induction of LR neoplasms was observed in animals given virus-positive inocula, rather than virus-negative or no inocula. Solid non-LR neoplasms that occur naturally in the BALB/cCr mouse were not induced. Thus, LR tumors were always induced regardless of type of tumor from which the viral inocula were derived. Furthermore, route of inoculation did not affect the results. The mean latent period of LR neoplasm induction was quite long (469 days), but was significantly different ($P < 0.05$) from that of naturally occurring LR neoplasms (634 days). Results were discussed in light of the oncogene hypothesis.

2656 ACTIVATION OF LEUKEMIA VIRUSES BY GRAFT-VERSUS-HOST AND MIXED-LYMPHOCYTE-CULTURE REACTIONS: ELECTRON MICROSCOPIC EVIDENCE OF C-TYPE PARTICLES. *(E.)* Andre-Schwartz, J. (New England Med. Ctr., Hosp., Boston, Mass.), R. S. Schwartz, M. S. Hirsch, S. M. Phillips and P. H. Black. *J Natl Cancer Inst* 51(2):507-518, 1973.

A possible link between the immune response and malignant lymphomas was previously uncovered by the finding that leukemia viruses are activated *in vivo* during chronic graft-versus-host (GVH) reaction in mice and *in vitro* during the immune response in the mixed-lymphocyte-culture (MLC) reaction. In a further study, murine leukemia viruses have now been detected by the mixed cell cytopathogenicity (XC) test using a combined electron microscopic and virologic analysis during the GVH and MLC reactions. The experimental design consisted of administering 4 i.p. weekly injections of parental BALB/c spleen cells to (BALB/c x A/J)F_1 hybrid mice and killing the mice 1 wk after the last injection. C-type particles were seen in positive XC test plates, in the spleens of mice undergoing the GVH reaction, and in the MLC. Viruses were not found in any control group, including mice undergoing immune responses other than the GVH reaction. The specific association of the viruses with transformed lymphocytes suggests that the type of response represented by GVH and MLC reactions can activate leukemia viruses by a specific immunologic mechanism.

2657 A COMPARATIVE STUDY OF THE BIOLOGIC AND MOLECULAR BASIS OF MURINE MAMMARY CARCINOMA: A MODEL FOR HUMAN BREAST CANCER. *(E.)* Schlom, J. (Columbia U. Coll. Physicians Surgeons, New York, N.Y.), R. Michalides, D. Kufe, R. Hehlmann, S. Spiegelman, P. Bentvelzen and P. Hageman. *J Natl Cancer Inst* 51(2):541-551, 1973.

The technique of molecular hybridization was used in a comparative study of the molecular and biological basis of murine mammary neoplasia as a model for the human disease. ^{3}H-DNA complementary to mouse mammary tumor virus (MMTV) RNA was synthesized in an endogenous reaction with MMTV particles. This DNA was used as a probe via molecular hybridization to establish the presence of MMTV-specific RNA in cellular RNA of "spontaneous" mammary tumors of several strains of mice, including the "nonproducer" BALB/c mammary tumors. Hybridization of MMTV-^{3}H-DNA to cellular RNA in these tests was scored as "positive" or "negative" according to amounts of ^{3}H-DNA hybridized to the larger cellular RNA molecules. MMTV-specific RNA was also found in certain normal tissues (spleen, kidney, and epididymis) of a high-mammary-cancer strain (GR). Aging or treatment with nonviral carcinogens (300 rads irradiation or 0.15% urethane in drinking water) also induced the appearance of MMTV-specific RNA in certain normal tissues of the low-mammary-cancer strains, C57BL and BALB/c. The relationship of the presence of MMTV-specific RNA to the etiology and pathogenesis of murine mammary neoplasia and its potential application to human breast cancer are discussed.

2658 MALIGNANT TRANSFORMATION OF HAMSTER EMBRYO FIBROBLASTS FOLLOWING EXPOSURE TO ULTRA-VIOLET-IRRADIATED HUMAN CYTOMEGALOVIRUS. *(E.)* Albrecht, T. (Milton S. Hershey Med. Ctr. Pennsylvania State U., Hershey) and F. Rapp. *Virology* 55(1):53-61, 1973.

To determine whether members of the herpes group of viruses other than Epstein-Barr virus and herpes simplex type 2 virus might be capable of oncogenic transformation of mammalian cells *in vitro*, the present study with cytomegalovirus was carried out. Clones of noncontact inhibited hamster embryo fibroblasts were first isolated following exposure to UV-irradiated cytomegalovirus (CMV). A continuous cell line (Cx-90-3B) was then established from one clone, which proved oncogenic when inoculated into weanling golden Syrian hamsters. CMV antigens could be demonstrated in the cytoplasm and on the surface of Cx-90-3B cells using human convalescent sera. Furthermore, serum from tumor bearing animals was shown to contain antibody to CMV-specified antigens. These observations are similar to those previously reported for herpes simplex viruses and represent the first demonstration that human CMV has transforming potential in mammalian cells.

2659 ISOLATION AND CHARACTERIZATION OF REVERTANT CELL LINES. II. GROWTH CONTROL OF A POLY-PLOID REVERTANT LINE DERIVED FROM SV40-TRANSFORMED 3T3 MOUSE CELLS. *(E.)* Pollack, R. (Cold Spring Harbor Lab., N.Y.) and A. Vogel. *J Cell Physiol* 82(1):93-100, 1973.

A polyploid revertant line FlSV101, isolated from an SV40-transformed 3T3 mouse fibroblast culture, was previously shown to have a low maximum cell density in spite of the continued presence in the revertant of SV40-specific DNA, RNA and T-antigen. The mechanism is now examined by which the revertant maintains a low saturation density. The fraction of cells in mitosis was determined by exposure of cells to Velban, trypsinization, fixation, staining and microscopic examination of at least 1000 nuclei. DNA synthesis was followed by incorporation of ^{32}P-phosphate or ^{3}H-thymidine, and determination of fraction of cells synthesizing DNA was carried out by radioautography of incorporated ^{3}H-thymidine. It was found that at confluence the revertant line FlSV101 and the original fibroblast line 3T3 were both reduced in mitotic index, fraction of cells synthesizing DNA, and rate of DNA synthesis. It appeared that the low saturation density of FlSV101 and 3T3 could not be due to a balance between continued DNA synthesis at the rate found in logarithmically growing cells and cell death, since none of the lines shed more than 2% of the cells per day.

2660 MOUSE MAMMARY TUMOUR VIRUS RNA-DEPENDENT DNA POLYMERASE: REQUIREMENTS AND PRODUCTS. *(E.)* Dickson, C. (Imperial Cancer Res. Fund, London, England). *J Gen Virol* 20(2):243-247, 1973.

Because mouse mammary tumor virus (MTV) differs from other members of the RNA tumor virus group with respect to its morphology and its antigenicity, an investigation was carried out to determine whether the requirements and products of its RNA-dependent DNA polymerase might also differ. It was found that the enzyme required a divalent cation, all four deoxynucleotide triphosphates, dithiothreitol and a nonionic detergent to elicit activity. Further, the initial product appeared to be a rapidly sedimenting RNA:DNA hybrid, followed after longer incubation by a predominantly slow sedimenting DNA product. These results for the MTV polymerase were similar to those obtained with other RNA tumor viruses, despite the unique characteristics of MTV with respect to morphology and antigenicity.

2661 DECREASE OF SENSITIVITY TO INTERFERON IN CELLS INFECTED BY MURINE LEUKEMIA-SARCOMA VIRUSES. *(E.)* Peries, J. (Inst. Leukemia Res., St. Louis Hosp., Paris, France), M. Canivet, M. Olivie, M. Salle and M. Boiron. *J Natl Cancer Inst* 51(2):455-463, 1973.

Decreased sensitivity of cells of the murine-sarcoma virus complex to protection by interferon against infection by certain viruses such as Newcastle virus was previously reported. The present work deals with further details and with new information about the DSI mechanism. The relationship of DSI to actual infection by Moloney murine sarcoma virus (MSV-M) was ascertained by specific virus neutralization and physical inactivation of the virus. DSI, which did not depend on the type of challenge virus testing interferon activity, began 3 hr after the end of MSV-M adsorption and was clearly established after 6 hr. It was observed both when MSV-M was inoculated before treatment of cells by interferon or after cells were submitted to the action of interferon for 8 hr. The phenomenon was not related to direct inhibitory action of the viral preparations, loss of the protective state due to accelerated cell growth, influence of viral replication on protein synthesis by cells, or stimulation of challenge virus multiplication. No substance counteracting the action of interferon was found in MSV-M-infected cells. The murine leukemia-sarcoma viruses may work against the postulated translation inhibitory protein, perhaps at the level of its mRNA.

2662 PREVALENCE OF TYPE-C VIRUS AND ANTIBODIES IN NORMAL CATS AND CATS WITH NEOPLASIA. *(E.)* Riggs, J. L. (Viral Rickettsial Dis. Lab., California State Dept. Public Hlth., Berkeley), L. S. Oshiro, D. O. N. Taylor and E. H. Lennette. *J Natl Cancer Inst* 51(2):449-454, 1973.

The correlation between neoplasia and prevalence of type-C virus and antibodies was tested in selected groups of cats with sarcomas or lymphomas, cats with non-neoplastic disease, and normal cats. Cats with suspected neoplasia were bled before euthanasia and a complete necropsy was performed. Samples of different tissues were obtained for electron microsocopy and for establishment of cell cultures. Of these cats, 83 of 122 had some type of neoplasia; the remainder had other non-neoplastic diseases. Examination of tissues by electron microscopy showed type-C virus in 58% of the cats with neoplasia and in 44% of those with non-neoplastic diseases. None of the tissues obtained from 11 "normal" cats was positive for type-C virus. It was suggested that the stress of the non-neoplastic process might have enabled a latent type-C virus to express itself as a fully competent entity. Alternatively, some of the positives may not have been the type-C leukemia-sarcoma virus but the feline syncytium-forming agent. Sera from 102 of the cats were examined by the indirect fluorescent antibody (IFA) procedure for antibodies against feline leukemia virus (FeLV). The sera from 41% of the cats with non-neoplastic diseases were positive, whereas sera from 33% of the cats with neoplastic diseases were positive. In contrast, 5% of "normal" cat sera was positive in the IFA procedure against FeLV. The presence of antibodies correlated with the presence of type-C particles in the same animal in only approximately 50% of the cats examined, possibly reflecting an impaired immune mechanism in some animals. The presence of antibody in 5% of the normal cats might be ascribed to previous transient appearance of type-C particles. Continuous cell cultures were established from involved tissues of 10 of the cats, and 6 of the 10 continued to produce type-C virus *in vitro*. Cell-free extracts from some tumors and from some of the cell cultures producing type-C virus produced neoplasia when inoculated into fetal or newborn kittens.

2663 TUMOR-SPECIFIC TRANSPLANTATION ANTIGENS IN RETICULUM CELL SARCOMAS AND LYMPHOMAS INDUCED BY THE FRIEND VIRUS COMPLEX. *(E.)* Fieldsteel, A. H. (Division Life Sci., Stanford Res. Inst., Menlo Park, Calif.), P. J. Dawson and C. Kurahara. *Cancer Res* 33(3):551-558, 1973.

2664 STUDIES ON THE MOUSE MAMMARY TUMOUR AGENT (MTA). IV. LACTATE AND MALATE DEHYDROGENASE ISOZYMES IN NORMAL MAMMARY GLAND OF RIIIf, C57BL AND IN VIRUS-INDUCED AND SPONTANEOUS MAMMARY ADENOCARCINOMA IN C57BL AND RIII MOUSE STRAINS. *(E.)* El-Fiky, S. (Med. Res. Inst., Alexandria, Egypt). *Acta histochem (Jena)* 46(1):136-139, 1972.

2665 PROPERTIES OF MOUSE LEUKEMIA VIRUSES. VI. THE CORE OF FRIEND VIRUS; ISOLATION AND CONSTITUENTS. *(E.)* Lange, J. (Max-Planck-Inst. Tübingen, Germany), H. Frank, G. Hunsmann, V. Moennig, R. Wollmann and W. Schafer. *Virology* 53(2):457-462, 1973.

2666 CYTOTOXICITY INDUCED BY MIXTURES OF ADENOVIRUS AND SPECIFIC ANTIBODIES. INDUCTION OF CYTOTOXIC AGGREGATES BY HOMOTYPIC ANTI-FIBRE SERA AND NEUTRALIZATION OF THE CYTOTOXICITY BY HOMOTYPIC ANTI-HEXON SERA. *(E.)* Ankerst, J. (Inst. Med. Microbiol., U. Lund, Sweden) and L. Kjellén. *Arch Gesamte Virusforsch* 41(1-2):99-105, 1973.

2667 VIRUS-LIKE PARTICLES IN HUMAN LEUKEMIC CELLS. *(E.)* Torrioli, M. (G. Mendel Inst. Med. Genetics, Rome, Italy), G. Torrioli-Riggio and L. Braconi-Romei. *Acta Genet Med Gemellol (Roma)* 21(3):271-273, 1972.

2668 THE ORIGIN OF PHAGES OCCURRING IN *IN VITRO*-CULTIVATED CROWN-GALL TISSUES. *(Fr.)* Tourneur, J. (Natl. Agric. Res. Ctr., Versailles, France), D. Garnier-Expert and G. Morel. *C R Acad Sci [D] (Paris)* 276(15):2265-2267, 1973.

2669 MULTINUCLEATED GIANT CELL FORMATION IN CULTURED CELLS BY MURINE LEUKEMIA VIRUS INFECTION. *(E.)* Koga, H. (Kyushu U., Katakasu, Fukjoka, Japan). *Gann* 64(3):321-322, 1973.

2670 BIOCHEMICAL ANALYSIS OF GROSS VIRUS SPECIFIC TUMOR ANTIGENS. *(E.)* Moroni, Ch. (Friedrich Miescher Inst., Basel, Switzerland). *Experientia* 29(6):754, 1973.

2671 CHARACTERIZATION OF A FIBROMA VIRUS ISOLATED FROM NATURALLY OCCURRING SKIN TUMORS IN DOMESTIC RABBITS. *(E.)* Raflo, C. P. (Ohio State U., Columbus), R. G. Olsen, S. P. Pakes and W. S. Webster. *Lab Anim Sci* 23(4):525-532, 1973.

2672 C-TYPE VIRUS PARTICLES IN AVIAN BURSA OF FABRICIUS CELLS GROWN *IN VITRO*. *(E.)* Nagi, S. A. (Texas A & M U., College Station,). C. F. Hall, R. W. Storts and C. A. Carson. *Am J Vet Res* 34(5):711-712, 1973.

2673 ANTITUMOUR ACTIVITY *IN VITRO* DEPENDENT ON IMMUNE B LYMPHOCYTES. *(E.)* Lamon, E. W. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), H. Wigzell, B. Anderson and E. Klein. *Nature [New Biol]* 244(137):209-211, 1973.

2674 EFFECTS OF INTERFERON ON LEUKAEMIA VIRUS ACTIVATION IN GRAFT VERSUS HOST DISEASE. *(E.)* Hirsch, M. S. (Harvard Med. Sch., Boston, Mass), D. A. Ellis, M. R. Proffitt, P. H. Black and M. A. Chirigos. *Nature [New Biol]* 244(134):102-103, 1973.

2675 NEUROLOGICAL MANIFESTATIONS OF BURKITT'S LYMPHOMA IN GHANA. *(E.)* Nkrumah, F. K. (U. Ghana Med. Sch., Accra) and I. V. Perkins. *Afr J Med Sci* 4(2):209-214, 1973.

2676 PROTECTION BY IMMUNE SPLEEN CELLS AGAINST CHALLENGE WITH MOLONEY SARCOMA VIRUS. EFFECT OF STAGE OF DONOR RESPONSE. *(E.)* Anonymous. *Transplantation* 15(5):510-513, 1973.

2677 URIDINE KINASE LEVEL IN CELLS INFECTED WITH WESTERN EQUINE ENCEPHALOMYELITIS, VESICULAR STOMATITIS, PSEUDORABIES AND POLYOMA VIRUSES. *(E.)* Rada, B. (Inst. Virol., Slovak Acad. Sci., Bratislava, Czechoslovakia) and J. Zemla. *Acta Virol* 17(2):111-115, 1973.

2678 *IN VITRO* EFFECTS OF SILICOTUNGSTATE ON SOME RNA VIRUSES. *(E.)* Jasmin, C. (Paul-Brousse Hosp., Villejuif, France), N. Raybaud, J. C. Chermann, D. Haapala, F. Sinoussi, C. B. Loustau, C. Bonissol, P. Kona and M. Raynaud. *Biomedicine* 18(4):319-327, 1973.

2679 DYNAMICS OF ACCUMULATION OF CAPSID PROTEINS OF TYPE 1 ADENOVIRUS. IMMUNOFLUORESCENCE AND CYTOFLUOROMETRIC DATA. *(E.)* Dyachenko, N. S. (D. K. Zabolotny Inst. Microbiol., Virol., Acad Sci. Ukrainian SSR, Kiev), L. N. Nosach, K. P. Gushcha and N. P. Vantsak. *Acta Virol* 17(2):116-123, 1973.

2680 KARYOLOGICAL ANALYSIS OF ROUS SARCOMA VIRUS-PRODUCING RAT-KANGAROO CELL LINES. *(E.)* Donner, L. (Inst. Exp. Biol., Genetics., Czechoslovak Acad. Sci., Prague), H. Sainerova and J. Svoboda. *Cytogenet Cell Genet* 12(2):117-127, 1973.

2681 FURTHER EVIDENCE FOR TWO LOCI WHICH CONTROL SUSCEPTIBILITY OF FOWL TO RSV(RAV-0). *(E.)* Pani, P. K. (Houghton Poultry Res. Station, Huntingdon, England) and L. N. Payne. *J Genet Virol* 19(2):235-244, 1973.

2682 CELL-FREE PREPARATIONS OF MAREK'S DISEASE VIRUS FROM POULTRY DUST. *(E.)* Hlozanek, I. (Inst. Exp. Biol., Genetics, Czechoslovak Acad. Sci., Prague), O. Mach and V. Jurajda. *Folia Biol* 19(2):118-123, 1973.

2683 RNA-DEPENDENT RNA POLYMERASE OCCURRING IN ROUS SARCOMA VIRUS VIRIONS. *(Rus.)* Perevozchikov, A. P. (N. N. Petrov Sci. Res. Inst. Onkol., Leningrad, USSR), O. K. Kuznetsov, Yu. P. Zerov, G. F. Pluzhnikova, and I. F. Seyts. *Dokl Akad Hauk SSR* 209(6):1445-1448, 1973.

2684 SOME ASSUMPTIONS CONCERNING THE ORIGIN OF "CANCER" GENES OF THE CARCINOGENIC VIRUSES. *(Rus.)* Altshteyn, A. D. (Moscow, USSR). *Vopr Onkol* 19(4):100, 1973.

2685 MECHANISMS OF VIRAL CARCINOGENESIS. *(Rus.)* Shlyankevich, M. A. (Moscow, USSR). *Vopr Onkol* 19(4):99, 1973.

2686 POLYPEPTIDE SPECIFIC FOR CELLS WITH ADENOVIRUS 2-SV40 HYBRID AD2+ND_1. *(E.)* Lopez-Revilla, R. (Salk Inst. Biological Studies, San Diego, Ca.) and G. Walter. *Nature* [*New Biol*] 244(136): 165-167, 1973.

2687 ESTABLISHMENT AND CHARACTERIZATION OF ETHIDIUM BROMIDE RESISTANCE IN SIMIAN VIRUS 40-TRANSFORMED HAMSTER CELLS. EFFECTS ON MITOCHONDRIA *IN VIVO*. *(E.)* Klietmann, W. (Med. Fac. Rhein-Westfalen Technical Coll., Aachen, Germany), N. Sato and M. M. K. Nass. *J Cell Biol* 58(1):11-26, 1973.

2688 THE *IN VITRO* GROWTH OF BONE MARROW CELLS FROM RAUSCHER LEUKEMIA VIRUS INFECTED MICE. *(E.)* Van Griensven, L. J. L. D. (Dept. Exp. Pathol., Med. Fac., Rotterdam, Netherlands), E. V. Hull and M. J. De Vries. *Biomedicine* 19(4):138-141, 1973.

2689 ELECTRON MICROSCOPIC DETECTION OF SIMIAN-TYPE VIRUS PARTICLES IN HUMAN MILK. *(E.)* Chopra, H. (Natl. Cancer Inst., Bethesda, Md.), P. Ebert, N. Woodside, J. Kvedar, S. Albert and M. Brennan. *Nature* [*New Biol*] 243(126):159-160, 1973.

2690 IMMUNITY TO VIRAL LEUKEMIA IN RATS AND THE ROUTES OF TRANSMISSION FROM MOTHER TO OFFSPRING. *(E.)* Ioachim, H. L. (Lenox Hill Hosp., New York, N.Y.), S. E. Keller, M. L. Gimovsky and S. Shepherd. *Cancer Res* 33(3):547-550, 1973.

See also:

* (Rev): 2402, 2404, 2406, 2412, 2417, 2418, 2428, 2434, 2437, 2438, 2439, 2440, 2441, 2442, 2443, 2444, 2445, 2446, 2458, 2463, 2472
* (Chem): 2509, 2536
* (Immun): 2717, 2719, 2725, 2727, 2730, 2731, 2733, 2751, 2753, 2754, 2759, 2767, 2768, 2800, 2816, 2822, 2823, 2855, 2858, 2859
* (Epid-Biom): 2917

2691 RIBONUCLEIC ACID-DEPENDENT RIBONUCLEIC ACID REPLICASE IN THE IMMUNE RESPONSE. *(E.)* Saito, K. (Sch. Med., Gunma U., Maebashi, Japan) and S. Mitsuhashi. *Jap J Microbiol* 17(2):117-121, 1973.

The authors showed previously that serial passive transfers of immune ribonucleic acid (iRNA) were successful in immunizing animals against infection with *Salmonella enteritidis* and in evoking antibodies against *Salmonella* flagella, suggesting that the iRNA could replicate actively in the recipient cells. The presence of RNA-dependent RNA replicase in the immune response is tested in the present work, using iRNA as a template. An iRNA preparation was made using phenol extracts of spleens of mice previously immunized with *Salmonella tennessee* flagella. An enzyme extract, also prepared from the spleens of these mice, was found capable of inducing the incorporation of tritiated uridine (^{3}H-UTP) into the acid-insoluble fraction in a cell-free system in the presence of the iRNA. The enzyme activity could be demonstrated in the spleens of immunized mice but not in normal ones, showing the importance of stimulation with the corresponding antigen. The activity was inhibited by two derivatives of rifamycin, known to inhibit reverse transcriptase, but was not inhibited by actinomycin D, mitomycin C or bleomycin A_2, which are known inhibitors of nucleic acid polymerase. Treatment with ribonuclease or heating at alkaline pH resulted in a loss of activity in added iRNA. The ^{3}H-UTP-labeled product was found to be resistant to ribonuclease treatment but became sensitive when the product was subjected to heat treatment, suggesting that the ^{3}H-UTP-incorporated product formed a hybrid with iRNA. The results suggested that the enzyme was a ribonucleic acid-dependent ribonucleic acid replicase which induced the incorporation of UTP into the acid-insoluble fraction using iRNA as a template and that the product may be a newly synthesized RNA which forms a hybrid with iRNA. The authors conclude that this enzyme activity may play a role in the antibody formation process.

2692 PROMOTION OF REPLICATION IN LYMPHOID CELLS BY SPECIFIC THIOLS AND DISULFIDES *IN VITRO*. EFFECTS ON MOUSE LYMPHOMA CELLS IN COMPARISON WITH SPLENIC LYMPHOCYTES. *(E.)* Broome, J. D. (New York U. Sch. Med., New York) and M. W. Jeng. *J Exp Med* 138(3):574-592, 1973.

Numerous lines of mouse lymphoid tumors (13 of 22 tested) showed, with increased sensitivity, a property of normal mouse splenic lymphocytes, the potential for growth promotion *in vitro* by specific thiols added to standard culture media. For lymphoma L1210 (V), structure activity relationships were examined; 9 of 30 thiols promoted growth; the most active was α-thioglycerol, effective at 0.2 μM. Thiols became oxidized under conditions of tissue culture and had half-lives of less than 8 hr. Disulfides of active thiols promoted growth of lymphoma cells. The mitogenic response of splenic lymphocytes to lectins was increased by thiols-disulfides which promoted the growth of lymphoma cells, but the response varied with the mitogen preparation used and under some condtions thiols-disulfides were inhibitory. In contrast to L1210 (V), various other lymphoma cell lines show different responses to thiols-disulfides *in vitro*, some are capable of growing independently and some do not grow even if thiols-disulfides are present. But among the numerous lines which do depend on these substances there appear to be similar structure-activity relationships.

2693 ACTIVE SUPPRESSION OF IMMUNOGLOBULIN ALLOTYPE SYNTHESIS. III. IDENTIFICATION OF T CELLS AS RESPONSIBLE FOR SUPPRESSION BY CELLS FROM SPLEEN, THYMUS, LYMPH NODE, AND BONE MARROW. *(E.)* Herzenberg, L. A. (Stanford U. Sch. Med., Cal.), E. L. Chan, M. M. Ravitch, R. J. Riblet and L. A. Herzenberg. *J Exp Med* 137(6):1311-1324, 1973.

Thymus-derived cells (T cells) that actively suppress production of IgG2a immunoglobulins carrying the Ig-1b allotype were found in adult (SJL x BALB/c)F_1 mice exposed to anti-Ig-1b early in life. The suppression is specific for Ig-1b. The allelic product, Ig-1a, is not affected. Spleen, lymph node, bone marrow, or thymus cells from suppressed mice suppress production of Ig-1b by syngeneic spleen cells from normal F_1 mice. When a mixture of suppressed and normal cells in transferred into lethally irradiated BALB/c mice, there is a short burst of Ig-1b production after which the levels of Ig-1b fall rapidly and are not detectable. Pretreatment of the cells from the suppressed mice with antiserum specific for T cells (anti-Thy-1b) plus complement before mixture destroys the suppressing activity. Similar results with suppressor cells were obtained *in vitro* using Mishell-Dutton cultures. Mixture of spleen cells from suppressed animals with sheep erythrocyte-primed syngeneic normal spleen before culture suppresses Ig-1b plaque-forming cell formation while leaving Ig-1a PFC unaffected. Treatment of the suppressed spleen with anti-Thy-1b before transfer removes the suppressing activity.

2694 CYTOSTATIC ELIMINATION OF SYNGENEIC RAT TUMOR CELLS *IN VITRO* BY NONSPECIFICALLY ACTIVATED MACROPHAGES. *(E.)* Keller, R. (Immunobiol. Res. Group, U. Zurich, Switzerland). *J Exp Med* 138(3):625-644, 1973.

Syngeneic tumor cell lines induced in inbred DA rats by polyoma virus, dimethylbenzanthracene, or methylcholanthrene were interacted *in vitro* with syngeneic effector cells. Polymorphonuclear leukocytes and macrophages were the effector cells employed. Activated, non-immune macrophages or exudative polymorphonuclear leukocytes produced pronounced morphological changes in syngeneic tumor cells as evidenced by a substantial reduction in tumor cell numbers and appearance of shrunken cells, even though there was no increase in cell debris. Polymorphonuclear leukocytes exerted a generally similar but quantitatively much diminished effect. These effector cells constantly produced a decrease in the incorporation by tumor cells of DNA and RNA precursors. The effector cells were quite refractory to high doses of X-irradiation. Interaction of target cells with activated, nonimmune macrophages yielded low but consistent signs of cytotoxicity, whereas polymorphonuclear leukocytes gave no such effects. Elimination of functional macrophages by silica, an agent specifically toxic for macrophages, resulted in unrestricted tumor cell proliferation despite continued generation of cytotoxicity. Accordingly, cytostatic mechanisms appear to play a predominant role in the elimination of tumor cells by nonimmune phagocytes. Evidence from a variety of experimental approaches suggest that the cytostatic effect is dependent on cell-to-cell contact. These findings are consistent with the thesis that the nonspecific mechanisms heretofore associated with resistance to infection can contribute significantly to the hosts defense against tumors.

2695 TUMOR-ASSOCIATED SPECIFICITY OF SERUM-MEDIATED INHIBITION OF LYMPHOCYTE STIMULATION BY AUTOCHTHONOUS HUMAN TUMORS.

(E.) Vánky, F. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), J. Stjernswärd, G. Klein, L. Steiner and L. Lindberg. *J Natl Cancer Inst* 51(1):25-32, 1973.

The specificity of serum-mediated inhibition of lymphocyte stimulation by autochthonous tumor cells was investigated. The ability of tumor cells, preincubated with various sera, to stimulate autochthonous lymphocytes to increased DNA synthesis in the mixed-lymphocyte-target-interaction test was compared with untreated cells. In 31 of 37 tests (84%), autochthonous sera significantly inhibited the tumor cells' ability to stimulate autochthonous lymphocytes. Preincubation with allogeneic sera from sarcoma patients inhibited the stimulatory effect of sarcoma biopsy cells on autochtonous lymphocytes in 27 of 40 tests (68%). Allogeneic sera from patients with "unrelated" tumors (carcinomas) and from healthy controls inhibited 1 of 11 tests (9%) and in 5 of 33 (15%), resp. Fresh tumor cells that failed to stimulate autochthonous lymphocytes initially become active after elution of the cells under physiologic conditions or at low pH. The stimulatory activity of the eluted tumor cells was inhibited with autochthonous serum. The data suggest that tumors of the same general histologic origin share antigenic characteristics that are not equally represented on tumors derived from other tissues, or on normal tissues. A lack of stimulatory activity of certain tumor biopsies may be due to surface-bound immunoglobulins. These results confirm an earlier finding that autochthonous sera may inhibit the stimulating effect of biopsy specimens of sarcoma cells on the patients' own lymphocytes.

2696 QUANTITATIVE EFFECTS OF NUTRITIONAL ESSENTIAL AMINO ACID DEFICIENCY UPON IMMUNE RESPONSES TO TUMORS IN MICE.

(E.) Jose, D. G. (Variety Club Heart Hosp., Minneapolis, Minn.) and R. A. Good. *J Exp Med* 137(1):1-9, 1973.

Mice were fed diets deficient in a single essential amino acid, and the primary immune responses to inoculation of allogenic tumor cells was measured by *in vitro* assay of cellular immunity. Moderate reduction of the amino acids phenylalanine-tyrosine, valine, threonine, methionine-cystine, isoleucine, and tryptophan in the diet produced profound depression of hemagglutinating and blocking antibody responses, although cytotoxic cell-mediated immunity remained intact. These diets had previously been shown to result in a selective depression of tumor growth in mice. Limitation of the amino acids arginine, histidine, and lysine in the diets gave rise to only slight depression of the immune responses. These diets had previously been shown to produce a proportional decrease in both tumor growth and host body weight. Moderate leucine restriction resulted in a paradoxical depression of cytotoxic cell-mediated immunity with little effect on serum blocking activity. Slight increases had previously been noted in the weight of tumors in mice fed leucine-restricted diets. Deficiency or imbalance of essential amino acids in the diet may produce profound depression of immune responses and apparent, marked changes in the immune resistance of the host animal to tumors.

2697 SYNTHESIS, ASSEMBLY AND SECRETION OF γ-GLOBULIN BY MOUSE MYELOMA CELLS. V. BALANCED AND UNBALANCED SYNTHESIS OF HEAVY AND LIGHT CHAINS BY IgG-PRODUCING TUMORS AND CELL LINES.

(E.) Baumal, R. (Sick Children's Hosp., Toronto, Canada) and M.D. Scharff. *J Immunol* 111(2):448-456, 1973.

The synthesis, assembly, and secretion by myeloma cells and by normal mouse lymph node cells were examined. Normal lymphoid cells from hyperimmunized BALB/c mice and cells from most but not all IgG-producing mouse myeloma tumors and cell lines synthesized more light (L) chains than heavy (H) chains. Most of the IgG-producing mouse myeloma tumors and cultured cell lines synthesized and secreted excess L chains. Two tumors, MOPC 21 and MOPC 173, lacked this ability; a number of other tumors produced equimolar amounts of H and L chains. In the case of MPC-11 tumor, excess L chains were synthesized by cloned tumors and cell lines. The amount of excess L chains synthesized and secreted varied greatly, but the average behavior of the tumors and cell lines studied was nearly identical to that of normal IgG-producing lymphoid cells.

2698 MOVEMENT OF LYMPHOCYTE SURFACE ANTIGENS AND RECEPTORS: THE FLUID NATURE OF THE LYMPHOCYTE PLASMA MEMBRANE AND ITS IMMUNOLOGICAL SIGNIFICANCE.

(E.) Raff, M. C. (Zool. Dept., U. Coll. London, England) and S. De Petris. *Fed Proc* 32(1):48-54, 1973.

Fluorescein- or ferritin-conjugated antibodies were used to study the distribution and behavior of immunoglobulins and other antigens on surfaces of mouse lymphocytes. When divalent anti-immunoglobulin antibodies reacted with the diffusely distributed immunoglobulin molecules on the surfaces of lymphocytes, they cross-linked the immunoglobulin molecules and caused them to aggregate into microscopically visible patches. The patches were then actively transported to one pole of the cell where they formed a cap and were pinocytosed. The phenomenon could be reversed by placing the affected cells back in culture. Monovalent binding by Fab fragments of anti-immunoglobulin molecules was ineffective in patch and cap formation, pointing to the importance of the cross-linking. Patch and cap formation also occurred with antigens and other ligands which could bind multivalently to their corresponding receptors on the cells. The triggering of the transformation of lymphocytes into blast cells by the action of antibodies or other agents directed against surface receptors also appeared to require multivalent binding. The author suggests that receptor redistribution may be important to the triggering. He also suggests the implication of antibody-induced pinocytosis in antigenic modulation and of antigen-induced redistribution and pinocytosis in the induction of immunological tolerance. In biochemical terms, the author envisages lymphocyte cell membrane proteins such as immunoglobulin as mobile units consisting of single or grouped protein molecules, floating and diffusing in the plane of a fluid lipid bilayer, a view consistent with the model proposed by Singer and Nicholson.

2699 IMMUNOLOGIC REJECTION OF DIETHYLNITROSAMINE-INDUCED HEPATOMAS IN STRAIN 2 GUINEA PIGS. PARTICIPATION OF BASOPHILIC LEUKOCYTES AND MACROPHAGE AGGREGATES. *(E.)* Dvorak, H. F. (Massachusetts General Hosp., Boston), A. M. Dvorak and W. H. Churchill. *J Exp Med* 137(3):751-775, 1973.

The morphologic events associated with the immunologic rejection by strain 2 guinea pigs of ascites variants of two lines of diethylnitrosamine-induced tumors have been studied by light and electron microscopy. Tumor injection sites in the skin of control animals exhibited clusters of viable, actively mitotic tumor cells along with a modest inflammatory infiltrate composed of lymphocytes, macrophages, neutrophils, and rare basophils. In contrast, similar injections of either tumor line in specifically sensitized guinea pigs elicited typical delayed-type skin reactions associated with tumor cell necrosis and a more extensive inflammatory infiltrate including a selective increase in the number of basophilic leukocytes (12%, line 1, or 23%, line 10, of total inflammatory cells). That basophils may have a role in tumor resistance *in vivo* is suggested by the close anatomic associations observed between basophils and tumor cells, and by the fact that basophils were the only inflammatory cell to demonstrate a relative increase in frequency in the lesions of sensitized as compared with control animals. Moreover, i.p. injection of line 1 tumor in specifically sensitized animals elicited a striking basophilia within 24 hr. Unlike macrophages, basophils did not phagocytose tumor cells but did evidence occasional extrusion of granules and frequently exhibited loss of granule staining density, a change that may be related to release of mediator substances. Electron microscope studies of line 1 tumor rejection in the peritoneal cavities of specifically sensitized guinea pigs demonstrated aggregations of "activated" macrophages, lymphocytes, basophils, and damaged or dead tumor cells. These aggregates, held together by complex interdigitations of macrophage villi, closely resembled those occurring *in vitro* among peritoneal exudate cells whose migration from capillary tubes was inhibited by migration inhibition factor (MIF). Moreover, cells in these aggregates, as well as macrophages inhbited by MIF *in vitro*, lacked a normal coating of cell surface material.

2700 CARCINOEMBRYONIC ANTIGEN (CEA) IN NON DIGESTIVE CANCEROUS AND NORMAL TISSUES. *(E.)* Pusztaszeri, G. (Inst. Biochem., U. Lausanne, Switzerland) and J. -P. Mach. *Immunochem* 10(3):197-204, 1973.

Although in initial series of tests, carcinoembryonic antigen (CEA) could be found by radioimmunoassay in the sera of patients with adenocarcinoma of the large bowel and in no other cases than digestive carcinoma, more recent studies showed relatively high levels of CEA in plasmas of patients with carcinoma of non-digestive organs as well as in plasmas of patients with some non-cancerous diseases. CEA, immunologically identical by gel precipitation analysis to CEA derived from colonic carcinoma, was obtained from perchloric acid (PCA) extracts of bronchial and breast tumors. The antigen was found to be 50-75 times less abundant in these tumors than in colonic carcinoma. It could also be detected in one normal breast in lactation and at 1000-4000 times lower concentrations in normal lung than in colonic carcinoma. The CEA present in normal tissues was distinct from a glycoprotein of smaller molecular wt which was previously extracted in much larger quantities from normal lung and spleen and shown to have only partial identity with CEA. The demonstration of the presence of CEA in non-digestive carcinoma by gel precipitation suggested that the CEA detected in the plasma of such patients by radioimmunoassay was also identical to colonic carcinoma CEA. This hypothesis was supported by a comparative study of plasma CEA from bronchial and colonic carcinoma, showing that CEA from both sources had the same elution pattern on Sephadex G-200 and gave parallel inhibition curves in the radioimmunoassay. All positive CEA radioimmunoassays, however, may not indicate the presence of an antigen identical to colonic carcinoma CEA, since serum glycoproteins containing no CEA were found to interfere in the radioimmunoassay; thus, there could arise false positive radioimmunoassays due to variations of the levels of such glycoproteins.

2701 ULTRASTRUCTURAL AND TISSUE CULTURE STUDIES OF A THYMIC LYMPHOMA INDUCED BY PERSISTENT ANTIGENIC STIMULATION AND IMMUNOSUPPRESSION. *(E.)* Krueger, G. R. F. (Natl. Cancer Inst., Bethesda, Md.), G. T. O'Conor, E. Sussman and T. S. Tralka. *Z Krebsforsch* 79(4):272-276, 1973.

Ultrastructural and immunologic studies were done on a malignant T-cell lymphoma of a BALB/c mouse grown in tissue culture. The original tumor was induced by coincident persistent antigenic stimulation and chronic immunosuppression. The lymphoma cells grew in tissue culture in a biphasic pattern: lymphoid cells proliferated in suspension and histiocytic cells attached to the bottom of the culture flask. While the lymphoid cells retained their T-cell specificities, the differentiating histiocytic cells showed diminishing Theta-antigenic sites and finally lost such specificities entirely. It is suggested that the Theta-negative histiocytes may be "end cells" with a limited proliferation capacity, while lymphoid T-cells are proliferating stem cells with a certain differentiating potential. Differentiation towards histiocytes apparently occurs simultaneously with the loss of T-cell characteristics.

2702 EFFECT OF ACTIVE IMMUNIZATION WITH IRRADIATED TUMOUR CELLS OF SPECIFIC SERUM INHIBITORS OF CELL-MEDIATED IMMUNITY IN PATIENTS WITH DISSEMINATED CANCER. *(E.)* Currie, G. A. (Chester Beatty Res. Inst., Surrey, England). *Br J Cancer* 28(1):25-35, 1973.

The sera from patients with advanced cancer were tested for their specific inhibitory effects on the cytotoxicity of autologous lymphocytes on tumor cells in a microculture assay. By adding a standard volume of the sera to suspensions of well-washed lymphocytes the inhibitory effect was quantitated by comparison with the effect of normal allogeneic serum. Significant levels of inhibitory activity were detected in seven patients (one massive primary melanoma, four with disseminated melanoma, one with metastatic hypernephroma and one with a recurrent leiomyosarcoma). The patient with a massive primary melanoma was treated by extensive surgical excision. This procedure was associated with the rapid and complete disappearance of the serum inhibitory effect. In the other cases surgical intervention was minimal and the serum inhibitor was unaffected. All six of these patients were then immunized with irradiated autologous tumor cells and the serum inhibitory activity assayed. In five cases the serum inhibitor rapidly became undetectable after a single immunization. The one patient who failed to respond in this manner had very extensive disease and died within two wk of the study. Repeated monthly immunization in the case of recurrent leiomyosarcoma was associated with the maintenance of the serum inhibitory activity at very low levels and with good clinical progress. The response to a single immunization is transient, the inhibitor becoming detectable again at 14-21 days. The possible role of circulating antigen in this serum inhibitory activity is discussed, as is the potential value of assaying the sera of cancer patients for serum inhibitory activity, as a means of monitoring the effects of treatment.

2703 DIFFERENCES IN THE UREA-EXTRACTED PROTEINS OF MOUSE EPIDERMIS AND SQUAMOUS CELL CARCINOMATA DETERMINED BY FLUORESCENCE MICROSCOPY. *(E.)* Carruthers, C. (Roswell Park Memorial Inst., Buffalo, N.Y.). *Br J Cancer* 28(1):36-45, 1973.

Fluorescence microscopy was used to demonstrate differences in the urea-extractable antigens of mouse epidermis and squamous cell carcinoma. When serum- and normal tissue sediment-absorbed antisera prepared against mouse epidermal urea-extracted proteins were further absorbed with carcinoma urea antigens, antisera specific for epidermis resulted. When antisera raised against the urea-extractable proteins of mouse squamous cell carcinomata were serum- and normal tissue sediment-absorbed and then further absorbed with epidermal urea antigens, antisera were prepared which stained papilloma and carcinoma, but not epidermis, and thus these antisera were not specific for carcinoma. Antisera prepared against the urea-extractable proteins of human epidermis reacted in immunodiffusion in agar with the epidermal urea proteins, but not with human squamous cell carcinoma urea proteins. Also antisera prepared against the carcinoma urea-extractable proteins reacted with these proteins in agar, but no reaction occurred with the epidermal urea-extractable proteins. These studies do not indicate the nature of the protein changes between epidermis and papilloma or carcinoma. There may be different proteins in epidermis than in papilloma or carcinoma even though the urea proteins of one of the related tissues can remove antibodies raised against any of the other two related tissues.

2704 EVIDENCE FOR FETAL ANTIGEN IN HUMAN SARCOMA. *(E.)* Mukherji, B. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.) and Y. Hirshaut. *Science* 181(4098):440-442, 1973.

A diffuse cytoplasmic antigen (S_2), initially thought to be specific to human sarcomas, was detected by the immunofluorescent technique in patients with other tumors as well. Antibody to the antigen was present in sera from 46 of 64 sarcoma patients and nearly a third of their family members. Sera from patients with carcinoma of breast, lung, ovary, and gastrointestinal tract, and with melanoma, also frequently demonstrated anti-S_2 reactivity. Among cell lines tested, S_2 antigen was present in sarcoma and benign giant cell tumor lines, embryonic tissue, and normal skin, both of tumor-bearing and normal individuals. The presence of S_2 in embryonic tissue suggests a fetal antigen, while the presence of antibody to S_2 in tumor patients and their relatives implies a propensity in these individuals for cellular dedifferentiation which may be a prerequisite for malignant transformation.

2705 ANTIBODIES AND SOLUBLE TUMOUR-SPECIFIC ANTIGENS IN BLOOD AND LYMPH OF RATS WITH CHEMICALLY INDUCED SARCOMATA. *(E.)* Thomson, D. M. P. (McGill U. Med. Clin., Montreal, Canada), S. Eccles and P. Alexander. *Br J Cancer* 28(1):6-15, 1973.

In confirmation of other studies it has been shown that antibody directed against the tumor specific transplantation-type antigens (TSTAs) cannot be detected in rats with a tumor growing intramuscularly but appears within a few days after excision of the tumor. Circulating antibody is found in the serum of rats with lymph node metastasis following excision of the primary. Absorption by the intramuscular tumor of circulating antibody does not account for the absence of antibody since the antibody levels in thoracic duct lymph in rats with tumors in the leg are also very low and rise rapidly after tumor excision. Antibody is released by the draining nodes directly into the lymph and must pass through the thoracic duct before entering the blood. Under these conditions low levels of antibody activity in lymph cannot be ascribed to absorption by the tumor. It is postulated that TSTA is released from the tumor into the lymph. Following injection of tumor cells into immunized rats the level of antibody falls and this is attributed to release of TSTA from the injected cells. It is possible that the biological behavior of antigenic tumors may be determined in part by the relative ease with which release of soluble low molecular TSTAs occur. It is suggested that a rapid rate of spontaneous release of soluble TSTA may facilitate escape from host control and therefore be associated with a high degree of malignancy. Tumor transplantation is associated with a persisting release of soluble antigen into the circulation. This antigenic burden is continuously present and continually renewed as long as the tumor mass exists and has been shown to affect the humoral response and might be expected to interfere also with specific cell-mediated immunity.

2706 LOCALIZATION OF RAT LIVER α-FOETOPROTEIN BY CELL AFFINITY LABELLING WITH TRITIATED OESTROGENS.

(E.) Uriel, J. (Inst. Sci. Res. Cancer, Villejuif, France), C. Aussel, D. Bouillon, B. de Nechaud and F. Loisillier. *Nature* [*New Biol*] 244(136):190-192, 1973.

The intracellular localization of alpha-fetoprotein (α-FP) in liver of Wistar rat fetuses, neonates, and adult animals with primary hepatomas induced by 3'-methyl-4-dimethylaminoazobenzene or dimethylnitrosamine was studied autoradiographically by affinity labeling with tritiated estrogens. In fetal and neonatal liver, the distribution of radioactivity was confined to hepatocyte-like cells overlying the nucleus and cytoplasmic membrane. In the liver of a 10-day-old rat, radiolabeled cells were also observed within a thin elongated area of bile duct proliferation, between the border of a portal vein and the hepatic parenchyma. Radiolabeled cells were found in tumors only when animals had detectable serum α-FP levels (greater than 10 μg/ml). The cells were most frequently situated at the border of sinusoid capillaries but were also observed within areas of fibrous-connective tissue or scattered among duct-like structures in zones of cholangiofibrosis. The cell-type producing α-FP appeared to be a round "transitional" liver cell distinct from normal or neoplastic hepatocytes. This type was present in hepatocellular carcinomas as well as in fetal and neonatal liver.

2707 CARCINOEMBRYONIC ANTIGEN-ANTIBODY COMPLEXES IN A PATIENT WITH COLONIC CARCINOMA AND NEPHROTIC SYNDROME.

(E.) Costanza, M. E. (New England Med. Ctr. Hosp., Boston, Mass.), V. Pinn, R. S. Schwartz and L. Nathanson. *N Eng J Med* 289(10):520-522, 1973.

The association of the nephrotic syndrome and neoplasms is uncommon. In one instance, deposits containing IgG and B_1C-globulin were detected on the renal glomeruli of a patient with Hodgkin's disease and the nephrotic syndrome. In another instance, IgG was found in the renal glomeruli of a patient with squamous-cell carcinoma of the lung and nephrotic syndrome; further, immunoglobulins eluted from the glomeruli of this patient reacted specifically with the patient's tumor cells. In the present case of colonic carcinoma with the nephrotic syndrome, immunofluorescent studies of renal biopsy showed the presence of immunoglobulins, complement, and carcinoembryonic antigen (CEA). The finding suggests that development of nephrosis in a patient with neoplasia may be a complication of the patient's immune response to the neoplasm, leading to formation of immune complexes of antitumor antibody and tumor antigen which deposit in the glomeruli. The CEA used in the study was prepared from a liver metastasis of the patient and was identified by a line of identity with authentic CEA in a gel diffusion test.

2708 IMMUNOGLOBULIN RECEPTORS ON B LYMPHOCYTES: SHIFTS IN IMMUNOGLOBULIN CLASS DURING IMMUNE RESPONSES.

(E.) Pierce, C. W. (Harvard Med. Sch., Boston, Mass.), R. Asofsky and S. M. Solliday. *Fed Proc* 32(1):41-43, 1973.

The relationship between the immunoglobulin class of antigen receptors on cell membranes of precursors of antibody-forming cells and the antibody ultimately secreted by progeny of these precursor cells after antigenic stimulation has been investigated. The analytical system used was the suppressive effects of class-specific antibody to mouse immunoglobulins on primary and secondary plaque-forming cell responses to heterologous erythrocytes in cultures of mouse spleen cells. Virgin precursor cells appear to have receptors of the IgM class regardless of the class of antibody eventually secreted. Single exposure to antigen induces a transient shift in receptor class from IgM to IgG. Prolonged immunization with large antigen doses induces a more permanent shift in receptor class from IgM to IgG. Low doses of antigen, which produce no shift in receptor class after a single injection can induce a transient shift after chronic administration. An idealized scheme for behavior of B lymphocytes in immune responses is derived. The virgin precursor cells with IgM receptors, after antigen stimulation, and under the influence of thymus-derived cell activity and possible genetic precommitment to immunoglobulin class, develop along either IgM or IgG response pathways. Precursor cells committed into the IgG pathway develop a second antigen receptor of the immunoglobulin class of the antibody to be secreted.

2709 IMMUNOFLUORESCENCE MEASUREMENT OF C1 INACTIVATOR (ALPHA 2 NEURAMINOGLYCOPROTEIN) ACTIVITY OF THE SURFACE OF HUMAN CARCINOMA CELLS.

(E.) Osther, K. (Dept. Path., Kommunehospitalet, Copenhagen, Denmark) and R. Linnemann. *Acta Pathol Microbiol Scand (B)* 81(3):365-372, 1973.

The presence of C1 inactivator on carcinoma cells was demonstrated by incubating the cells with fluorescein isothiocyanate conjugated rabbit anti human C1 inactivator followed by measurement of cell fluorescence on a Leitz MPV I cytophotometer. The control material, consisting of cells from pleural and ascites fluid of patients suffering from nonmalignant diseases, showed a statistically significant difference from the malignant cells. The possibility that the C1 inactivator functions as a defense for the carcinoma cells against the humoral immune system is discussed.

2710 NASOPHARYNGEAL CARCINOMA (NPC). VI. PRESENCE OF AN EBV NUCLEAR ANTIGEN IN FRESH TUMOUR BIOPSIES. PRELIMINARY RESULTS. *(E.)* de-Thé, G. (Int. Agency Cancer Res., Lyon, France), D. V. Ablashi, A. Liabeuf and N. Mourali. *Biomedicine* 19(8):349-352, 1973.

While the etiological role of Epstein-Barr herpesvirus (EBV) in the genesis of Burkitt's lymphoma is generally accepted, it is more questionable in the case of nasopharyngeal carcinoma (NPC), since this tumor is a carcinoma infiltrated by lymphocytes and the precise role of these lymphocytes in the development of the tumor is unknown. To investigate the presence of EBV specific antigen in the NPC tumors as well as its localization in lymphocytes and/or epithelial cells, the anti-complement immunofluorescence test (ACIF) of Reedman and Klein was applied to touch smears and cell suspensions from five fresh NPC biopsies. The ACIF test, which is for EBV specific nuclear antigen (NA), was positive for 15-56% of the cells. The positive cells belonged mostly to the lymphoid series; however, epithelial-like cells were occasionally positive. Additional tests for identification of the NA-positive cells in the NPC biopsies are in progress.

2711 IDENTIFICATION OF A NOVEL CELL TYPE IN PERIPHERAL LYMPHOID ORGANS OF MICE. I. MORPHOLOGY, QUANTITATION, TISSUE DISTRIBUTION. *(E.)* Steinman, R. M. (Rockefeller U., New York, N.Y.) and Z. A. Cohn. *J Exp Med* 137(5):1142-1162, 1973.

The morphology, quantitation, and tissue distribution of a large stellate cell identified *in vitro* is described. This novel cell was found in adherent cell populations prepared from peripheral lymphoid organs (spleen, lymph node, Peyer's patch) of outbred NCS mice and inbred DBA/aJ, C3H, BALB/cJ, C57BL, and C57BL X DBA/2J mice. Although present in small numbers (0.1-1.6% of the total nucleated cells), the cells have distinct morphological features. The nucleus is large, refractile, contorted in shape, and contains small nucleoli (usually 2). The cytoplasm is abundant, is arranged in processes of varying length and width, and contains many large spherical mitochondria. In the living state, the cells undergo characteristic movements and do not appear to engage in active endocytosis. The term dendritic cell is suggested for this novel cell type.

2712 HISTOCOMPATIBILITY-LINKED GENETIC CONTROL OF DISEASE SUSCEPTIBILITY. MURINE LYMPHOCYTIC CHORIOMENINGITIS VIRUS INFECTION. *(E.)* Oldstone, M. B. A. (Scripps Clin. Res. Fdn., La Jolla, Calif.), F. J. Dixon, G. F. Mitchell and H. O. McDevitt. *J Exp Med* 137(5):1201-1212, 1973.

The susceptibility of murine strains to acute lymphocytic choriomeningitis (LCM) virus infection was genetically determined and the question of whether the tendency for increased susceptibility was linked to the major histocompatibility (H-2) locus was studied. Acute necrotizing inflammatory disease after intracerebral injection of LCM virus is largely dependent on the host immune response to the virus and is controlled partly by a dominant gene which is closely linked to the H-2 locus. The F_1 hybrid (H-$2^{q/k}$) from mating a susceptible SWR/J mouse (H-$2^{q/q}$) to a resistant C3H/HeJ mouse (H-$2^{k/k}$) is susceptible to LCM virus disease. When such hybrids (H-$2^{q/k}$) are backcrossed to susceptible parents (H2-$^{q/q}$), all F_2 offspring (H-$2^{q/q}$, H-$2^{q/k}$) are highly susceptible. In contrast, hybrid (H-$2^{q/k}$) backcross to resistant parents (H-$2^{k/k}$) results in half of the F_2 offspring being susceptible (H-$2^{q/k}$) while the other half are resistant (H-$2^{k/k}$). Similarly, in congenic H-$2^{q/q}$ and H-$2^{k/k}$ mice, H-$2^{q/q}$ mice are relatively susceptible to acute LCM disease, whereas H-$2^{k/k}$ are resistant.

2713 *IN VITRO* STUDIES ON MECHANISM OF THE "ECLIPSE" OF CELL-MEDIATED IMMUNITY IN MICE BEARING ADVANCED TUMORS. *(E.)* Youn, J. K. (Gustave-Roussy Inst., Villejuif, France), D. Le Francois and G. Barski. *J Natl Cancer Inst* 50(4):921-926, 1973.

When mouse tumor target cells (BALB/c T5 tumor) were exposed to peritoneal cells (PC) sensitized against the tumor target, colony inhibition (CI) activity of PC was observed to disappear in PC from mice with tumors $\geq$ 10 mm in size. This "eclipse" of cell-mediated immunity in tumor-bearing mice was immunologically specific; it was not seen in mice with an antigenically unrelated tumor. To investigate the "eclipse" phenomenon, PC from T5 tumor-bearing mice were incubated with serum from T5-bearing mice before exposure of PC to T5 target cells. These preincubated PC were less inhibitory to target cell colony formation than were T5-bearing mouse PC preincubated with serum from normal controls. When this same serum was preincubated with T5 tumor target cells and the target was exposed to the same PC as in the previous experiment, PC exerted a significant blocking effect on the colony formation of the target cells.

2714 CELL INTERACTIONS BETWEEN HISTOINCOMPATIBLE T AND B LYMPHOCYTES. I. ALLOGENEIC EFFECT BY IRRADIATED HOST T CELLS ON ADOPTIVELY TRANSFERRED HISTOINCOMPATIBLE B LYMPHOCYTES. *(E.)* Hamaoka, T. (Harvard Med. Sch., Boston, Mass.), D. P. Osborne, Jr. and D. H. Katz. *J Exp Med* 137(3):1393-1404, 1973.

An allogeneic effect on 2,4 dinitrophenyl (DNP)-keyhole limpet hemocyanin-primed lymphocytes was obtained after adoptive transfer of such cells from BALB/c mice into a heavily irradiated (600 R) allogeneic host. The existence of an allogeneic effect was supported by the finding that under such conditions DNP-KLH-primed cells developed significantly enhanced secondary anti-DNP antibody responses to DNP-bovine gamma globulin (BGG), whether or not the irradiated allogeneic host possessed BGG-specific helper cells. This allogeneic effect resulted from the capacity of residual, apparently radioresistant, T cells in the irradiated host to exert an active effect on the transferred histoincompatable B lymphocytes. This conclusion was derived from two experiments. In the first, an allogeneic effect was shown to occur on DNP-primed F_1 spleen cells that had been adoptively transferred to irradiated parental recipients; the second experiment demonstrated the development of an allogeneic effect on anti-θ-treated, DNP-specific donor cells transferred to irradiated allogeneic hosts.

2715 ESCAPE FROM ISOANTISERUM INHIBITION OF LYMPHOCYTE-MEDIATED CYTOXICITY. *(E.)*

Faanes, R. B. (Variety Club Heart Hosp., Minneapolis, Minn.), Y. S. Choi and R. A. Good. *J Exp Med* 137(1): 171-182, 1973.

Isoantiserum (IS) inhibition of lymphocyte-mediated cytotoxicity (LMC) was studied using an *in vitro* ^{51}Cr release assay system. In the early phase of incubation of mastocytoma cells from DBA/2J mice, IS prepared in C57/B16 mice inhibited LMC, but as incubation continued LMC irreversibly overcame IS inhibition (the "escape" phenomenon). The course of escape was not altered by addition of fresh antiserum. It is theorized that antibody directed toward an actively metabolizing target cell induces a change in the cell membrane that alters further interaction with the antibody. Sensitivity to lymphocyte cytotoxicity is maintained.

2716 SURFACE IMMUNOGLOBULINS ON LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKAEMIA AND LYMPHOSARCOMA. *(E.)*

Wilson, J. D. (Walter and Eliza Hall Inst., Melbourne, Australia) and A. D. F. Hurdle. *Br J Haematol* 24(5):563-569, 1973.

In an earlier work, human lymphocytes treated with labelled anti immunoglobulins showed two populations, one with high and the other with low density of surface immunoglobulin, referred to as B and T lymphocytes, resp. More recently, leukemic cells from patients with chronic lymphocytic leukemia (CLL) were found to be of the B cell type. The present communication extends this work to patients with either CLL or lymphosarcoma (LS), using a sandwich radioimmunolabeling technique. CLL lymphocytes were found to comprise a single population with labeling characteristics of B lymphocytes in eight patients and of T lymphocytes in two patients. LS lymphocytes fell into the T cell type in two patients and of B cell type in one patient. Unlike CLL lymphocytes, however, LS lymphocytes revealed 2 or 3 populations, probably reflecting the fact that the circulating lymphosarcoma cells had not totally replaced the normal peripheral lymphocytes. Clinical features of the CLL patients suggested that T cell leukemia may be more benign than B cell leukemia, but the series was too short for a definite conclusion. There were no essential clinical differences among the lymphosarcoma patients. The value of reclassifying CLL and other neoplasms of lymphoid tissue by their density of surface immunoglobulin remains to be established. The present technique did, however, add a diagnostic method for use in cases of lymphocytosis, since the presence of a lymphocyte population homogeneous in surface immunoglobulin labeling would be strong evidence that the lymphocytes were neoplastic.

2717 *IN VITRO* ASSESSMENT OF CELLULAR IMMUNITY TO RAUSCHER LEUKEMIA VIRUS. *(E.)*

Mortensen, R. F. (Dept. Microbiol., Pennsylvania State U., University Park), W. S. Ceglowski and H. Friedman. *J Immunol* 111(3):657-660, 1973.

Cell-mediated immunity to Rauscher leukemia virus (RLV) was studied using the macrophage migration inhibition assay. Cell-mediated immunity to purified RLV antigen was detected in splenic lymphocytes of the leukemia resistant C57BL/6 mouse strain inoculated with infectious RLV. In contrast, no cellular immunity to RLV could be detected in splenic lymphocytes of the leukemia susceptible BALB/c mouse strain upon infection with RLV. Both strains of mice exhibited an active cell-mediated immune response following inoculation with heat-inactivated RLV. Appropriate controls ruled out a contribution of either antibody or antibody-antigen complexes as mediators of the observed inhibition of macrophage migration. These data support the view that an inhibition of cell-mediated immune function may be a prerequisite to the induction of viral leukemia.

2718 INCREASE IN INCIDENCE OF RETICULUM CELL SARCOMA IN F_1 HYBRID MICE THYMECTOMIZED AND BEARING THYMUS GRAFTS FROM PARENTAL STRAIN. *(E.)*

Nakakuki, K. (Sch. Med., Mie U., Japan) and Y. Nishizuka. *Gann* 64(2):161-166, 1973.

Previous work has shown that thymectomy prevents the development of lymphoma in mice spontaneously susceptible to the disease, that grafting of the thymus in thymectomized animals restores their susceptibility, and that only grafts from high-leukemia strain mice exert the leukemogenic influence. In the present experiments, (AKRxSL)F_1 hybrid mice were thymectomized at 35 days of age and grafted subcutaneously with whole thymus tissue from AKR, SL, or F_1 hybrid mice of the same age. Since the lymphomas of the parental AKR mice are almost all thymic while those of the parental SL strain are exclusively non-thymic, it was of interest to investigate whether this difference might affect the lymphoma-restoring ability of the grafts. The grafts degenerated at first but became reconstituted by 20 days. The animals were examined biweekly for 2 yr and all were autopsied. The incidence of lymphoid tumors was reduced from 81.9 % to 47.4 % in the F_1 hybrids following thymectomy. Grafting of AKR thymus restored the incidence to 82.6 %, but grafting of SL or F_1 thymus effected no restoration. The failure of SL thymus grafts relative to AKR thymus grafts was attributed to absence of leukemia virus in the former. The failure of F_1 thymus grafts relative to both F_1 thymus and AKR thymus were expected to contain leukemia virus. The authors proposed, on the basis of available evidence, that continuous host lymphoid tissue damage by grafted AKR thymus cells could have contributed to the leukemogenic effect; because of genetic identity, the grafted F_1 hybrid mice might have resulted from the interaction of two different mechanisms, viral and immunological.

2719 SPONTANEOUS AGE-DEPENDENT PRODUCTION OF ANTIBODIES AGAINST THE VIRION IN CBA/BLn MICE INFECTED NEONATALLY WITH THE MAMMARY TUMOUR VIRUS (MTV). *(E.)* Zotter, S. (Med. Acad. "Carl Gustav Carus," Dresden, E. Germany), M. Müller and H. Grossmann. *Exp Pathol (Jena)* 8(2):115-121, 1973.

Spontaneous occurrence of antibodies to the mammary tumor virus (MTV) B-particle was studied in naturally MTV-infected CBA/Bln mice of different ages. The micro-Ouchterlony immunodiffusion assay and the indirect immunofluorescence test were used. In the immunofluorescence assay, spontaneous anti-MTV antibodies were revealed in all tested sera from adult MTV-bearing animals, while the micro-Ouchterlony technique gave positive results for only 31 of 108 sera. In the immunodiffusion assay, the number of positive sera in adult females increased with age but was not significantly influenced by the number of litters/animal. Immunofluorescence studies on newborns immediately after birth and on several foster-nursed infant mice showed that the antibody is transmitted *in utero* as well as by milk. Young mice MTV-infected by milk seem to start production of antibodies at age 3 or 4 wk and for that reason induction of antibody formation may take place during virus penetration into the lymphatic system and/or in the stage of viremia. It is concluded that the antibody response after MTV infection is the most sensitive criterion of MTV infection in CBA mice.

2720 ENZYMATIC AND IMMUNOLOGICAL STUDIES ON PYRUVATE CARBOXYLASE IN LIVERS AND LIVER TUMORS. *(E.)* Chang, L. O. (U. Virginia Sch. Med., Charlottesville) and H. P. Morris. *Cancer Res* 33(9): 2034-2041, 1973.

It was found that one Reuber hepatoma and seven Morris hepatomas with different growth rates and various degrees of differentiation contained pyruvate carboxylase (PC) activity ranging from 3 to 113% of control values. The levels of PC activity in the regenerating liver, spontaneous mouse hepatoma, and host livers of tumor-bearing rats were estimated in the range of normal rat liver. In the group of tumors studied, two hepatomas of intermediate growth rate and four highly and well-differentiated, slow-growing tumors had negligible or little PC activity. However, one hepatoma, 9618A, had a normal level of PC activity. Antibodies prepared in rabbits against the purified mitochondrial enzyme showed identical titration curves against the nuclear and cytosol PC of liver. A single continuous precipitation line was obtained on Ouchterlony double-diffusion precipitation analysis when purified mitochondrial enzyme, crude mitochondrial preparations of rat tissues and hepatomas, were compared. It was also found that the immunological, enzymatic, and electrophoretic properties of the tumor PC were the same as those of normal adult and newborn rat liver. The amount of enzyme protein was quantitatively reduced in all but one of the liver tumors studied. It was also observed that the number of mitochondria in tumors was reduced in terms of mitochondrial protein content per g fresh weight of tissue. The decrease in the mitochondrial PC in the hepatomas may well partially reflect the decrease in the number of mitochondria in the tumors.

2721 SYNTHESIS OF IMMUNOGLOBULINS BY MEMBRANE-BOUND POLYSOMES AND FREE POLYSOMES FROM PLASMACYTOMA CELLS. *(E.)* Pryme, I. F. (U. Bergen, Norway), O. Garatun-Tjeldsto, P. J. Birckbichler, J. K. Weltman and R. M. Dowben. *Eur J Biochem* 33(2):374-378, 1973.

Free polysomes and membrane-bound polysomes were prepared from tissue-cultured BALB/c mouse plasmacytoma cells (MPC-11 and MOPC-70e) and interacted with ^{125}I-labelled F(ab) fragments of rabbit antibody to the mouse myeloma proteins. Peaks of reactive polysomes were found in the 6—8 oligomer and 16—20 oligomer region of membrane-bound polysomes but not of free polysomes. When used in an amino-acid-incorporating system *in vitro*, MPC-11 membrane-bound polysomes gave rise primarily to light-chain immunoglobulins and MOPC-70e membrane-bound polysomes gave rise exclusively to light-chain immunoglobulins which were identified after reduction and acetylation and separation on dodecylsulfate-gel electrophoresis. Free polysomes did not give rise to significant amounts of heavy chain or light chain immunoglobulin, indicating that the synthesis of the secreted protein, immunoglobulin, is entirely associated with membrane-bound polysomes.

2722 EVIDENCE FOR SPECIFIC TUMOR CYTOTOXIC ANTIBODIES IN SERUM OF CERVICAL CANCER PATIENTS. *(E.)* Weintraub, I. (UCLA Med. Ctr., Los Angeles, Calif.), I. Klisak, L. D. Lagasse and J. E. Byfield. *Am J Obstet Gynecol* 116(7):985-992, 1973.

Antibodies against cell surface antigens of cervical carcinoma were detected using microcytotoxicity and complement fixation tests. The cytotoxic reaction was dependent on rabbit complement. CX1B, a cervical epidermoid carcinoma strain grown in the authors' laboratory was employed exclusively in these tests. Specific destruction of cells is defined as that state when less than 40% of the cells remain viable after a four hr exposure to antibody and complement; cytotoxicity under these circumstances can be related to cervical carcinoma-related antibodies. Nonspecific destruction as high as 20% was routinely observed as test background. The evidence for antibodies specific to cervical carcinoma is based on: (a) the serum of cervical carcinoma patients which produced specific destruction of the malignant CX1B line did not affect a normal cervix strain; (b) none of the control sera elicited a specific reaction; (c) absorption of specific serum of cervical carcinoma patients by membrane fragments of CX1B reversed the positive result; and (d) three out of four specific cytotoxic sera could be correlated with positive complement fixation test. Correlation with clinical data revealed higher titers of tumor related antibodies in Stage I which may be interpreted as evidence that circulating antigen-antibody complexes are not common early in this disease.

2723 ALTERATIONS IN LYMPHOCYTE POPULATIONS IN TUMORIGENESIS. I. LYMPHOCYTE TRAPPING.

(E.) Zatz, M. M. (A. Einstein Coll. Med., Bronx, N. Y.), A. White and A. L. Goldstein. *J Immunol* 111(3):706-711, 1973.

The question of whether the lymphoid system responds to an antigenic, virus-induced tumor was investigated by inoculating CBA/J and BALB/cJ female mice with the oncogenic Moloney sarcoma virus (MSV). This experimental model was then used to quantitate changes in lymphocyte traffic and to correlate the magnitude and kinetics of trapping of ^{51}Cr-labeled lymphocytes in the responding lymphoid tissues with the cycle of MSV tumor growth and regression. Significant numbers of labeled cells were trapped in the draining lymph nodes and spleens of tumor-bearing mice. The kinetics of lymphocyte trapping correlated with the cycle of growth and spontaneous regression which is characteristic of this tumor in healthy mice. Trapping first occurred after injection of the virus and maximal retention of lymphocytes corresponded to the time of tumor regression. It would seem that lymphocyte trapping reflects an alteration in host immunity, which results in arrest of tumor growth and subsequent regression. It is unclear, however, whether lymphocyte trapping is associated with changes in cellular or humoral immunity to the tumor.

2724 IMMUNE CAPACITY AND RESPONSE TO ANTIGENIC TUMORS.

(E.) Risdall, R. J. (Dept. Surg. Microbiol., U. Minnesota, Minneapolis), J. C. Aust and C. F. McKhann. *Cancer Res* 33(9):2078-2085, 1973.

Adult C3H mice responded to the grafting of a syngeneic sarcoma with a vigorous hyperplasia in the regional lymph nodes and spleen. These changes, otherwise progressive until the death of the animals, were reversed when the tumor was removed surgically. The hyperplastic response was not seen in animals previously immunized to the same tumor nor was it seen in tumor-bearing animals displaying concomitant immunity in response to a challenge of the same tumor. In fact, an inverse relationship was observed between the capacity of the tumor to induce transplantation resistance *versus* the growth of the tumor and the magnitude of the lymphadenopathy. These reactions were all found to be tumor specific. Histologically, the hyperplasia was seen to be due to a plasmacytosis and histiocytosis of the involved lymphatic tissue. An evaluation of the immune capacity of these animals to nonrelated antigens was undertaken. The response of tumor-bearing animals to sheep red blood cells, as measured by the Jerne plaque assay, and their capacity to mount a graft-*versus*-host reaction, as measured by the Simonsen assay, were both comparable to normal controls. Thus significant immunosuppression by the tumor was not seen. It is suggested that a specific defect exists in the regional nodes, possibly due to the "weak" nature of the tumor antigens, and that the tumor becomes established irreversibly before an effective immune response develops. The massive plasmacytosis and histiocytosis are probably in response to an established, progressively growing tumor releasing large amounts of antigenic material and suggest that antibody, possibly "blocking" antibody, is being produced.

2725 REJECTION OF POLYOMA VIRUS-INDUCED NEOPLASMS IN MICE INOCULATED WITH COMPLEMENT AND ANTISERUM.

(E.) Negroni, G. (Imperial Cancer Res. Fund Lab., London, England) and E. Hunter. *J Natl Cancer Inst* 51(1):265-268, 1973.

Antibodies against cell antigens had no effect on the growth of polyoma virus-induced neoplasms in C3H/Bi mice. However, these antibodies, when given with guinea pig serum (complement), induced tumor rejection. This suggests that the humoral immunity was defective because of the poor complementary activity of the mouse serum. The rejection was not absolute, and some tumors developed, even after treatment with antiserum and complement. Therefore, tumors might have developed by an immune selection similar to that previously observed *in vitro*. The tumor cells, however, were sensitive to immune lymphocytes; an explanation of tumor growth, therefore, must also assume an insufficient cellular immune response. The rejection by complement and antibodies did not occur when the antiserum was contaminated with antibodies to calf serum proteins. It is possible that antibodies to calf serum reacted with calf serum proteins at the cell surface and thus prevented the specific anticell antibody from reacting with the cell antigen.

2726 IMMUNOSUPPRESSION AND MALIGNANT LYMPHOMAS IN GRAFT-*VERSUS*-HOST REACTIONS.

(E.) Solnik, C. (Tufts U. Sch. Med., Boston, Mass.), H. Gleichmann, M. Kavanah and R. S. Schwartz. *Cancer Res* 33(9):2068-2077, 1973.

The immune responses of mice undergoing the graft-*versus*-host reaction were tested with a battery of four antigens. Three donor-host combinations were selected on the basis of their susceptibility to the development of malignant lymphomas. The results were highly variable. In general, antigens eliciting responses independent of thymus-derived (T) cells (lipopolysaccharide) or bone marrow-derived cells (skin allograft rejection) were normal, whereas antigens eliciting cooperative responses between T- and bone marrow-derived-cells (sheep red blood cells and dinitrophenyl-keyhole limpet hemocyanin) were depressed. Augmentation of primary immune responses by the graft-*versus*-host reaction was found in four different experiments. Evidence for an environmental immunosuppressant could not be found. Deliberate immunosuppression of otherwise normal mice with anti-mouse thymocyte serum failed to cause the appearance of tumors. Those mice with the lowest incidence of lymphomas developed severe depression of certain of their immune responses. Under the experimental conditions studied, it appears that the graft-*versus*-host reaction can: (a) block cooperation between T- and bone marrow-derived cells; (b) augment certain immune responses, perhaps by means of stimulants released from T-cells; and (c) provoke the development of lymphomas in a setting devoid of significant immunosuppression.

2727 ANTIBODIES ASSOCIATED WITH EPSTEIN-BARR VIRUS IN BURKITT'S LYMPHOMA DURING INJECTIONS OF BCG OR IRRADIATED AUTOCHTHONOUS TUMOR CELLS. *(E.)* Gunven, P. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), G. Klein, J. Onyango, G. Henle, W. Henle, P. Clifford, S. Singh, A. Demissie and A. Svedmyr. *J Natl Cancer Inst* 51(1):45-56, 1973.

Antibodies to Epstein-Barr virus-associated antigens and herpes simplex, varicella, cytomegalovirus, measles, and respiratory syncytial virus-associated antigens were titrated in five patients with Burkitt's lymphoma. Whereas neither cytostatic drug therapy and tumor regression nor inoculations of irradiated autochthonous tumor cells regularly affected the titers, BCG inoculations increased the antibody titers to Epstein-Barr virus-associated cell membrane antigens by 1 to 2 $\log_2$ units. After repeated BCG inoculations, the antimembrane-antigen titers returned to pretreatment levels in four of five patients studied.

2728 ANTIGEN RECEPTORS ON LYMPHOCYTES. *(E.)* Unanue, E. R. (Harvard Med. Sch., Boston, Mass.), H. D. Engers and M. J. Karnovsky. *Fed Proc* 32(1):44-47, 1973.

A brief analysis on the nature of antigen receptors on B and T lymphocytes is reported. Most B lymphocytes have a mean of about 10^5 immunoglobulin molecules distributed at random on their surface membranes. These immunoglobulin molecules can be easily detected by immunofluorescence or autoradiography by means of appropriate antibodies. These immunoglobulin receptors appear to bind antigen, a process that is required for the immunological expression of the cell. The nature of this bond has not been determined. The immunoglobulin molecules can be easily displaced on interaction with a ligand at 37 C, and hence are not firmly attached. The binding is a product of a particular B lymphocyte and does not represent nonspecific cytophylic immunoglobulins. Whether or not T lymphocytes have immunoglobulin molecules remains controversial. T lymphocytes cannot discriminate among antigens as specifically as B lymphocytes. In most thymic or T cells of spleens immunoglobulin molecules have not been found. The thymus contains a few cells with surface immunoglobulin, many of which do not react with the antibodies to the alloantigen θ. A few thymic cells have been found to bind antigen.

2729 ISOANTIGENS A, B AND H IN BENIGN AND MALIGNANT LESIONS OF BREAST. *(E.)* Gupta, R. K. (St. Catherines Hosp., Ontario, Canada) and R. Schuster. *Am J Pathol* 72(2):253-258, 1973.

Eighty-five specimens of breast tissue were grouped and investigated by the technic of mixed cell agglutination reaction (MCAR) for studying isoantigens A, B and H (O). MCAR was found to be strongly positive at the epithelium of acini in all the 25 subjects with benign lesions (10 fibroadenomas, 10 cystic mastitis and 5 gynecomastias). On the other hand, all the subjects with diagnosis of primary carcinomas in breast (45 subjects) and their metastases (15 subjects) were found to be uniformly negative for any agglutination reaction, a finding identical with those of other workers. In view of these findings, it seemed that the isoantigens in primary and metastatic malignancies of breast are always lost, while in the benign lesions isoantigens are always present. Although the exact mechanism is not understood, it seems that, in malignancies with a glandular differentiation, some differences exist when these are compared to malignancies with a squamous differentiation.

2730 ANTIBODIES IN NORMAL AND LEUKEMIC CATS TO FELINE ONCORNAVIRUS-ASSOCIATED CELL MEMBRANE ANTIGENS. *(E.)* Jarrett, W. (Leukemia Res. Unit, U. Glasgow, Scotland), M. Essex, L. Mackey, O. Jarrett and H. Laird. *J Natl Cancer Inst* 51(1):261-263, 1973.

Serologic findings in 22 cats with naturally occurring, well-characterized hematopoietic neoplasms and in 28 normal adult cats were described. The test was an indirect flourescent antibody technique for feline oncornavirus-associated cell membrane antigen of A, B, and C subgroup specificities. Thirty-nine percent of normals and forty-five percent of cats with neoplasms had antibodies. The study did not show any particular relationship between antibody level and type, cytology or stage of hematopoietic neoplasm, or whether virus was demonstrable. Results indicate a group of animals exists with tumors which, while not having demonstrable virus at death, have had contact with virus, since they had antibodies.

2731 ANALYSIS OF SURFACE ANTIGENS ON SIMIAN VIRUS 40-TRANSFORMED CELLS. II. EXPOSURE OF SIMIAN VIRUS 40-INDUCED ANTIGENS ON TRANSFORMED RABBIT KIDNEY AND INBRED HAMSTER KIDNEY CELLS BY PHOSPHOLIPASE C. *(E.)* Collins, J. J. (Harvard Med. Sch., Boston, Mass.) and P. H. Black. *J Natl Cancer Inst* 51(1):115-134, 1973.

The surface antigens on simian virus 40 (SV40)-transformed rabbit and inbred hamster (LSH) cells were analyzed. Immune sera were raised in hamsters by tumor induction and excision and in rabbits by a long-term immunization schedule. Specific immune sera did not react with the appropriate target cells unless the cells were pretreated with phospholipase C, whereas pretreatment with trypsin, sialidase, hyaluronidase, or phospholipases A and D did not allow the detection of the SV40-induced antigens. The antigens detected did not cross-react with other SV40-transformed cell lines, including other transformed LSH clones derived from the same population of inbred hamster kidney cells. The antigens could be detected only on the corresponding immunizing cells and thus represented virus-induced, host-coded antigens unique to individual transformed lines, and in the cloned LSH cells, unique to individual transformation events. Antigen expression was influenced by the physiologic state of the target cells: Cells obtained from hyperconfluent cultures did not stain as well after phospholipase C treatment as those collected from subconfluent cultures. Synchronization experiments revealed that cells in late G_1 or S phase did not stain after phospholipase C treatment, while some staining of cells in mitosis was achieved after enzyme treatment. Cells harvested with ethylenebis (oxyethylenenitrolo) tetraacetic acid (EGTA) stained more satisfactorily after enzyme treatment than those removed with EDTA. It is proposed that the virus-induced surface antigens are either in a cryptic form covered by a membrane component that contains phospholipid, or else in a manner which requires phospholipase C-mediated reorganization of the membrane architecture for their detection by immunofluorescence.

2732 LYMPHOCYTE SEQUESTRATION: ITS POSSIBLE ROLE IN TUMOR IMMUNITY.

(E.) Stutman, O. (U. Minnesota Med. Sch., Minneapolis). *Transplantation Proc* 5(1):969-973, 1973.

Thoracic duct lymphocytes (TDL) from normal and tumor-bearing C3Hf/Umc mice were labeled with ^{51}Cr and injected i.v. into normal syngeneic mice. Four hr later, the organ distribution of radioactive label was observed in TDL recipients. TDL from tumor-bearing mice migrated mainly to the liver and showed up less frequently in spleen and lymph nodes than did TDL from normals. Incubation of TDL from tumor-bearers prior to labeling and subsequent injection removed the propensity of these TDL to migrate to the liver. This propensity was more pronounced, the larger the size of the tumors on tumor-bearing TDL donors. Removal of tumors from donors produced a rapid disappearance of liver-migrating TDL. B cells made up an important fraction of the liver-migrating TDL. It appeared that antigen-antibody complexes influenced the altered migratory pattern of TDL from tumor-bearing mice.

2733 ANALYSIS OF SURFACE ANTIGENS ON SIMIAN VIRUS 40-TRANSFORMED CELLS. I. UNIQUE ANTIGENICITY OF SIMIAN VIRUS 40-TRANSFORMED OUTBRED HAMSTER KIDNEY CELL LINES.

(E.) Collins, J. J. (Harvard Med. Sch., Boston, Mass.) and P. H. Black. *J Natl Cancer Inst* 51(1):95-114, 1973.

To determine the nature of the simian virus 40 (SV40) surface (S) antigen, purified SV40 and autochthonous-transformed kidney cells were used to immunize outbred golden Syrian hamsters. Immunization by repeated inoculations of virus and transformed cells or with whole cells or plasma cell membranes in complete Freund adjuvant were ineffective in raising antisera against surface membrane antigens as determined *in vitro* by the the membrane immunofluorescence (MIF) technique. Immunization by the induction of a progressively growing tumor yielded humoral activity detectable by MIF. Tumor excision studies demonstrated that the level of serum MIF activity was influenced by the tumor mass, which may act both as an antigenic stimulus and as an "antibody-sponge," and that the size of the tumor was critical in determining the dominant effect. In addition, a small tumor regrowth after excision facilitated the appearance of humoral activity which possibly reflected an anamnestic response. The pattern of reactivity with immune autochthonous sera indicated that SV40-transformed outbred hamster kidney cell lines were characterized by unique, virus-induced, host-coded antigens. Immunization of homologous hamsters raised activity primarily against hamster isoantigens, possibly to the exclusion of the virus-induced antigens. The relationship of the SV40 S antigen to normal fetal hamster antigens was not confirmed. A survey of the literature dealing with papovavirus-induced surface antigens and a discussion relating alterations in the surface membrane biochemistry to the antigenicity of virus-transformed cells are presented.

2734 THE SYNTHESIS, ASSEMBLY AND SECRETION OF GAMMA GLOBULIN BY MOUSE MYELOMA CELLS.

(E.) Buxbaum, J. (Manhattan VA Hosp., New York, N.Y.) and M. D. Scharff. *J Exp Med* 138(1):278-288, 1973.

The study of the synthesis, assembly, and secretion of IgM by seven murine myeloma tumors in BALB/c mice has revealed that free mu chain can be detected intracellularly after release from the ribosome. It combines with light chains to form μL. The major intracellular protein in six of the seven tumors was the 8S subunit. One tumor contained considerable amounts of 19S material intracellularly. Those tumors that did not contain 19S IgM intracellularly appeared to assemble the subunits outside the cell. Subunits produced by partial reduction of extracellular IgM differed in carbohydrate composition and conformation from those found intracellularly. The transition between the intra- and extracellular form of the IgM subunit could take place at the time of secretion.

2735 POLYINOSINIC-POLYCYTIDYLIC ACID IN TWO-STAGE SKIN CARCINOGENESIS. EFFECT ON EPIDERMAL GROWTH PARAMETERS AND INTERFERON INDUCTION IN TREATED MICE.

(E.) Elgjo, K. (Rikshospital, Oslo, Norway) and M. Degre. *J Natl Cancer Inst* 51(1):171-177, 1973.

Repeated i.p. administration of synthetic double-stranded RNA polyinosinic-polycytidylic acid (poly I:C) inhibited two-stage carcinogenesis in mice. Poly I:C treatment during the whole experimental period reduced the tumor yield, mortality, and latency period. The tumor yield and mortality were decreased even when poly I:C administration was started at the time of appearance of the first tumor. Interferon production after a single dose of poly I:C was not influenced by repeated topical applications of 3-methylcholanthrene or by previous treatment with poly I:C. A single small dose of poly I:C had no effect on normal epidermal DNA synthesis or mitotic rate, but inhibited the incorporation of tritiated thymidine into DNA of hyperplastic mouse epidermis. This finding may account for the longer latency period and the smaller number of tumors in animals treated with poly I:C during carcinogenesis. The results also indicate that poly I:C affects processes occurring during promotion. This may be either an unspecific damage to the epidermal cells or a more specific effect in rapidly proliferating cells.

2736 INCREASED INCIDENCE OF LUNG METASTASES FOLLOWING TREATMENT OF RATS BEARING HEPATOMAS WITH IRRADIATED TUMOUR CELLS AND THE BENEFICIAL EFFECT OF *CORYNEBACTERIUM PARVUM* IN THIS SYSTEM. *(E.)*

Proctor, J. (Chester Beatty Res. Inst., Sutton, Surrey, England), C. M. Rudenstam and P. Alexander. *Biomedicine* 19(6):248-252, 1973.

Immunization of inbred rats with irradiated tumor cells, prior to excision of a hepatoma growing in the leg, either facilitated or suppressed the blood-borne spread of metastases to the lung, depending on the amount of irradiated tumor used for immunization. Combination of *Corynebacterium parvum* or B.C.G. with irradiated tumor reversed the facilitating effect of large doses of irradiated tumor cells. Neither of these reticuloendothelial system stimulants when used alone had an effect on tumor growth or dissemination if given prior to removal of the tumor growing in the leg. Following tumor excision, immunization with irradiated tumor did not affect the incidence or number of lung metastases compared to controls, but significantly retarded their growth. Immunization with *Corynebacterium parvum* alone as well as in combination with irradiated cells, reduced the incidence and number of lung metastases when given after excision of the leg tumor. These results confirm earlier studies in showing that in experimental animals immunization with irradiated tumor cells may, depending on conditions, promote or hinder tumor growth. The harmful effect may be attributed to a rise in the level of tumor-specific antigens present in a soluble form in the circulation. The beneficial effect of immunizing with irradiated tumor cells is attributed to stimulation of lymphoid tissue distant to the growing tumor. The net effect on the tumor of immunization with irradiated tumor cells may thus be determined by a balance between 1) release of antigen into the circulation which interferes with an established host reaction; and 2) an increase in the immune response of the host, both humoral and cell-mediated, due to stimulating extra lymphoid tissue.

2737 ANTIBODY-INDUCED CHANGES IN EXPRESSION OF AN IMMUNOGLOBULIN SURFACE ANTIGEN. *(E.)*

Knopf, P. M. (Armand Hammer Ctr. Cancer Biol., Salk Inst., San Diego, Cal.), A. Destree and R. Hyman. *Eur J Immunol* 3(5):251-259, 1973.

The objective of this study was to determine whether Ig-related antigenic determinants (Ig-mem) present on the surfaces of cells of an IgG-secreting mouse myeloma line might be the immediate precursors of the IgG which these cells secrete. The Ig-mem was detected by complement-dependent sensitivity to lysis with rabbit anti-Fab and by a binding assay using ^{125}I-labeled rabbit anti-Ig. It was reasoned that the ^{125}I-labeled anti-Ig, attached to the Ig-mem while the cells were at 0 C, would permit determining the fate of the Ig-mem during subsequent incubation of the cells at the higher temperatures where secretion of IgG would occur. If Ig-mem were the immediate precursor of secreted IgG, then it should leave the cells soon after the cells were shifted to the higher temperatures, carrying with it the labeled antibody. Otherwise, the labeled antibody would be expected to remain bound to the cells. Experiments showed that shifting the temperature to 24 C or 37 C led to a rapid loss of sensitivity to lysis by complement. They also showed a loss in the capacity of the rabbit anti-Ig to be detected on the cell surface as tested with the aid of sheep anti-rabbit Ig. Further, these reactions occurred without any appreciable loss of radioactivity from cells at 24 C. The ^{125}I-labeled antibody on the cells gradually became degraded, as could be detected by the liberation of free iodotyrosine; this occurred more rapidly at 37 C than at 24 C. The results as a whole were interpreted as an antibody-induced interiorization of the Ig-mem, together with its bound antibody. The degradation of the ^{125}I-labeled antibody which was observed when sensitized cells were incubated at 37 C was believed to be due to intracellular enzymatic action on the interiorized antibody. The interiorization reaction did not occur when ^{125}I-labeled, univalent Fab fragment of the rabbit antibody was used. Instead, most of this reagent remained bound to cells for at least an hour at 24 or 37 C. These findings indicated to the authors that the bulk of Ig-mem may not be an immediate precursor of the secreted IgG, in transit through the plasma membrane.

2738 DIFFERENCES IN THE TUMOR-ASSOCIATED REACTIVITY OF BLOOD LYMPHOCYTES AND TUMOR-DRAINING LYMPH NODE CELLS IN SARCOMA PATIENTS. *(E.)*

Vánky, F. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), J. Stjernswärd, U. Nilsonne and R. Sundblad. *J Natl Cancer Inst* 51(1):17-24, 1973.

The immunologic reactivity and tumor-associated immunity of lymphoid cells from lymph nodes (LNC) draining large tumors and circulating blood lymphocytes (CBL) from the same patients were studied in the mixed-lymphocyte-target-interaction test. LNC draining large sarcomas with a history of long duration were specifically nonreactive against autochthonous tumor cells. Autochthonous tumors stimulated DNA synthesis in CBL in 7 of 12 patients tested, but tumor-draining LNC did not react against the same tumor cells. LNC were stimulated to the same degree as CBL by phytohemagglutinin or allogeneic lymphocytes. CBL reacted against autochthonous LNC from the tumor area in 7 of 12 patients. Control LNC from patients without malignant disease stimulated autochthonous CBL in only 1 of 13 patients. LNC from tumor patients were less reactive against allogeneic cells than LNC from controls. The results demonstrate a tumor-specific nonreactivity to tumors by LNC draining large tumors.

2739 IMMUNOBIOLOGICAL ENTITY OF HUMAN LEUKEMIA REPRODUCED IN CAYMAN. *(E.)*

Kwapinski, J. B. G. (Dept. Med. Microbiol., U. Manitoba, Winnipeg, Canada). *Oncology* 27(6):543-549, 1973.

Preliminary data are presented from a test of Kwapinski's hypothesis that the agent of human leukemia is a molecular parasite that established an immunobiological association with animal cells before or during the era of reptiles. A specific objective of the experiments was the transfer and reproduction of human leukemic factors (antigens) in *Caiman lithoriensis* selected as a representative of modern reptiles. Leukemic factors from extracts of bone marrow and spleen of two cases of acute lymphoblastic and acute myeloid leukemia were prepared by processes involving filtration through a 0.45-μm membrane, electrophoresis on agarose gel, and concentration by lyophilization. Five caymans were injected i.p. with the purified extract; five control caymans, with extract heated at 100 C for 15 min. Animals treated with nonheated material died in 8-9 wk. Their lymph nodes and spleens were enlarged, and extracts of these tissues were found by immunodiffusion to react with their sera but not with sera of the control caymans. The extracts also reacted with five of eight sera obtained from cases of human leukemia. The data indicate that a human leukemic entity was reproduced in caymans, yielding sufficient antigen to be detected by immunodiffusion. Additional immunological reactions are reported which suggest the existence of a phyloimmunological community, arising from the evolution of reptiles and mammals from a common ancestor.

2740 HUMAN LYMPHOCYTE SUBPOPULATIONS. HUMAN THYMUS-LYMPHOID TISSUE (HTL) ANTIGEN-POSITIVE LYMPHOCYTES FORMING ROSETTES WITH SHEEP ERYTHROCYTES AND HTL ANTIGEN-NEGATIVE LYMPHOCYTES INTERACTING WITH ANTIGEN-ANTIBODY-COMPLEMENT COMPLEXES.

(E.) Yata, J. (Dept. Pediatr., Toho U., Tokyo, Japan), I. Tsukimoto and T. Tachibana. *Clin Exp Immunol* 14(3):319-326, 1973.

Various human lymphoid tissues were studied to determine the distribution of two different lymphocyte populations, one characterized by adherence of sheep erythrocytes (E) to form rosettes and the other by adherence of sheep erythrocyte-antibody-complement complexes (EA(IgM)C43) to form rosettes. The relationship between these two populations on the one hand and the existence or absence of human thymus tissue (HTL) antigen on the other was examined. The tissue cells studied were peripheral blood leukocytes and cell suspensions of thymus, bone marrow, spleen, lymph node, and tonsil, mostly from infants and children. The different lymphoid suspensions were mixed with appropriately prepared suspensions of either E or EA(IgM)C43, incubated 1 hr at 37 C, allowed to settle, smeared on a glass slide, dried, and stained with Giemsa or examined for immunofluorescence by a previously described test for HTL antigen. Lymphoid cells were observed for the presence of HTL antigen and binding of sheep erythrocyte at the same time under the UV microscope. Almost all E rosette-forming lymphocytes were HTL positive, while cells binding EA(IgM)C43 were always HTL negative. From these data and additional considerations the authors conclude that E-rosette-forming and HTL antigen-positive lymphocytes represent T cells of thymus origin and that EA(IgM)C43-rosette-forming and HTL antigen-negative cells are not thymus-dependent and may be precursors of antibody-forming cells. However, while the thymus cells showed the highest percentages of E-rosette-forming cells, they also showed the highest percentages of EA(IgM)C43-rosette-forming cells; also, although cells from bone marrow showed the lowest percentages of E-rosette-forming cells, they also showed the lowest percentages of EA(IgM)C43-rosette-forming cells.

2741 *IN VITRO* INDUCTION OF TUMOR-SPECIFIC IMMUNITY. I. PARAMETERS OF ACTIVATION AND CYTOTOXIC REACTIVITY OF MOUSE LYMPHOID CELLS IMMUNIZED *IN VITRO* AGAINST SYNGENEIC AND ALLOGENEIC PLASMA CELL TUMORS.

(E.) Wagner, H. (Hall Inst. Med. Res., Victoria, Australia) and M. Rollinghoff. *J Exp Med* 138(1):1-15, 1973.

Induction of tumor-specific immunity *in vitro* was accomplished by cocultivation of cortisone-resistant murine thymocytes or spleen cells with irradiated syngeneic plasma cell tumors (PCT). The cytotoxic activity generated could be detected in a short-term ^{51}Cr-release assay. Optimal cytotoxic activity against PCT-associated transplantation antigens (TATA) was generated after 7 days in culture. Unlike cytotoxic responses to tumor allografts in which the cytotoxic activity was directed against allogeneic transplantation antigens, the cytotoxic activity obtained in the syngeneic tumor system was specific to the immunizing syngeneic PCT. Similar parameters of induction of cytotoxic responses in *in vitro* tumor allograft responses and in the syngeneic tumor system suggested that both reactions are cell-mediated cytotoxic immune responses. With regard to the magnitude of cytotoxic responses obtained, allogeneic transplantation antigens induced about a 30-fold higher cytotoxic immune response than plasma cell TATA. The results are consistent with the concept that *in vitro* tumor allograft responses and *in vitro* responses against TATA of PCT are similar in quality, but differ in the magnitude of the cytotoxic response provoked.

2742 THE APPEARANCE OF RETICULUM CELL SARCOMA AT THE SITE OF ANTILYMPHOCYTE GLOBULIN INJECTION.

(E.) Cotton, J. R. (U. Texas Med. Branch, Galveston), H. E. Sarles, A. R. Remmers, Jr., J. D. Lindley, G. A. Beathard, D. L. Cottom, J. C. Fish, C. M. Townsend, Jr. and S. E. Ritzmann. *Transplantation* 16(2):154-157, 1973.

A case history is presented of a patient who developed a reticulum cell sarcoma at the site of antilymphocyte globulin (ALG) injection following 1 yr of therapy. The patient was prepared for cadaveric renal transplantation by depletion of circulating lymphocytes by thoracic duct drainage. He subsequently received a cadaveric kidney followed by immunosuppressive drugs as well as a prolonged course of ALG. Five months after discontinuing ALG he developed a reticulum cell sarcoma at the site of injection. He was treated with resection of the neoplasm, reduction of immunosuppressive drugs, and local irradiation. He developed tumor spread to the inguinal lymph nodes but subsequently showed no evidence of further tumor spread. The most likely explanation for the occurrence of a malignant tumor at the site of ALG injection centers around the known property of ALG to induce blastic transformation of lymphocytes. If a large amount of ALG is deposited in soft tissues and remains for long periods of time, circulating lymphocytes brought into contact with this material would be stimulated to undergo blastic transformation. It is feasible to consider that with time the local supply of complement would be dissipated, leaving lymphocytes free to proliferate into malignant lymphomas.

2743 REGULATIONS OF THE IMMUNE RESPONSE. II. QUALITATIVE AND QUANTITATIVE DIFFERENCES BETWEEN THYMUS- AND BONE MARROW-DERIVED LYMPHOCYTES IN THE RECOGNITION OF ANTIGEN. *(E.)* Hoffman, M. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.) and J. W. Kappler. *J Exp Med* 137(3):721-739, 1973.

The specificity of antigen recognition by thymus-derived helper cells (T cells) and antibody was examined in 8-12 wk old hybrid mice (BDF$_1$), heterologous erythrocyte antigens from sheep (SRBC), goat (GRBC),burro (BRBC), chicken (CRBC), and toad (TRBC). Antibody specificity was tested by several functional assays: hemagglutination, hemolysis, and immune suppression. The specificity of T cells was determined by titrating their ability to aid the *in vitro* antitrinitrophenol responses of mouse spleen cultures immunized with the hapten coupled to the various test erythrocytes as carrier. Anti-SRBC antibody cross-reacted with GRBC but not with BRBC, CRBC, or TRBC. In contrast, SRBC-primed helper T cells crossed-reacted with both GRBC and BRBC but not with CRBC or TRBC, thus indicating a difference in the specificity of antigen recognition between the cellular and the humoral immune responses.

2744 THE INFLUENCE OF IMMUNOSUPPRESSION AND SEX HORMONE MANIPULATION ON MAMMARY TUMOUROGENESIS IN THE RAT. *(E.)* Hunt, P. S. (Dept. Surg., Monash U., Melbourne, Australia) and E. D. Holyoke. *Aust J Exp Biol Med Sci* 51(pt 3):417-420, 1973.

The 7,12-dimethylbenzanthracene (DMBA) induced rat mammary tumor model was chosen as a system for the investigation of the effect of sex hormones and immunosuppression on later incidence of tumors. At 50 days of age three of four groups of 40 female syngeneic Sprague Dawley rats were oophorectomized and one group sham operated. At 75 days of age all rats were given 3 mg of DMBA in olive oil by gastric feeding. Each of the four groups was subdivided into two subgroups, one receiving antithymocyte globulin (ATG) and the other normal rabbit globulin. After induction of mammary tumor by DMBA two of the four groups were started on i.m. estradiol 2.5 µg daily and i.m. testosterone 1 mg daily. Prior oophorectomy significantly reduced tumor incidence. No increase in tumor incidence after immunosuppression with ATG was shown unless the rats were previously oophorectomised. Subsequent treatment with oestradiol restored the carcinogenic action of DMBA in oophorectomised animals and testosterone inhibited tumor development in oophorectomised, immunosuppressed rats.

2745 FOETAL ANTIGENS CROSS-REACTIVE WITH TUMOUR-SPECIFIC TRANSPLANTATION ANTIGENS. *(E.)* LeMevel, B. P. (Natl. Cancer Inst., Bethesda, Md.) and S. A. Wells, Jr. *Nature [New Biol]* 244(136): 183-184, 1973.

Most investigators who demonstrated the relationship between fetal antigens and antigens of experimental tumor cells have used rodent neoplasms induced by oncogenic viruses. Studies on the similarity of fetal antigens to chemical carcinogen-induced tumor cell antigens have been fewer and less conclusive. For this reason, the similarity between fetal antigens and tumor-specific transplantation antigens of methylcholanthrene-induced sarcoma cells of C57Bl/6n mice was investigated. Separate groups of 100 3-4 month-old female mice each were immunized in courses of five injections 8 days apart with 10^5-10^6 syngeneic lymphocytes syngeneic MCA-10 tumor cells, or fetal cells from 10-12-day-old whole C57Bl/6n embryos. All cells were X-irradiated before injection. On the 11th day, each group, together with a group of 100 8-10-month-old multiparous female mice exposed to fetal antigen during pregnancy, was subdivided into three groups which were then challenged with 10^3, 10^4, or 10^5 syngeneic MCA-10 tumor cells. Compared with the control lymphocyte-immunized animals, mice immunized with MCA-10 cells showed the greatest protection against challenge with 10^5 cells, followed by mice immunized with fetal cells. When challenged with 10^4 cells, multiparous animals as well as those immunized with tumor cells and fetal cells had significantly fewer tumors than controls.

2746 TUMORIGENICITY ASSAYS IN NONHUMAN PRIMATES TREATED WITH ANTITHYMOCYTE GLOBULIN. *(E.)* Petricciani, J. C. (Food Drug Admin., Rockville, Md.), R. L. Kirschstein, J. E. Hines, R. E. Wallace and D. P. Martin. *J Natl Cancer Inst* 51(1):191-196, 1973.

Newborn infant and juvenile African green (*Cercopithecus aethiops*) and rhesus monkeys (*Macaca mulatta*) treated with antithymocyte globulin (13-50 mg/kg injected s.c. one day before and one and 3 days after cell inoculation) were used to test for the tumorigenic potential of cell lines of human neoplastic, mammal virus-transformed, primate abnormal karyology, and human and nonhuman primate diploid origin. Cells of neoplastic origin and those transformed with virus (1.0 ml i.m.) were tumorigenic in this test system, whereas the other cell types evidenced no tumor formation upon open biopsy between days 9 and 16 and microscopic examination of inoculation sites after 9 months. Amongst the cells of neoplastic origin KB cells gave rise to grossly observable masses and clear histologic evidence of tumor cell growth, and HeLa, HEp-2, and Led-WIDR cells produced palpable masses which were generally smaller than the KB tumor masses. The histology of biopsies of these inoculation sites showed a much more vigorous reaction of connective tissue, lymphocytes, and plasma cells than KB inoculation sites. Amongst the virus-transformed cell lines, Led-T$_1$ and Led-SVH$_2$ were tumorigenic in a manner resembling HeLa, HEp-2 or Fed-WIDR, and TV-106-CI was tumorigenic in two of three animals tested. The results suggest that the nonhuman primate conditioned with antithymocyte globulin can be a useful system to assess the tumorigenic potential of cells for man.

2747 CARCINOEMBRYONIC ANTIGEN AND BLOOD GROUP SUBSTANCES.

(E.) Alastair, D. (Dept. Immunol., Wenner-Gren Inst., Stockholm, Sweden), R. Simmons and P. Perlmann. *Cancer Res* 33(2):313-322, 1973.

Immunochemical studies support the view that carcinoembryonic antigens from entodermally derived adenocarcinomata are incomplete blood group substances of the ABO system. The deficient antigens have a tumor-specific activity, due to unmasking of a structural sequence that is cryptic in the normal situation. Two other serological activities of the tumor antigens are determined by N-acetylgalactosaminyl peptide and antigenic Complex I sequences identical to those found in blood group substances. The structural results imply that the genetic changes in oncogenesis include deletion or repression of the gene(s) controlling the trasferase(s) for the terminal sequences of classical ABO-active glycoproteins. The development of specific serodiagnostic tests for tumors of entodermal origin will probably depend on the production of sera with high activity for the tumor-specific determinant and little or no activity for the normal blood group sequences also found in carcinoembryonic antigen.

2748 LEUKOCYTE INTERFERON PRODUCTION, RNA SYNTHESIS, AND PHA RESPONSE IN PATIENTS WITH INFECTIOUS MONONUCLEOSIS.

(E.) Rassiga Pidot, A. L. (Dartmouth-Hitchcock Med. Ctr., Hanover, N. H.), L. H. Maurer and O. R. McIntyre. *Blood* 42(2):175-185, 1973.

Since interferon (IF) production in response to virus infection is important in limiting *in vivo* virus proliferation, and since leukocytes incubated *in vitro* with virus are capable of producing IF, the present study was carried out to determine the ability of leukocytes obtained at diagnosis and throughout the course of infectious mononucleosis (IM) to produce IF either spontaneously or in response to *in vitro* viral infection. Sequential measurements of *in vitro* Newcastle disease virus-induced IF synthesis, of spontaneous RNA synthesis, and of phytohemagglutinin (PHA)-induced blastic transformation were made in leukocytes derived from 10 IM patients. IF assay was based on the ability of test fluid to protect a monolayer culture of foreskin-derived fibroblasts against challenge with vesicular stomatitis virus. RNA synthesis in control and PHA-stimulated cultures was followed by uptake of ^{3}H-uridine. It was found that the median leukocyte IF production on the day of diagnosis was significantly depressed, 35 international units (IU) IF/2 x 10^6 leukocytes, in comparison to a median titer of 463 IU after recovery or to a median of 406 IU found in cultures from normal individuals. No spontaneous production of leukocyte IF or circulating serum IF was noted. An increased rate of spontaneous leukocyte RNA synthesis during the first 2 hr of culture was noted in 9 of the 10 subjects. Cultures from 4 of the 10 patients showed a markedly defective response to PHA after 96-144 hr incubation. A suggestive, but not statistically significant, correlation was noted between increased spontaneous RNA synthesis, defective late PHA transformation, and defective virus-induced IF production. All defects persisted during the 1 wk study period and were resolved after recovery. It was concluded that major temporary alterations occur in several parameters of leukocyte function in IM.

2749 ISOLATION OF α-FETOPROTEIN-PRODUCING CELLS FROM YOSHIDA ASCITES SARCOMA AND ITS CLONE.

(E.) Isaka, H. (Sasaki Inst., Hokkaido U., Tokyo, Japan), S. Umehara, H. Hirai, Y. Tsukada and H. Wataba. *Gann* 64(2):133-138, 1973.

It is accepted that α-fetoprotein is a product of hepatic cells of embryonic, neonatal, and cancerous state as well as of a "regenerating condition" found in the rat. It was of interest, therefore, to determine whether evidence for production of this protein in Yoshida ascites tumors might be obtained by means of *in vitro* cloning of the tumor. Yoshida tumor was originally considered to be of hepatic origin, and some evidence had previously been reported for production of α-fetoprotein in karyotypic variants of the tumor. Tests for the protein were carried out on culture media from the clones, using double diffusion, single radial immunodiffusion and ^{125}I-radioimmunoassay methods. Out of the 10 clones, two distinctly produced α-fetoprotein. Out of 13 subclones of one of the productive clones, eight produced the protein in variable amounts. This phenotypic variation in production of α-fetoprotein was not accompanied by chromosomal variation. Moreover, the clones and the original ascites tumor had the same karyotype. It is likely that the Yoshida ascites tumor originally contained cells producing α-fetoprotein but that the concentration of the protein produced is insufficient for detection. The data provide a basis for the theory on the liver cell origin of Yoshida ascites tumor.

2750 7S ANTI-TUMOUR ANTIBODIES AND ACTIVATED Fc IN MACROPHAGE TUMOUR CELL INTERACTION.

(E.) Fakhri, O. (Westminster Med. Sch., London, England), H. McLaughlin and J. R. Hobbs. *Eur J Cancer* 9(1):19-23, 1973.

Addition of normal peritoneal macrophages from non-tumor-bearing rats or mice to suspensions of mouse ascitic plasmacytoma cells which had been coated with rat 7S anti-mouse plasmacytoma antibody resulted in aggregation of macrophages and tumor cells within 30 min. Within 1 hr, all target cells were dead, as determined by the Trypan blue exclusion test. Aspiration of peritoneal cavities of mice one hour after i.p. inoculation of coated tumor cells showed an *in vivo* aggregation similar to that seen *in vitro*. Control tests showed that no aggregation occurred when uncoated tumor cells were inoculated. Protective immunization was demonstrated by the finding that tumors developed in mice which received as much as 200,000 7S-coated tumor cells, whereas, 50% of control mice receiving only 1000 uncoated tumor cells died. Inoculation of 500,000 coated tumor cells, however, resulted in tumor growth in all animals tested, and tumors also developed in animals which had been inoculated with 50,000 coated cells and which were challenged one month later with 10,000 uncoated tumor cells. Coating of tumor cells with Fab alone failed to activate peritoneal macrophages. That Fc must be activated before it can interact with macrophages was suggested from experiments in which rat or mouse macrophages incubated with "recycled" 7S (i.e. free of aggregated 7S antibodies and activated Fc) failed to aggregate tumor cells.

2751 NONVIRION ANTIGENS PRODUCED BY HERPES SIMPLEX VIRUSES 1 AND 2. *(E.)* Tarro, G. (Inst. Clin. Med., U. Naples, Italy) and A. B. Sabin. *Proc Natl Acad Sci USA* 70(4):1032-1036, 1973.

As a means of investigating the possible role of herpes simplex virus in the etiology of certain human tumors, a study was made of nonvirion antigens found to be present in cultured cells infected by the virus. Tests for antigen-antibody reaction were made by complement fixation. Guinea pig antisera to nonvirion antigens from cells injected with Type 1 herpes virus failed to react with type 2 nonvirion antigens. However, type 2 nonvirion antisera reacted with both type 1 and type 2 nonvirion antigens. This indicated that type 1 virus codes only for type 1 nonvirion antigen, while type 2 virus codes not only for an immunologically distinct type 2 nonvirion antigen but also for enough type 1 nonvirion antigen to stimulate antibody production for it. Sera from patients with lesions caused by type 2 virus and from randomly selected adults failed to react with either type 2 or type 1 nonvirion antigens. Basic information was thus provided for tests yet to be carried out on the reactivity of sera from patients with different types of cancer.

2752 THYMIC CHANGES IN THE MAGNESIUM-DEPLETED RAT. *(E.)* Alcock, N. W. (Sloan-Kettering Inst. Cancer Res., New York, N. Y.), M. E. Shils, P. H. Lieberman and R. A. Erlandson. *Cancer Res* 33(9):2196-2204, 1973.

The effects of magnesium-deficient diet fed to male Sprague-Dawley rats for approximately 65 days was assessed with special reference to changes in the thymus. The thymus was enlarged in 18 to 52% of deficient animals surviving more than 6 to 7 wk in various experiments. The remainder demonstrated glands that were smaller than controls. The enlarged thymuses showed marked cellular changes with the normal structure being replaced by cells that morphologically resembled transformed lymphocytes. Of the small glands, 19% had focal or lobular cellular changes similar to those seen in enlarged thymuses. No distant metastases were found and the changes have been interpreted as hyperplastic rather than neoplastic. Prolonged magnesium depletion was accompanied by hypomagnesemia and hypercalcemia or normocalcemia. Marked leukocytosis was present during the early stages of the deficiency. Splenomegaly was consistently found in the magnesium-depleted animals. The sequence of events by which magnesium deficiency in the rat causes the changes described is unknown. The present work suggests that two distinct populations of abnormal thymuses exist among the magnesium-depleted rats. The very small thymuses seen in some deficient animals may represent a further stage in thymic alteration. The abnormalities may be related to some immunological change in the thymocytes leading to transformation and division of cells with consequent enlargement of the gland and loss of normal architecture.

2753 BLASTOGENIC TRANSFORMATION ASSAY FOR DETECTION OF IMMUNOLOGIC REACTIVITY TO AVIAN ONCORNAVIRUSES. *(E.)* Lopez, D. M. (U. Miami Sch. Med., Fla.), M. M. Sigel and P. Meyers. *J Natl Cancer Inst* 51(1):303-305, 1973.

Studies were undertaken to determine if it is possible to detect immunologic reactivity to avian leukosis-sarcoma viruses in infected chickens by the leukocyte blastogenic assay. The leukocytes of white Leghorn chickens immunized with live Rous-associated viruses (RAV), Rav-1 or RAV-6, responded in an *in vitro* leukocyte blastogenic transformation assay when stimulated with either the homologous or heterologous viruses as antigens. This phenomenon appeared related to a cellular response to a common or cross-reacting cell surface antigen induced by several virus subgroups of the avian leukosis-sarcoma complex and seemed to correlate well with other recent *in vivo* and *in vitro* studies. The reactivity of immune leukocytes to heterologous virus is likely a manifestation of cellular immunity to a tumor-specific surface antigen (TSSA) or virus-induced surface antigen (VISA) common to the avian oncornaviruses. Whether this VISA or TSSA is present in the tissue culture preparations of virus or whether it is acquired and carried by the virion itself as a consequence of budding from an infected cell is not known.

2754 IMMUNOGLOBULIN AND GLUCOSE-6-PHOSPHATE DEHYDROGENASE AS MARKERS OF CELLULAR ORIGIN IN BURKITT LYMPHOMA. *(E.)* Fialkow, P. J. (Dept. Med. Genetics, U. Washington, Seattle), E. Klein, G. Klein, P. Clifford and S. Singh. *J Exp Med* 138(1):89-102, 1973.

Two independent marker systems, G-6-PD isoenzymes and cell membrane-associated IgM, were used to trace the cellular origin of Burkitt lymphoma. Application of the G-6-PD system is dependent upon the fact that, in accordance with inactivity of one X chromosome in each somatic cell, females heterozygous for the usual B gene (Gd^B) at the X-linked G-6-PD locus and the variant allele Gd^A (or Gd^{A-}) have two types of cells. Gd^B is active in one cell population, which consequently produces B type enzyme; in the other population Gd^A is active, producing the variant A enzyme. Therefore, tumors with a clonal origin in a Gd^B/Gd^A heterozygote should exhibit only one enzyme type (B or A) whereas those with multicellular origin may show both A and B enzymes. Utilization of the immunoglobulin system is based upon the supposition that in lymphoid neoplasms with clonal origin either all or none of the tumor cells should have surface-associated IgM and K-reactivities. Thirty-three of 34 relatively homogeneous (with respect to content of neoplastic cells) individual Burkitt tumors from 19 G-6-PD heterozygotes had single enzyme phenotypes. Similarly, of 95 tumors tested, 92 consisted essentially of IgM(+) or (-) cells. Two neoplasms could not be definitely classified and one tumor had two cell populations. These data suggest a clonal origin for most Burkitt tumors, but the one neoplasm with a double G-6-PD phenotype (A/B) and the one tumor that had two populations of cells with respect to surface IgM, could have originated from multiple cells. G-6-PD was determined in each of two tumors from seven heterozygotes and in all cases both tumors had the same single enzyme phenotype. Surface-associated IgM was tested in four tumors from one patient, three from another, and in two neoplasms from 11 patients. With one exception, all tumors from the same patient were concordant with respect to IgM. These findings suggest that the entire disease has a clonal origin, i.e., it emerges at one focus and then spreads to other parts of the body.

2755 ANTIGENICITY OF RADIOSTRONTIUM-INDUCED OSTEOSARCOMAS.

(E.) Nilsson, A. (Res. Inst. Natl. Defense, Sundbyberg, Sweden), L. Revesz and K. H. Eriksson. *Radiat Res* 52(2): 395-408, 1972.

To test the antigenicity of osteosarcomas obtained from CBA mice in which the tumors were induced by i.p. injection of 0.7 μCi/g body wt of ^{90}Sr $(NO_3)_2$, heavily preirradiated tumor material was used to immunize syngeneic mice. One group of controls consisted of mice administered preirradiated normal syngeneic tissue; another group of controls was untreated. All mice were ultimately challenged with graded doses between 5×10^1 and 1×10^5 of viable cells. A parallel set of experiments was carried out in which the mice were subjected to 400 R whole body irradiation subsequent to immunization and 24 hr prior to challenge. For the unirradiated animals, immunization with normal cells inhibited the growth of tumor grafts, thought to be due to an unspecific boosting of the primary response; immunization with tumor cells increased the growth of tumor grafts, possibly a reflection of immunological enhancement. For the irradiated animals, on the other hand, immunization with normal cells caused no significant inhibition, while immunization with tumor cells did inhibit growth of challenge cells. The different results obtained with irradiated, compared to nonirradiated, animals was explained by the effect of whole body irradiation in suppressing the primary immune response, not the secondary response. It was concluded that the relative transplantation resistance of irradiated animals pretreated with irradiated tumor cells may be attributed to an immune reaction to some specific transplantation antigen possessed by the strontium-induced tumors and lacking in normal tissues.

2756 DETECTION OF CIRCULATING HEPATOMA D23 ANTIGEN AND IMMUNE COMPLEXES IN TUMOUR BEARER SERUM.

(E.) Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), J. G. Bowen and M. R. Price. *Br J Cancer* 28(1):16-24, 1973.

Serum from rats bearing progressively growing aminoazo dye-induced rat hepatomas has been fractionated by Sephadex G150 gel filtration chromatography and isolated fractions were examined by indirect membrane immunofluorescence techniques to detect tumor specific antigen and antibody. Hepatoma D23-specific antigenic activity was associated with material (of approximate molecular wt <150,000) isolated in the inclusion volume of the gel at pH 7.3. The fraction excluded from the gel (of approximate molecular wt >150,000) was adjusted to pH 3.0 and further separated by Sephadex G150 gel filtration chromatography at pH 3.0 into gel included and excluded fractions. Hepatoma D23 specific antibody, demonstrable by membrane immunofluorescence staining of hepatoma D23 cells, was found to be eluted in the excluded volume and specific antigenic activity was retarded into the included volume of the gel. These results indicate that hepatoma D23 bearer serum contains free circulating tumor specific antigen in excess, together with specific immune complexes. A combination of several factors may be operative in determining the relative concentration of free and complexed tumor antigen, such as the height of the tumor immune response, the relative contribution of cellular and humoral reactions and the extent of tumor antigen release governed by tumor cell turnover. The presence of the free and complexed tumor antigen in tumor bearer serum is discussed in terms of "blocking" phenomena whereby serum factors may protect tumor cells from sensitized lymphocyte cytotoxic attack.

2757 RADIOIMMUNOASSAY OF MOUSE PROLACTIN. PROLACTIN LEVELS IN ISOGRAFT-BEARING ORCHIDECTOMIZED MICE.

(E.) Boot, L. M. (Netherlands Cancer Inst., Amsterdam), H. G. Kwa and G. Röpcke. *Eur J Cancer* 9(3):185-193, 1973.

Two groups of 24 (C57BL X CBA)F_1 mice each were orchidectomized at the age of 6-8 wk and received a hypophyseal isograft under the kidney capsule. One group received progesterone pellets implanted subcutaneously. Each group was divided into four sub-groups, which received estrone in the drinking water at four dose levels: respectively 1 mg/liter, 250 μg/liter, 12.5 μg/liter and 1.25 μg/liter. Plasma samples were taken from each mouse by puncture of the retrobulbar venous sinus on days 7, 21, 190, 270, 360 and 450 (day of operation = day 0). The assay results in the group of mice receiving no implants of progesterone pellets were suggestive of a dose-response relationship between estrone and plasma prolactin values extending over the whole range of the estrone dose levels employed. The higher levels of prolactin observed in the 1 mg/liter sub-group compared to those in the 250 μg/liter sub-group, however, could be ascribed to the contribution by the *in situ* glands, which enlarged tumorously in all the mice only in this sub-group. A real estrone dose-related secretory response of the isografts was observed in the three sub-groups respectively on 250 μg/liter, 12.5 μg/liter and 1.25 μg/liter. In contrast only suggestive evidence was obtained for a dose-response relationship between either estrone or plasma prolactin levels induced by this steroid on the one hand and mammary tumor incidence or the average age attained by the tumor-bearing mice in the various sub-groups on the other. Progesterone was found to have a clear-cut synergistic effect on the carcinogenic action of continuously increased plasma prolactin levels. More mammary tumors developed earlier in the presence of progesterone. The results in the mice on 1 mg/liter estrone in their drinking water indicated a "protective" action by higher dose levels of estrone against the induction of mammary tumors. The induction of pituitary tumors - to a size incompatible with survival by compression of the brain before the induction of mammary tumors - could not satisfactorily explain this for all cases, since in progesterone supplemented mice the development of tumorous enlargement of the pituitary by 1 mg/liter estrone in drinking water was completely prevented and yet the mammary tumor incidence declined.

2758 MOBILITY OF CARBOHYDRATE CONTAINING SITES ON THE SURFACE MEMBRANE IN RELATION TO THE CONTROL OF CELL GROWTH.

(E.) Inbar, M. (Dept. Genetics, Weizmann Inst. Sci., Rehovot, Israel) and L. Sachs. *FEBS Letters* 32(1):124-128, 1973.

Fluorescein-conjugated concanavalin A (F-con A) was used as a probe to study the fluidity of normal and transformed hamster embryo fibroblasts and murine lymphocytes. Incubation of normal fibroblasts with F-con A produced a random distribution of fluorescence. Incubation of normal rat lymphocytes with F-con A resulted in a nonrandom distribution of fluorescence in the form of "caps". Viral or chemically transformed hamster cells or viral induced mouse lymphoma cells showed random clusters of fluorescence following incubation with F-con A. Similar numbers of F-con A molecules were bound per cell in each case. These results suggest that in cells which form a solid tissue, the transformation of normal into malignant cells is associated with increased membrane fluidity of the F-con A binding sites. However, cells normally in suspension show a decreased membrane fluidity upon malignant transformation.

2759 SYNTHESIS OF EPSTEIN-BARR VIRUS ANTIGENS AND DNA IN ACTIVATED BURKITT SOMATIC CELL HYBRIDS.

(E.) Glaser, R. (Milton S. Hershey Med. Ctr. Pennsylvania State U., Hershey), M. Nonoyama, B. Decker and F. Rapp. *Virology* 55(1):62-69, 1973.

The synthesis of Epstein-Barr virus (EBV) specific antigens and the increase in EBV DNA have been observed previously for somatic cell hybrids of Burkitt lymphoblastoid cells (D98/HR-1) after treatment with 5-iododeoxyuridine (IUDR). The present study was carried out to determine the particular antigens detectable and the level of EBV DNA induced under these conditions. The antigens investigated were early antigens (EA), membrane antigens (MA) and virus capsid antigens (VCA). It was determined by subcloning the cultures that every cell contained complete and inducible EBV genomes as revealed by treatment with IUDR. Nuclear EA was observed in IUDR-treated cells in the absence of EBV-specific DNA synthesis. MA and VCA were only observed in cells after removal of IUDR and the initiation of EBV DNA synthesis. EBV DNA synthesis occurred rapidly after removal of IUDR and in two distinct bursts.

2760 SPECIFIC SUPPRESSION OF IMMUNE RESPONSES. ANTIBODY DIRECTED AGAINST EITHER ANTIGEN OR RECEPTORS FOR ANTIGEN CAN SUPPRESS IMMUNITY SPECIFICALLY.

(E.) Rowley, D. A. (Dept. Path., U. Chicago, Illinois), F. W. Fitch, F.P. Stuart, H. Köhler and H. Cosenza. *Science* 181:1133-1141, 1973.

Specific suppression of immune reactivity produced in normal adult animals by antibody and by antigen is discussed. The mechanism of homeostasis of suppression depends on continued exposure to antigen and on active response by the host. The active response may include production of antibody directed against specific receptors as well as antibody directed against antigen. Thus, specific regulation of both antibody and cell mediated immunity to an antigen might be achieved by the use of only the biological agents of the response: antigen, antibody, and possibly antibody to receptors. It is implied that these biological agents are responsible for autoregulation of immune reactions occurring in nature. These agents may be used to suppress or reverse immune responses for approriate clinical objectives.

2761 THE EFFECT OF CARCINOGENS ON IMMUNE RESPONSES IN THE GUINEA-PIG.

(E.) Dale, M. M. (Dept. Pharmacol., U. Coll. London, England) and J. R. Hedges. *Br J Exp Pathol* 54(4):437-448, 1973.

A corollary of the view that immunological mechanisms may act as a defense against tumors is the view that one aspect of the action of carcinogenic chemicals may be a primary immunosuppression. To test the latter, the short-term and long-term effects of the administration of two carcinogens, diethylnitrosamine and methylcholanthrene, on immunological responses in guinea-pigs have been investigated. Immune responsiveness was measured by histamine release from the lungs of animals sensitized to ovalbumin, delayed hypersensitivity reaction to tuberculin, autoimmunity to testis antigen, and normal lymphocyte transfer reactions with cells from carcinogen-treated donors injected into irradiated recipients. The results showed that in the period immediately after commencement of carcinogen administration, one aspect of the delayed hypersensitivity reaction to tuberculin (induration) was decreased in the carcinogen-treated animals as compared with the controls. This could possibly reflect a reaction to the immediate toxic effect of the chemicals. In the main long-term study, however, in which the immunological responses were tested 6-9 months after commencement of carcinogen treatment (i.e. where the administration schedules, especially for diethylnitrosamine, could and did result in subsequent tumor induction), there was no immunodepression indicated by any of the tests used. It is considered probable that the tumorigenic effect of the two carcinogens is due to their direct carcinogenic properties and is not aided by any substantial nonspecific immunosuppression.

2762 THE RESPONSE TO PHYTOHAEMAGGLUTININ (PHA) OF LYMPHOCYTES FROM CANCER PATIENTS. *(E.)*

Kumar, S. (Dept. Bact. Virol., U. Manchester, England) and G. Taylor. *J Clin Pathol* 26(7):476-479, 1973.

Cellular immune potential in 100 cancer patients and 31 normal subjects was assessed by uptake of ^{3}H-thymidine by lymphocytes in the presence and absence of phytohemagglutinin (PHA). The effects of addition of autologous or homologous serum to the culture medium were also tested. Significant differences were found between normal subjects and cancer patients in both the time course and degree of uptake. Thus, the peak uptake of ^{3}H-thymidine by lymphocytes from healthy individuals occurred at 72 hr, either in the presence or absence of PHA; the peak uptake for cancer patients was often later under the same conditions. This confirmed some earlier reports. It was suggested that the comparatively low responses observed in the absence of PHA might have been due to antibiotics and serum-gelatin mixture in the culture media. Sera from cancer patients depressed the response of normal lymphocytes while sera from normal patients restored the response of lymphocytes from cancer patients to normal, indicating either the presence of an inhibitor, or the absence of a growth factor in the sera of cancer patients. It was not known whether the depressed lymphocyte transformation and the inhibitory action of serum of cancer patients represented partial failure of immunological surveillance and hence might be important etiologically or was a result of the diseased state. Certain of the findings suggested, however, a tumor-dependent effect rather than operation of an etiological factor.

2763 HAEMOPOIETIC STEM CELLS AND PROGENITORS OF FUNCTIONAL T-LYMPHOCYTES IN THE BONE MARROW OF 'NUDE' MICE. *(E.)*

Pritchard, H. (Dept. Zool., U. Edinburgh, Scotland) and H. S. Micklem. *Clin Exp Immunol* 14(4):597-607, 1973.

This study was designed to explore various possible lesions which might cause failure in thymic development. The stem cell potential of thymusless *nu/nu* mice measured in terms of colony forming unit (CFU) ratio and the capacity to reprieve lethally irradiated mice was similar to that of *nu*/+ and CBA mice. The thymus of *nu/nu*→ T6T6 radiation chimaeras was repopulated by donor cells. Proliferation of donor cells, presumed to be T-lymphocytes, was observed in regional lymph nodes 3 days after sensitization with oxazolone. T6T6 thymus grafts implanted subcutaneously in *nu/nu* mice restored the response to C57BL skin grafts. The mitotic figures in the thymus implant 30 and more days after grafting were almost exclusively of host karyotype. The high proportion of host cells dividing in the lymph nodes of thymus-grafted *nu/nu* mice suggested that T-cells derived originally from *nu/nu* bone marrow were present. Allogeneic epithelial thymus grafts partially restored the response of *nu/nu* mice to oxazolone. It is concluded that the failure of thymic development in *nu/nu* mice is not due to an absence of thymocyte precursors in the bone marrow or to anything in the general physiological environment which prevents their differentiation to thymocytes and T-lymphocytes. It is presumably the result of defective embryonic development of the epithelial rudiment.

2764 DOES THE ABSENCE OF IMMUNOLOGIC SURVEILLANCE AFFECT THE TUMOR INCIDENCE IN "NUDE" MICE? FIRST RECORDED SPONTANEOUS LYMPHOMA IN A "NUDE" MOUSE. *(E.)*

Custer, R. S. (Fox Chase Ctr. Cancer Med. Sci., Philadelphia, Pa.), H. C. Outzen, G. J. Eaton and R. T. Prehn. *J Natl Cancer Inst* 51(2):707-711, 1973.

Because of absence of their thymuses and consequent high susceptibility to infection, "nude" mice have a short lifespan which may account for their apparent freedom from spontaneous malignant disease. The unusual finding of a malignant lymphoma, reticulum cell type B, is described in this work. Because the predominant tumors in immunologically suppressed humans or lower animals are lymphoreticular, it might be expected that the predominant tumors in "nude" mice would be the same. It was suggested that the apparent complete freedom of "nude" mice from epithelial or mesenchymal tumors may arise through lack of immunostimulation by T cells, i.e., lack of immunologic "boost" during the incipient phase of tumor development that is either helpful or required for tumorigenesis. The athymic "nude" mice, deficient in thymus-derived (T) cell-mediated immunologic responses, may provide a model for testing immunosurveillance and/or immunostimulation as active modulators of tumor regression or development. The apparent rarity of lymphoreticular tumors in this stock is of interest; it may elucidate the role of immune responses in oncogenesis.

2765 LYMPHOCYTE ACTIVATION. V. QUANTITATION OF THE PROLIFERATIVE RESPONSES TO MITOGENS USING DEFINED T AND B CELL POPULATIONS. *(E.)*

Janossy, G. (Div. Immunol., Clin. Res. Ctr., Harrow, England), M. F. Greaves, M. J. Doenhoff and J. Snajdr. *Clin Exp Immunol* 14(4):581-596, 1973.

Although mitogens are frequently used for polyclonal lymphocyte stimulation in both experimental and clinical studies, biological tests of this kind encounter several technical difficulties. Assays are required where response measured by ^{3}H-thymidine uptake increases when the number of activated cells increases. This requirement can be approached by application of a suitable "calibration" curve, obtained by relating cell concentration to isotope incorporation. A linear relationship between the responding cell number and incorporated thymidine could be achieved by adding sufficient amount of thymidine in the form of isotope of relatively low specific activity (50 mCi/mmol). In the present experiments, thymocytes from cortisone-treated CBA/Lac mice (T cells) and spleen cells from T cell-deprived mice (B cells) were mixed in different known proportions and stimulated by Concanavalin A (Con-A), which is a T cell mitogen, or lipopolysaccharide (LPS), which is a mouse B cell mitogen. The validity of the use of mitogens was also tested with the T6 chromosome marker method and cell surface immunoglobulin analysis. It was found that the selective responses of T and B cells to T or B cell specific mitogens were maintained in cultures containing varying numbers of T and B cells. The only indication that B cell activating or 'potentiating' factors are released by Con-A-stimulated T lymphocytes was observed in cultures with a high proportion (75%) of 'T' cells. Radio-autographic analysis revealed that under the culture conditions in which the majority of T cells from the spleen were stimulated by Con-A, only 20-30% of B cell population responded to LPS and pokeweed mitogen.

2766 SYNTHESIS OF IMMUNOGLOBULIN AND NUCLEAR PROTEIN IN SYNCHRONIZED MOUSE MYELOMA CELLS.

(E.) Liberti, P. (Dept. Biol., Massachusetts Inst. Technology, Cambridge) and C. Baglioni. *J Cell Physiol* 82(1):113-120, 1973.

The synthesis of immunoglobulin and of nuclear proteins was studied in synchronized mouse myeloma cells of the C1 line. Synchronization was obtained by double thymidine block. Protein synthesis was studied by 30 min pulses with ^{3}H-proline, lysis of cultured cells at intervals with hypotonic buffer containing the detergent Nonidet P40, and separation of nuclei and cytoplasm and of acid-soluble nuclear and cytoplasmic proteins. Immunoglobulins in the cytoplasm were determined by precipitation methods. It was found that C1 cells synthesize immunoglobulin at a relatively constant rate throughout the cell cycle except for mitosis, when a decrease in the rate of synthesis of total protein and of immunoglobulin was observed. Cell synchrony around mitosis was not sufficiently good to determine whether immunoglobulin is synthesized at all at that phase of the cell cycle. Nuclear protein and in particular histones appear to be synthesized synchronously with DNA during the S phase of the cycle. Disagreement between these results and those reported by previous investigators may have been due to differences in methods.

2767 COMPARATIVE SEROLOGICAL STUDIES ON TYPE C VIRUSES OF VARIOUS MAMMALS.

(E.) Schäfer, W. (Max-Planck-Inst. Virus Res., Tübingen, Germany), L. Pister, G. Hunsmann and V. Moennig. *Nature* [*New Biol*] 245(142):75-77, 1973.

It was previously supposed that only one type of interspecies antigenic determinant could be detected by the Ouchterlony test. The present work indicates that this is not the case. Friend- (FLV), Rauscher- (RLV) and Gross- (GLV) leukemia viruses, representative of murine virus (MuV), feline leukemia Rickard (FeLV) and feline sarcoma Gardner (FeSV), representative of feline viruses (FeV), woolly monkey sarcoma virus (SSV), pig virus (PV) and cat-derived human virus (RD114) were studied in Ouchterlony tests following treatment with Tween 80 and ether to release their constituents, centrifugation to remove undegraded products, and concentration of degraded fractions by pressure dialysis. The antisera used were those prepared against FLV, Moloney murine sarcoma virus induced rat tumors, and FeLV. From the results it was proposed that at least two different types of interspecies determinants (*a* and *b*) occur in mammalian C-viruses, which are distributed differently in the various representatives. Cat, murine, rat and hamster C-viruses seem to contain both antigens in appreciable amounts. In contrast, SSV and PV seem to have only one determinant (*b*) in detectable amounts. RD114 virus was similar to SSV and PV except that it seemed to contain in addition a non-interspecies determinant, probably of the cat virus gs-species type. The results suggest that serological attempts to demonstrate C-viruses in man may be more successful if sera with gs-interspecies antibody spectra as wide as possible are used.

2768 PRESENCE OF CELLULAR ANTIGENS IN THE SERUM OF HAMSTERS WITH TUMORS INDUCED BY S.C. INOCULATION OF SV-40 VIRUS-TRANSFORMED HAMSTER FIBROBLASTS.

(Fr.) Maziere, J.-Cl. (St. Antoine Res. Unit. Paris, France), J. P. Dandeu, M. Lux and E. Barbu. *C R Acad Sci* [*D*] *(Paris)* 276(17):2481-2484, 1973.

2769 LYMPHOCYTE TRANSFORMATION IN MALIGNANT DISEASE.

(E.) Edwards, A. J. (St. Bartholomew's Hosp., London, England) and M. R. Lee. *Lancet* 1(7813):1186-1187, 1973.

2770 CANCER AND IMMUNOSURVEILLANCE.

(E.) MacGregor, G. A. (Wood Cottage, Chilworth, Surrey, England). *Lancet* 1(7813):1185, 1973.

2771 MACROGLOBULIN PRODUCING CHRONIC LYMPHOCYTIC LEUKEMIA WITHOUT MACROGLOBULINEMIA.

(Ger.) Stein, H. (Inst. Path., U. Kiel, West Germany), E. Kaiserling, K. Lennert and M. R. Parwaresch. *Klin Wochenschr* 51(8):389-396, 1973.

2772 HEPATITIS-ASSOCIATED ANTIGEN AND BURKITT'S LYMPHOMA.

(E.) Cevik, N. (Dept. Pediatrics, Hacettepe U., Ankara, Turkey), A. Pirnar and B. Say. *Lancet* 1(7814):1257, 1973.

2773 ANTIGEN SOLUBILIZED FROM HUMAN SOLID TUMOURS: LYMPHOCYTE STIMULATION AND CUTANEOUS DELAYED HYPERSENSITIVITY.

(E.) Mavlight, G. M. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), U. Ambus, J. U. Gutterman, E. M. Hersh and C. M. McBride. *Nature* [*New Biol*] 243(127):188-190, 1973.

2774 HAPTEN-SPECIFIC IgE ANTIBODY RESPONSES IN M MICE. I. SECONDARY IgE RESPONSES IN IRRADIATED RECIPIENTS OF SYNGENEIC PRIMED SPLEEN CELLS. *(E.)* Hamaoka, T. (Harvard Med. Sch., Boston, Mass.), D. H. Katz, K. J. Bloch and B. Benacerraf. *J Exp Med* 138(1):306-311, 1973.

2775 REGULATION OF AUTOSENSITIZATION. THE IMMUNE ACTIVATION AND SPECIFIC INHIBITION OF SELF-RECOGNIZING THYMUS-DERIVED LYMPHOCYTES. *(E.)* Cohen, I. R. (Dept. Cell Biol., Weizmann Inst. Sci., Rehovot, Israel) and H. Wekerle. *J Exp Med* 137(2):224-238, 1973.

2776 BINDING OF ANTIGEN BY IMMUNOCYTES. I. EFFECTS OF LIGAND VALENCE ON BINDING AFFINITY ON MOPC 315 CELLS FOR DNP CONJUGATES. *(E.)* Bystryn, J.-C. (New York U. Sch. Med., N.Y.), G. W. Siskind and J. W. Uhr. *J Exp Med* 137(2):301-316, 1973.

2777 FATE OF ANTIGEN-BINDING IN UNRESPONSIVE AND IMMUNE MICE. *(E.)* Louis, J. (Scripps Clin., Res. Fdn., La Jolla, Calif.), J. M. Chiller and W. O. Weigle. *J Exp Med* 137(2):461-469, 1973.

2778 LEUKOCYTE LOCOMOTION AND CHEMOTAXIS. NEW METHODS FOR EVALUATION, AND DEMONSTRATION OF A CELL-DERIVED CHEMOTACTIC FACTOR. *(E.)* Zigmond, S. H. (Rockefeller U., New York, N.Y.) and J. G. Hirsch. *J Exp Med* 137(2):387-410, 1973.

2779 IDENTIFICATION OF A MAJOR BASIC PROTEIN IN GUINEA PIG EOSINOPHIL GRANULES. *(E.)* Gleich, G. J. (Mayo Fdn., Rochester, Minn.), D. A. Loegering and J. E. Maldonado. *J Exp Med* 137(6):1459-1471, 1973.

2780 CELL-MEDIATED IMMUNITY AND BLOCKING SERUM ACTIVITY TO TOLERATED ALLOGRAFTS IN RATS. *(E.)* Bansal, S. C. (Fred Hutchinson Cancer Ctr., Seattle, Wash.), K. E. Hellström, I. Hellström and H. O. Sjögren. *J Exp Med* 137(3):590-602, 1973.

2781 NORMAL IMMUNOSUPPRESSIVE PROTEIN AND IMMUNOLOGICAL SURVEILLANCE. *(E.)* Nelken, D. (Hadassah U. Hosp., Jerusalem, Israel). *Lancet* (7817):1444-1445, 1973.

2782 SALIVARY IMMUNOGLOBULINS IN PATIENTS WITH OROPHARYNGEAL AND BRONCHOPULMONARY CARCINOMA. *(E.)* Mandel, M. A. (Case Western Reserve U. Med. Sch., Cleveland, Ohio), K. Dvorak and J. J. DeCosse. *Cancer* 31(6):1408-1413, 1973.

2783 IDENTITY OF CARCINO-EMBRYONAL ANTIGEN AND ANTIGEN OF MUCUS-PRODUCING CELLS. *(E.)* Rogalsky, V. Y. (P. A. Herzen Inst. Oncology, Moscow, U.S.S.R.). *Lancet* (7815):1322-1323, 1973.

2784 MONOCLONAL IgA AND IgM IN THE SERUM OF A SINGLE PATIENT (SC). I. SHARING OF INDIVIDUALLY SPECIFIC DETERMINANTS BETWEEN IgA AND IgM. *(E.)* Yagi, Y. (Roswell Park Mem. Inst., Buffalo, N.Y.) and D. Pressman. *J Immunol* 110(2):335-349, 1973.

2785 THE CYTOTOXIC EFFECT OF MOUSE MACROPHAGES ON SYNGENEIC AND ALLOGENEIC ERYTHROCYTES. *(E.)* Melsom, H. (Norwegian Radium Hosp., Oslo, Norway) and R. Seljelid. *J Exp Med* 137(3):807-820, 1973.

2786 LIGAND-INDUCED MOVEMENT OF LYMPHOCYTE MEMBRANE MACROMOLECULES. III. RELATIONSHIP BETWEEN THE FORMATION AND FATE OF ANTI-IG-SURFACE IG COMPLEXES AND CELL METABOLISM. *(E.)* Unanue, E. R. (Harvard Med. Sch., Boston, Mass.), M. J. Karnovsky and H. D. Engers. *J Exp Med* 137(3):675-689, 1973.

2787 CELL-MEDIATED LYMPHOLYSIS. IMPORTANCE OF SEROLOGICALLY DEFINED *H-2* REGIONS. *(E.)* Alter, B. J. (Dept. Med. Genetics, U. Wisconsin, Madison), D. J. Schendel, M. L. Bach, F. H. Bach, J. Klein and J. H. Stimpfling. *J Exp Med* 137(5):1303-1309, 1973.

2788 THYMUS RECOVERY IN TUMOUR-BEARING RATS AFTER TUMOUR REMOVAL. *(E.)* Toma, V. (Dept. Animal Physiology, Babes-Bolyai U., Cluj, Romania) and G. Simu. *Oncology* 27(4):289-293, 1973.

2789 ALPHA-FETOPROTEIN AND CARCINOEMBRYONIC ANTIGEN IN A CASE OF GASTRIC CARCINOMA METASTATIC TO THE LIVER. *(E.)* Bierfeld, J. L. (U. Miami Sch. Med., Fla.), M. I. Scheiner, D. R. Schultz, M. D. Moral and A. I. Rogers. *Am J Dig Dis* 18(5):517-520, 1973.

2790 SOME BIOCHEMICAL PROPERTIES OF THYMUS LEUKEMIA ANTIGENS SOLUBILIZED FROM CELL MEMBRANES BY PAPAIN DIGESTION. *(E.)* Muramatsu, T. (A. Einstein Coll. Med., New York, N.Y.), S. G. Nathenson, E. A. Boyse and L. J. Old. *J Exp Med* 137(5):1256-1274, 1973.

2791 GENETIC CONTROL OF THE IMMUNE RESPOSE. FREQUENCY AND CHARACTERISTICS OF ANTIGEN-BINDING CELLS IN HIGH AND LOW RESPONDER MICE. *(E.)* Hämmerling, G. J. (Stanford U. Sch. Med., Calif.), T. Masuda and H. O. McDevitt. *J Exp Med* 137(5):1180-1200, 1973.

2792 ENHANCED GROWTH OF FIBROSARCOMA IN MICE TREATED WITH PHYTOHEMAGGLUTININ. *(E.)* Wagener, D. J. T. (Dept. Radiotherap., Nuclear Med., U. Nijmegen, Netherlands). *Cancer Res* 33(6):1295-1300, 1973.

2793 HYPOTHESIS: BOUND C3 AS THE SECOND SIGNAL FOR B-CELL ACTIVATION. *(E.)* Dukor, P. (Pharmaceuticals Division, Ciba-Geigy Ltd., Basle, Switzerland) and K. U. Hartmann. *Cell Immunol* 7(3): 349-356, 1973.

2794 NONSPECIFIC CYTOTOXIC EFFECTS OF ANTIGEN-TRANSFORMED LYMPHOCYTES. KINETICS, CELL-REQUIREMENTS AND THE ROLE OF RECRUITMENT. *(E.)* Butterworth, A. E. (Dept. Path., U. Cambridge, England). *Cell Immunol* 7(3):357-369, 1973.

2795 POTENTIATION AND MODULATION OF THE IMMUNE RESPONSE OF GUINEA PIGS TO POORLY IMMUNOGENIC PROTEIN-HAPTEN CONJUGATES BY PRETREATMENT WITH THE MER FRACTION OF ATTENUATED TUBERCLE BACILLI. *(E.)* Ben-Efraim, S. (Dept. Human Microbiol., Tel Aviv U., Israel), M. Constantini-Sourojon and D. Weiss. *Cell Immunol* 7(3):370-379, 1973.

2796 EFFECTS OF ANTILYMPHOCYTE SERUM ON THYMIC INDEPENDENT IMMUNITY. I. LACK OF IMMUNOSUPPRESSIVE ACTION ON THE ANTIBODY RESPONSE TO *E. COLI* LIPOPOLYSACCHARIDE. *(E.)* Barth, R. F. (U. Kansas Med. Ctr., Kansas City), O. Singla and P. Ahlers. *Cell Immunol* 7(3):380-388, 1973.

2797 INTERACTION AND RELEASE OF SOLUBLE IMMUNE COMPLEXES FROM MOUSE B LYMPHOCYTES. *(E.)* Eden, A. (New York U. Sch. Med., N.Y.), C. Bianco, B. Bogart and V. Nussenzweig. *Cell Immunol* 7(3): 474-483, 1973.

2798 MECHANISM OF BINDING OF SOLUBLE IMMUNE COMPL COMPLEXES TO LYMPHOCYTES. *(E.)* Eden, A. (New York U. Sch. Med., N.Y.), C. Bianco and V. Nussenzweig. *Cell Immunol* 7(3):459-473, 1973.

2799 INHIBITION OF ERYTHROPOIESIS IN THE SPLEENS OF IRRADIATED MICE INJECTED WITH ALLOGENIC LYMPHOCYTES. REACTIVITY OF LYMPHOCYTES AGAINST NON-H-2 TRANSPLANTATION ANTIGENS. *(E.)* Blomgren, H. (Karolinska Inst., Stockholm, Sweden) and B. Andersson. *Cell Immunol* 7(3):453-458, 1973.

2800 INFLUENCE OF DIBUTYRYL CYCLIC-AMP AND THEOPHYLLINE ON CELL SURFACE ANTIGENS ON ONCORNAVIRUS TRANSFORMED CELLS. *(E.)* Kurth, R. (Virology Division, R. Koch Inst., West Berlin, Germany) and H. Bauer. *Nature* [*New Biol*] 243(129):243-245, 1973.

2801 SPECIFIC ACTIVATION OF THE BONE MARROW-DERIVED LYMPHOCYTE BY ANTIGEN PRESENTED IN A NON-MULTIVALENT FORM. EVIDENCE FOR A TWO-SIGNAL MECHANISM OF TRIGGERING. *(E.)* Schrader, J. W. (Walter and Eliza Hall Inst. Med. Res., Melbourne, Victoria, Australia). *J Exp Med* 137(3):844-849, 1973.

2802 CLINICAL EVALUATION OF CARCINOEMBRYONIC ANTIGEN TEST. *(E.)* Meeker, W. R. Jr. (U. Kentucky, Lexington), R. Kashmiri, L. Hunter, W. Clapp and W. O. Griffen, Jr. *Arch Surg* 107(2):266-274, 1973.

2803 AN EVALUATION OF HUMORAL ANTIBODY RESPONSES IN PATIENTS WITH CARCINOMA OF THE CERVIX. *(E.)* Vos, G. H. (Natal Inst. Immunology, Durban, South Africa), M. G. Hammond, D. Vos, B. G. Grobbelaar, H. P. Auslander and G. Marescotti. *J Obstet Gynecol Br Commonw* 79:1040-1046, 1972.

2804 A PROPOSAL FOR A STANDARDIZED SYSTEM OF REPORTING HUMAN LYMPH NODE MORPHOLOGY IN RE RELATION TO IMMUNOLOGICAL FUNCTION. *(E.)* Cottier, H. (Dept. Path., U. Berne, Switzerland), J. Turk and L. Sobin. *J Clin Pathol* 26(5):317-331, 1973.

2805 TRANSFER OF RESISTANCE TO TUMOR WITH LYMPHOID CELLS FROM IMMUNIZED ALLOGENEIC DONORS. *(E.)* Jurin, M. (U. Texas, M. D. Anderson Hosp., Tumor Inst., Houston) and H. D. Suit. *Tex Rep Biol Med* 31(1):29-36, 1973.

2806 THE NATURE OF COMPLEMENT-INDUCED LESIONS IN MEMBRANES. *(E.)* Humphrey, J. H. (Natl. Inst. Med. Res., London, England). *Haematologia* 6(3):319-325, 1972.

2807 ENRICHMENT OF MEMORY CELLS CARRYING RECEPTORS FOR A PROTEIN ANTIGEN (HSA). *(E.)* Sulitzeanu, D. (Queen's U., Dept. Path., Kingston, Ontario, Canada) and M. Axelrad. *Immunology* 24(5): 803-811, 1973.

2808 A MEMBRANE ANTIGEN OF RABBIT THYMUS CELLS. *(E.)* Fradelizi, D. P. (Inst. Immunology, U. Toronto, Canada), C. T. Chou, B. Cinader and S. Dubiski. *Cell Immunol* 7(3):484-501, 1973.

2809 CELLULAR DEFENSE MECHANISMS IN PATIENTS WITH MALIGNANT TUMORS. *(Ger.)* Kohout, J. (Wilhelmine Hosp. Vienna, Austria). *Osterr Z Erforsch Bekaempf Krebskr* 27(6):365-374, 1972.

2810 ERYTHROLEUKEMIA WITH TWO ERYTHROCYTE POPULATIONS: COMBINATION OF A WEAKENED GROUP ANTIGEN, A MAJOR ADENYLATE KINASE DEFICIT, AND AN INCREASE IN HEMOGLOBIN A_2 IN ONE POPULATION. *(Fr.)* Kahn, A. (Xavier-Bichat Hosp. Hemat. Serv., Paris, France), P. Boivin, M. Wroklans and J. Hakim. *Nouv Rev Fr Hematol* 12(5):609-630, 1972.

2811 THE HISTOLOGICAL RESPONSE TO A XENOGENEIC TUMOUR IN MICE. *(E.)* Simpson, E. (Clin. Res. Ctr., Harrow, Middlesex, England) and P. C. L. Beverley. *Clin Exp Immunol* 14(3):443-455, 1973.

2812 EFFECTS OF ANTITUMOUR IMMUNITY ON PREGNANCY IN THE MOUSE. *(E.)* Parmiani, G. (Natl. Inst. Study, Cure Tumors, Milan, Italy) and G. Della Porta. *Nature* [*New Biol*] 241(105):26-28, 1973.

2813 HALO NEVI AND MELANOMA. *(E.)* Epstein, W. L. (Dept. Dermatology, Med., U. California, San Francisco), R. Sagebeil, L. Spitler, J. Wybran, W. B. Reed and M. S. Blois. *JAMA* 225(4): 373-377, 1973.

2814 THYMUS-DERIVED PROLIFERATING LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKAEMIA. *(E.)* Lille, I. (Ctr. Rene Hugenin, Saint Cloud, France), A. Desplaces, L. Meeus, R. T. Saracino and J. C. Brouet. *Lancet* (7823):263-264, 1973.

2815 DOUBLE MYELOMA. PRODUCTION OF BOTH IgG TYPE LAMBDA MYELOMA PROTEINS BY A SINGLE PLASMA CELL LINE. *(E.)* Rudders, R. A. (Med. Coll. Wisconsin, Milwaukee), V. Yakulis and P. Heller. *Am J Med* 55(2):215-221, 1973.

2816 *IN VITRO* SYNTHESIS OF DNA COMPLEMENTARY TO mRNA DERIVED FROM A LIGHT CHAIN-PRODUCING MYELOMA TUMOUR. *(E.)* Aviv, H. (Natl. Inst. Hlth., Bethesda, Md.), S. Packman, Swan, J. Ross and P. Leder. *Nature* [*New Biol*] 241(110):174-176, 1973.

2817 CELL-MEDIATED IMMUNITY TO HUMAN TUMORS. ABROGATION BY SERUM FACTORS AND NONSPECIFIC EFFECTS OF ORAL BCG THERAPY. *(E.)* Falk, R. E. (U. Toronto, Ontario, Canada), P. Mann and B. Langer. *Arch Surg* 107(2):261-265, 1973.

2818 MACROPHAGE AND NEOPLASTIC CELL INTERACTIONS DURING THE INDUCTION OF ANTINEOPLASTIC IMMUNITY. *(It.)* Bellelli, L. (Aguila Free U. Med. Sci., Italy). *Boll Soc Ital Biol Sper* 48(11):293-294, 1972.

2819 RABBIT α-FETOPROTEIN: IMMUNOLOGICAL PURIFICATION AND PARTIAL CHARACTERIZATION. *(E.)* Pihko, H. (Dept. Serology, Bacteriology, U. Helsinki, Finland), J. Lindgren and E. Ruoslahti. *Immunochemistry* 10(6):381-385, 1973.

2820 THETA ANTIGEN AND IMMUNOGLOBULIN ON A TISSUE-CULTURED MOUSE LYMPHOMA. *(E.)* Boylston, A. W. (Natl. Inst. Hlth., Bethesda, Md.). *Immunology* 24(5):851-857, 1973.

2821 THE EFFECT OF HOST AGE ON THE TRANSPLANTABILITY OF THE WALKER 256 CARCINOSARCOMA. *(E.)* Kerkvliet, N. L. (Radiation Ctr., Oregan St. U., Corvallis) and D. J. Kimeldorf. *J Gerontol* 28(3): 276-280, 1973.

2822 IMMUNOLOGICAL STUDIES ON A CASE OF RECURRENT BURKITT'S LYMPHOMA DURING IMMUNE STIMULANT TREATMENT. *(E.)* Gunvén, P. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), G. Klein, W. Henle, G. Henle, G. Rocchi, J. F. Hewetson, A. Guerra, P. Clifford, S. Singh, A. Demissie and A. Svedmyr. *Int J Cancer* 12(1):115-123, 1973.

2823 HUMAN ANTIGEN FOR THE PAUL-BUNNELL ANTIBODY. *(E.)* Lowenthal, R. M. (Clin. Res. Ctr., Harrow, Middlesex, England), S. L. McLauchlan and D. A. J. Tyrrell. *Nature* [*New Biol*] 244 (134):109-111, 1973.

2824 SPONTANEOUS REGRESSION OF METASTATIC TESTICULAR CANCER. *(E.)* Birkhead, B. M. (U. Louisville Radiation Ctr., Ky.) and R. M. Scott. *Cancer* 32(1):125-129, 1973.

2825 QUANTITATIVE STUDIES ON RNA ACCUMULATION IN HUMAN PHA-STIMULATED LYMPHOCYTES DURING BLAST TRANSFORMATION. *(E.)* Sörén, L. (Dept. Radiobiol., Res. Inst. Swedish Natl. Defence, Sundyberg) and P. Biberfeld. *Exp Cell Res* 79(2):359-367, 1973.

2826 CARCINOEMBRYONIC ANTIGEN (CEA): SEPARATION OF CEA-REACTING MOLECULES FROM TUMOR, FETAL GUT, MECONIUM AND NORMAL COLON. *(E.)* Rule, A. H. (Mount Sinai Sch. Med., City U. New York, N.Y.) and C. Goleski-Reilly. *Immunol Commun* 2(2):213-226, 1973.

2827 COMMON ANTIGENIC COMPONENT IN OVARIAN CARCINOMAS: DEMONSTRATION BY DOUBLE DIFFUSION AND IMMUNOFLUORESCENCE TECHNIQUES. *(E.)* Dorsett, B. H. (Lenox Hill Hosp., New York, N.Y.) and H. L. Ioachim. *Immunol Commun* 2(2):173-184, 1973.

2828 SURFACE IMMUNOGLOBULINS OF CIRCULATING LYMPHOCYTES IN MOUSE PLASMACYTOMA. III. THE EFFECT OF PLASMACYTOMA RNA ON THE IMMUNE RESPONSE. *(E.)* Yakulis, V. (U. Illinois Abraham Lincoln Sch. Med., Chicago, Ill.), V. Cabana, D. Giacomoni and P. Heller. *Immunol Commun* 2(2):129-139, 1973.

2829 IMMUNE RESPONSE AGAINST HAMSTER ERYTHROCYTES IN THE LOW-RESPONDER MOUSE STRAINS. VI. PASSIVE TRANSFER OF THE RESPONSIVENESS AGAINST HAMSTER ERYTHROCYTES WITH LYMPHOID CELLS. *(E.)* Nomoto, K. (Sch. Med., Kyushu U., Fukuoka, Japan), R. Makidono and K. Takeya. *Jap J Microbiol* 17(2): 99-104, 1973.

2830 THE NEPHROTIC SYNDROME, MALIGNANCY AND IMMUNOSUPPRESSION. *(E.)* Rabkin, R. (Dept. Med., U. Cape Town, South Africa), G. N. Thatcher, L. H. Diamond and L. Eales. *S Afr Med Tydskr* 47(14):605-606, 1973.

2831 THYMUS DEPENDENCE IN THE RAT OF LYMPHOCYTES RESPONDING TO STIMULATION WITH SPECIFIC ANTIGEN, ALLOGENEIC LYMPHOCYTES AND NON-SPECIFIC MITOGENS. *(E.)* Knight, S. C. (Med. Sch., U. Birmingham, England), B. Newey and N. R. Ling. *Cytobios* 25(7):35-49, 1973.

2832 THE IMMUNOLOGICAL ANALYSIS OF SOLUBLE HUMAN LEUKEMIA ASSOCIATED ANTIGENS. *(E.)* Yong, D. C. T. (Dept. Med. Microbiol., U. Manitoba, Winnipeg, Canada) and J. B. G. Kwapinski. *Can J Microbiol* 19(6):741-745, 1973.

2833 HETEROGENEITY OF THE EFFECTOR CELLS IN THE CYTOTOXIC REACTION AGAINST ALLOGENEIC LYMPHOMA CELLS. *(E.)* Forman, J. (Karolinska Inst., Stockholm, Sweden) and S. Britton. *J Exp Med* 137(2):369-386, 1973.

2834 SERUM IMMUNOGLOBULIN CONTENT IN NORMAL AND LYMPHOID TUMOR-BEARING *XENOPUS LAEVIS*. *(E.)* Hadji-Azimi, I. (Exp. Zoology Station, Geneva, Switzerland). *Cancer Res* 33(6):1177-1182, 1973.

2835 MIXED TUMOR CHALLENGE OF STRAIN SPECIFIC AND NONSPECIFIC TA3 MOUSE ASCITES MAMMARY ADENOCARCINOMA. *(E.)* Nowotny, A. (Temple U. Sch. Med., Philadelphia, Pa.) and J. Grohsman. *Int Arch Allergy* 44(3):434-440, 1973.

2836 *IN-VITRO* AND *IN-VIVO* ADHERENCE OF ALLERGISED LYMPH-NODE CELLS TO BP8 ASCITES TUMOUR CELLS, IN THE PRESENCE OF POLYAMINES AND QUARTERNARY AMMONIUM COMPOUNDS. *(E.)* Crocker, J. (Dept. Path., U. Cambridge, England) and D. B. Cater. *J Pathol* 109(4):323-334, 1973.

2837 TUMOR-SPECIFIC IMMUNITY AND THE OTOLARYNGOLOGIST. *(E.)* Neel, H. B., III (Mayo Fdn., Rochester, Minn.) and R. E. Ritts, Jr. *Ann Otol* 82(3):323-329, 1973.

2838 LYMPHOCYTE SURFACE IMMUNOGLOBULINS IN HODGKIN'S DISEASE. *(E.)* Gergely, P. (U. Debrecen, Hungary), Gy. Szegedi, E. Berenyi and Gy. Petranyi. *N Engl J Med* 289(4):220, 1973.

2839 CONCOMITANT IMMUNITY AND SPECIFIC DESENSITIZATION IN MURINE TUMOR HOSTS. *(E.)* Vaage, J. (U. Texas, Houston, M. D. Anderson Hosp., Tumor Inst.). *Isr J Med Sci* 9(3):332-343, 1973.

2840 EVIDENCE FOR TRANSFORMATION OF HUMAN B LYMPHOCYTES BY PHA. *(E.)* Phillips, B. (Middlesex Hosp. Med. Sch., London, England) and I. M. Roitt. *Nature [New Biol]* 241(112):254-256, 1973.

2841 SHEEP RED CELL BINDING TO HUMAN LYMPHOCYTES TREATED WITH NEURAMINIDASE: ENHANCEMENT OF T CELL BINDING AND IDENTIFICATION OF A SUBPOPULATION OF B CELLS. *(E.)* Bentwich, Z. (Rockefeller U., New Yrok, N.Y.), S. D. Douglas, E. Skutelsky and H. G. Kunkel. *J Exp Med* 137(6):1532-1537, 1973.

2842 CELL INTERACTIONS BETWEEN HISTOINCOMPATIBLE T AND B LYMPHOCYTES. II. FAILURE OF PHYSIOLOGIC COOPERATIVE INTERACTIONS BETWEEN T AND B LYMPHOCYTES FROM ALLOGENEIC DONOR STRAINS IN HUMORAL RESPONSE TO HAPTEN-PROTEIN CONJUGATES. *(E.)* Katz, D. H. (Harvard Med. Sch., Boston, Mass.), T. Hamaoka and B. Benacerraf. *J Exp Med* 137(6):1405-1418, 1973.

2843 THE PRODUCTION OF VESICULAR STOMATITIS VIRUS BY ANTIGEN- OR MITOGEN-STIMULATED LYMPHOCYTES AND CONTINUOUS LYMPHOBLASTOID LINES. *(E.)* Nowakowski, M. (A. Einstein Coll. Med., New York, N.Y.), J. D. Feldman, S. Kano and B. R. Bloom. *J Exp Med* 137(4):1042-1059, 1973.

2844 LYMPHOCYTE RESPONSE TO POKEWEED MITOGEN IN HODGKIN'S DISEASE. *(E.)* Cohnen, G. (Dept. Med. U. Essen, Germany), E. König, W. Augener and G. Brittinger. *Biomedicine* 19(6):239-243, 1973.

2845 PREFERENTIAL RECOMBINATION OF H AND L CHAINS OF IgG MYELOMA PROTEINS OF IDENTICAL SUBCLASSES. *(E.)* Gergely, J. (Natl. Inst. Haematology, Blood Transfusion, Budapest, Romania), G. A. Medgyesi, M. P.-C. Nagy and E. Puskás. *Nature [New Biol]* 241(107):92-93, 1973.

2846 NEUROBLASTOMA CELLS AND 14-3-2, A BRAIN SPECIFIC PROTEIN. *(E.)* Augusti-Tocco, G. (Lab. Molecular Embryology, Naples, Italy), L. Casola and A. Grasso. *Cell Differentiation* 2(3):157-161, 1973.

2847 LONG-TERM SURVIVAL OF HELA TUMOURS IN MICE TREATED WITH ANTILYMPHOCYTE SERUM. *(E.)* Franks, C. R. (Natl. Inst. Biol. Standards, Control, Holly Hill, Hampstead, London, England), K. Curtis and F. T. Perkins. *Br J Cancer* 27(5):390-395, 1973.

2848 LYMPHOCYTE TRANSFORMATION IN LARGE BOWEL CANCER. *(E.)* Lauder, I. (Dept. Path., Surg., U. Newcastle-upon-Tyne, England) and G. Bone. *Br J Cancer* 27(5):409-413, 1973.

2849 EFFECT OF BCG ON DELAYED HYPERSENSITIVITY RESPONSES OF PATIENTS WITH NEOPLASTIC DISEASE. *(E.)* Sokal, J. E. (Roswell Park Mem. Inst., Buffalo, N.Y.), C. W. Aungst and T. Han. *Int J Cancer* 12(1):242-249, 1973.

2850 ANTI-TUMOR ACTIVITY IN HUMAN SERUM: ANTIBODIES DETECTING BLOOD-GROUP-A-LIKE ANTIGEN ON THE SURFACE OF TUMOR CELLS IN CULTURE. *(E.)* Bloom, E. T. (Dept. Microbiol., Immunology, U. California, Los Angeles), J. L. Fahey, I. A. Peterson, G. Geering, M. Bernhard and G. Trempe. *Int J Cancer* 12(1):21-31, 1973.

2851 CARCINOEMBRYONIC ANTIGEN (CEA): COMPARISON OF THE FARR AND SOLID-PHASE METHODS FOR DETECTION OF CEA. *(E.)* McPherson, T. A. (Dept. Med., Immunology, Surg., U. Alberta, Edmonton, Canada), P. R. Band, M. Grace, H. A. Hyde and V. C. Patwardhan. *Int J Cancer* 12(1):42-54, 1973.

2852 INHIBITION OF LYMPHOCYTE CYTOTOXICITY FOR HUMAN COLON CARCINOMA BY TREATMENT WITH SOLUBILIZED TUMOUR MEMBRANE FRACTIONS. *(E.)* Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), M. J. Embleton and M. R. Price. *Int J Cancer* 12(1):84-92, 1973.

2853 MULTIHORMONAL ACTIVITIES OF NORMAL AND NEOPLASTIC PITUITARY CELLS AS INDICATED BY IMMUNOHISTOCHEMICAL STAINING. *(E.)* Ueda, G. (Columbia U. Coll. Physicians, Surg., New York, N. Y.), P. Moy and J. Furth. *Int J Cancer* 12(1):100-114, 1973.

2854 ANALYSIS OF CELL MEMBRANE ANTIGENS USING RADIOIODINATED, PURIFIED ANTIBODIES. II. A FACTOR IN THE SERUM OF HAMSTERS BEARING SV_{40}-INDUCED TUMORS WHICH REACTS WITH ANTIBODIES TO SV_{40}-TRANSFORMED CELL MEMBRANES. *(E.)* Kedar, E. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel), G. Spira and D. Sulitzeanu. *Isr J Med Sci* 9(3):277-283, 1973.

2855 ANALYSIS OF CELL MEMBRANE ANTIGENS USING RADIOIODINATED, PURIFIED ANTIBODIES. I. SOLUBILIZATION AND PRELIMINARY FRACTIONATION OF MEMBRANE ANTIGENS OF CELLS TRANSFORMED BY SV_{40} VIRUS. *(E.)* Kedar, E. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel), A. Aaronov and D. Sulitzeanu. *Isr J Med Sci* 9(3):266-276, 1973.

2856 THE RESPONSE TO PLANT MITOGENS OF TWO SUBPOPULATIONS OF LYMPHOCYTES IN A CASE OF CHRONIC LYMPHOCYTIC LEUKAEMIA. *(E.)* Smith, J. L. (Dept. Med., U. Cambridge, England), J. C. Cawley and C. R. Barker. *Clin Exp Immunol* 14(3):397-408, 1973.

2857 THE RELATIONSHIP BETWEEN β_2-MICROGLOBULIN AND IMMUNOGLOBULIN IN CULTURED HUMAN LYMPHOID CELL LINES. *(E.)* Hütteroth, T. H. (Cornell U. Med. Coll., New York, N.Y.), H. Cleve, S. D. Litwin and M. D. Poulik. *J Exp Med* 137(3):838-843, 1973

2858 CHARACTERIZATION OF THE SV40 T ANTIGEN. *(E.)* Del Villano, B. C. (Wistar Inst., Philadelphia, Pa.) and V. Defendi. *Virology* 51(1): 34-46, 1973.

2859 RADIOIMMUNOASSAY OF MAMMALIAN TYPE C VIRAL PROTEINS. III. DETECTION OF VIRAL ANTIGEN IN NORMAL MURINE CELLS AND TISSUES. *(E.)* Parks, W. P. (Natl. Inst. Hlth., Bethesda, Md.), D. M. Livingston, G. J. Todaro, R. E. Benveniste and E. M. Scolnick. *J Exp Med* 137(3):622-635, 1973.

2860 LECTIN-INDUCED BLAST TRANSFORMATION OF HUMAN LYMPHOCYTES. *(E.)* Reichert, C. F. (U. Michigan Med. Sch., Ann Arbor), P. M. Pan, K. P. Mathews and I. J. Goldstein. *Nature* [*New Biol*] 242 (118):146-148, 1973.

2861 MONOCLONAL HYPERGLOBULINEMIC PURPURA IN MULTIPLE MYELOMA. IgG ANTI-IgG COMPLEXES AS A PATHOGENETIC FACTOR. *(E.)* Kodama, H. (Okayama U. Med. Sch., Shikata-Cho, Japan), T. Masuda and K. Tanioku. *Jap J Dermatol* 82(2):41-46, 1972.

2862 *p*H-DEPENDENT CHANGES IN COMPOSITION OF CARCINOEMBRYONIC ANTIGEN. *(E.)* Hughes, N. R. (Prince-of-Wales Hosp., Randwick, New South Wales, Australia). *Nature* [*New Biol*] 243(5409): 523-526, 1973.

2863 PHYTOHAEMAGGLUININ-RESPONSIVE LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKEMIA. *(E.)* Pegoraro, L. (Med. Clin., U. Turin, Italy) and F. Gavosto. *Lancet* (7818):1508, 1973.

2864 A SENSITIVE IMMUNOCHEMICAL METHOD FOR THE DETERMINATION OF THE REGAN ISOENZYME IN SERUM. *(E.)* Usategui-Gomez, M. (Miles Lab., Inc., Elkhart, Ind.), F. M. Yeager and A. F. de Castro. *Cancer Res* 33(7):1574-1577, 1973.

2865 EXCRETION OF ALPHA-FOETOPROTEIN IN THE URINE OF PREGNANT RATS AND HEPATOMA-BEARING ANIMALS. *(E.)* Okon, E. (Hadassah Med. Sch., Jerusalem, Israel), E. Rosenmann, T. Dishon and J. H. Boss. *Br J Cancer* 27(5):362-369, 1973.

2866 EFFECT OF HUMAN CHORIONIC SOMATOMAMMATROPHIN AND HUMAN CHORIONIC GONADOTROPHIN ON PHYTOHAEMAGGLUTININ-INDUCED LYMPHOCYTE TRANSFORMATION. *(E.)* Contractor, S. F. (Charing Cross Hosp. Med. Sch., London, England) and H. Davies. *Nature* [*New Biol*] 243(130):284-286, 1973.

2867 THE CARCINOEMBRYONIC ANTIGENS OF HUMAN TUMOURS. *(E.)* Burtin, P. (Inst. Sci. Res., Cancer, Villejuif, France), D. Buffe and S. von Kleist. *Triangle* 11(4):123-129, 1973.

2868 IMMUNE RESPONSE IN AGED MICE. CHANGE OF CELL-MEDIATED IMMUNITY WITH AGING. *(E.)* Kishimoto, S. (Med. Sch., Osaka U., Japan), S. Shigemoto and Y. Yamamura. *Transplantation* 15(5): 455-459, 1973.

2869 INTERFERON INDUCED BY TUMORAL DNA IN A HOMOLOGOUS SYSTEM. *(E.)* Teyssie, A. R. (A. H. Roffo Inst. Oncology, Buenos Aires, Argentina), B. A. Holstein, R. R. Pirosky and S. S. Kohan. *Acta Virol* 17(2):151-154, 1973.

2870 SIZE HETEROGENEITY OF HUMAN IgA MYELOMA PROTEINS. RELATIONSHIP BETWEEN POLYMERS AND "J" CHAIN. *(E.)* Fine, J. M. (Natl. Ctr. Blood Transfusion, Paris, France), P. Lambin and D. Frommel. *Biomedicine* 18(2):145-151, 1973.

2871 A HUMAN IgA-MYELOMA PROTEIN WITH ANTI-STREPTOCOCCAL HYALURONIDASE (ASH) ACTIVITY. *(E.)* Videbaek, A. (Gentofte Hosp., Hellerup, Denmark), B. Mansa and E. Kjems. *Scand J Haematol* 10(3):181-185, 1973.

2872 THE CLINICAL SIGNIFICANCE OF LEUCOCYTE ISOANTIGENS. *(Ger.)* Mayr, W. R. (Basel Inst. Immunology, Switzerland). *Wien Klin Wochenschr* 85(21):373-378, 1973.

2873 CLINICAL ASPECTS OF TUMOR IMMUNOLOGY. *(Rus.)* Gorodilova, V. V. (P. A. Gertsen Sci. Res. Inst. Onkol. Moscow, USSR). *Vopr Onkol* 19(3):9-17, 1973.

2874 PRIMARY ANTIBODY FORMATION IN PATIENTS WITH MALIGNANT DISEASES OF THE LYMPHORETICULAR SYSTEM AND WITH METASTASIZING TUMORS. *(Ger.)* Krull, P. (Clin., Med. Highsch., Hannover, West Germany) and H. Deicher. *Z Immunol Forsch* 145(1):70-77, 1973.

2875 TIME RELATIONSHIPS BETWEEN INJECTION OF ANTIGEN AND ADJUVANT. IV. STUDIES ON ANTIGENIC COMPETITION WITH SPECIAL REFERENCE TO THE ROLE OF BACTERIAL ADJUVANTS. *(E.)* Finger, H. (Inst. Hygiene, Microbiol., U. Würzburg, West Germany) and P. Emmerling. *Z Immunol Forsch* 145(1):78-94, 1973.

2876 RELATIONSHIP OF BCG-INDUCED PULMONARY DELAYED HYPERSENSITIVITY TO ACCELERATED GRANULOMA FORMATION IN RABBIT LUNGS: EFFECT OF CORTISONE ACETATE. *(E.)* Moore, V. L. (Bowman Gray Sch. Med., Wake Forest U., Winston-Salem, N.C.) and Q. N. Myrvik. *Infect Immun* 7(5):764-770, 1973.

2877 ADVANCES IN IMMUNOPATHOLOGY. *(E.)* Anonymous. *Nature* [*New Biol*] 243(129):225-226, 1973.

2878 IMMUNOLOGY OF THE CANCER PATIENT. *(E.)* Anonymous. *Med J Aust* 1(13):622-624, 1973.

2879 A STUDY OF SPECIES-SPECIFIC AND SPECIFIC TUMOR ANTIGENS IN CELLS OF INTERSPECIFIC SOMATIC HYBRIDS. *(E.)* Klyuchareva, T. E. (Acad. Med. Sci. USSR, Moscow), V. A. Mateeva and G. I. Deichman. *Bull Exp Biol Med* 74(11):1420-1422, 1973.

2880 THE QUANTITATIVE FEATURES OF IMMUNOGLOBULIN G, A, AND M IN CHILDREN WITH PARABLASTIC LEUKEMIA AFTER BCG VACCINATION. *(Ger.)* Blau, H.-J. (Childrens' Clin. U. Rostock, E. Germany) and H. Kruse. *Folia Haematol (Leipz)* 99(2/3):152-156, 1973.

2881 DEMONSTRATION OF COMMON SARCOMA-ASSOCIATED ANTIGEN(S) IN AN ESTABLISHED HUMAN NEUROGENIC SARCOMA CELL LINE. *(E.)* Drewinko, B. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), C. T. Montgomery and J. M. Trujillo. *Cancer Res* 33(3):601-605, 1973.

2882 INFLUENCE OF TUMOR ANTIGEN ON MAINTENANCE *VERSUS* DEPRESSION OF TUMOR-SPECIFIC IMMUNITY. *(E.)* Vaage, J. (Massachusetts Gen. Hosp., Boston) *Cancer Res* 33(3):493-503, 1973.

2883 SUPPRESSION OF AUTOCHTHONOUS GRAFTS OF SPONTANEOUS MAMMARY TUMOR BY INDUCED ALLOGENEIC GRAFT REJECTION MECHANISM. *(E.)* Tokuzen, R. (Natl. Cancer Ctr. Res. Inst., Tokyo, Japan) and W. Nakahara. *Cancer Res* 33(4):645-647, 1973.

2884 FACTORS INFLUENCING THE ASSAY FOR TARGET CELLS OF FRIEND SPLEEN FOCUS-FORMING VIRUS. *(E.)* Thomson, S. (Dept. Microbiol., Mahidol U., Bangkok, Thailand) and P. Srivatanakul. *Cancer Res* 33(4):717-720, 1973.

2885 *HL-A* ANTIGENS IN SOLID TUMORS. *(E.)* Takasugi, M. (Dept. Surg., U. California, Los Angeles), P. I. Terasaki. B. Henderson, M. R. Mickey, H. Menck and R. Thompson. *Cancer Res* 33(4):648-650, 1973.

2886 NEURAMINIDASE INDUCED LOSS IN THE TRANSPLANTABILITY OF MURINE LEUKAEMIA L 1210, INDUCTION OF IMMUNOPROTECTION AND THE TRANSFER OF INDUCED IMMUNITY TO NORMAL DBA/2 MICE BY SERUM AND PERITONEAL CELLS. *(E.)* Sethi, K. K. (Inst. Microbiol., Immunol., U. Bonn, West Germany) and H. Brandis. *Br J Cancer* 27(2):106-113, 1973.

2887 IMMUNITY IN THE ACUTE LEUKAEMIAS. *(E.)* Mitchell, M. S. (Yale U., Sch. Med., New Haven, Conn.). *Lancet* 1(7813):1192, 1973.

2888 HUMORAL MEDIATORS OF DELAYED CELLULAR IMMUNITY. *(Fr.)* Dryll, A. (Lariboisiere Clin. Rheumatol. Paris, France) and S. De Seze. *Sem Hop Paris* 48(42):2717-2724, 1972.

2889 MACROCOLONY ASSAYS IN THE RAT OF ALLOGENEIC Y-P388 AND W-256 TUMOUR CELLS INJECTED INTRAVENOUSLY: DEPENDENCE OF COLONY FORMING EFFICIENCY ON AGE OF HOST AND IMMUNITY. *(E.)* Van den Brenk, H. A. S. (St. Thomas' Hosp., London, England), C. Sharpington and C. Orton. *Br J Cancer* 27(2):134-152, 1973.

2890 THE PRESENCE OF TUMOUR-SPECIFIC MEMBRANE ANTIGEN IN THE SERUM OF RATS WITH CHEMICALLY INDUCED SARCOMATA. *(E.)* Thomson, D. M. P. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England), K. Steele and P. Alexander. *Br J Cancer* 27(1):27-34, 1973.

See also:

* (Rev): 2401, 2409, 2414, 2420, 2421, 2425, 2426, 2430, 2435, 2444, 2445, 2478
* (Chem): 2515, 2518, 2522, 2527, 2531,
* (Viral): 2627, 2628, 2637, 2662, 2666, 2673, 2674

2891 SOME BIOCHEMICAL AND HISTOCHEMICAL ASPECTS IN HYPERPLASIA AND CARCINOMA OF THE HUMAN PROSTATE.

(E.) Nilsson, T. (Div. Urol., State U. New York, Buffalo) and J. Müntzing. *J Surg Oncol* 5(3):205-214, 1973.

A study of histochemical and biochemical enzyme activity determination in hyperplastic and untreated carcinomatous prostatic tissue is presented. The histochemical results are in agreement with earlier investigations and the biochemical findings corroborate grossly the histochemical findings. A comparison of conventional estrogen therapy to Estramustine phosphate, (Estracyt^R), therapy is discussed from both the histochemical and biochemical points of view. The enzyme activity quotient, acid phosphatase to β-glucuronidase, is pointed out as a possible indicator in following the therapeutic response, and in determination of the origin of metastases. Lipofuscin pigment is demonstrated to a much higher frequency after Estracyt treatment than after conventional estrogen. This indicates that this substance might have an intracellular effect which is different and more pronounced than that of estrogen.

2892 FELINE LEUKEMIA AFTER FELINE INFECTIOUS ANEMIA.

(E.) Priester, W. A. (Natl. Cancer Inst., Bethesda, Md.) and H. M. Hayes. *J Natl Cancer Inst* 51(1):289-291, 1973.

Severe induced anemia can act as a triggering mechanism in the experimental induction of leukemia in irradiated rats and leukemia-prone mice. A case-control prospective study of 297 cats with feline infectious anemia (FIA) was conducted to determine if this phenomenon applies to other species under more natural conditions. Six developed feline leukemia (FeL) as compared with 0.5 expected ($P<0.005$) on the basis of control data. A relationship between the diseases apparently existed but causality could not be implied because of the short periods between diagnoses of the two conditions: prodromal FeL may have activated a latent FIA infection or FIA may have triggered FeL. If the order of association is FIA followed by FeL, anemia produced by FIA may enhance activity of the reticuloendothelial tissue, which in turn favors development of FeL.

2893 PHILADELPHIA CHROMOSOME-NEGATIVE, LYSOZYME-POSITIVE CHRONIC MYELOID LEUKEMIA.

(E.) Mintz, U. (Tel-Aviv U. Med. Sch., Israel), J. Pinkhas, A. I. Pick and A. De Vries. *Haematologia (Budap)* 7(1):3-6, 1973.

Previous investigators raised the possibility that at least two types of chronic myeloid leukemia (CML) exist. One type is the most common form with normal or increased platelet count, positive Philadelphia chromosome, no lysozyme activity in the serum or in the urine, good response to busulfan, and an average survival of about 3 1/2 yr. The second type is characterized by the presence of increased lysozyme activity in both the serum and the urine, absence of Philadelphia chromosome, low platelet count, poor response to busulfan and other chemotherapeutic agents, and a short life-span. The present investigators describe two patients corresponding to the latter type. The authors also discuss the possibility, suggested by previous investigators, that lysozymuria might appear when the renal tubular threshold is exceeded due to increased serum lysozyme levels, or when there is tubular damage; their own work supports these explanations. The high serum lysozyme may be due to excessive production by the leukemic white blood cells.

2894 THE BLASTIC CRISIS OF CHRONIC MYELOID LEUKAEMIA: ACUTE TRANSFORMATION OF A PRE-LEUKAEMIC CONDITION?

(E.) Pedersen, B. (Cancer Res. Inst., Aarhus, Denmark). *Br J Haematol* 25(2): 141-145, 1973.

Supporting the suggestion previously made by Killmann, the author proposes that the chronic phase of chronic myeloid leukemia (CML) is a preleukemic condition which has a very high *a priori* probability of turning into acute myeloid leukemia (AML) by way of a blastic crisis. Myeloblasts observed in the chronic phase generally contain no periodic acid-Schiff (PAS) positive substances, no lipids staining with Sudan black B, and no peroxidase activity, while those observed in the blastic crisis may be PAS, Sudan black B and peroxidase positive. Further, blastic crisis is chracterized by karyotype abnormalities, high frequencies of primitive forms of granulocyte precursors, and prolonged generation times of primitive granulocyte precursors. However, signs of changes from the normal appearing cells of the chronic phase to the abnormal cells of the blastic phase begin to appear in the chronic phase before clinical and hematological signs of deterioration develop. If karyotype studies were performed when the patients show signs of decreased sensitivity to drugs and more than sporadic aneuploidy, a radical elimination of the abnormal cells might then be obtained by administration of intensive treatment, resulting in improved life expectancy.

2895 THE RELATIONSHIP OF EMBOLIC HOMOGENEITY, NUMBER, SIZE AND VIABILITY TO THE INCIDENCE OF EXPERIMENTAL METASTASIS.

(E.) Fidler, I. J. (Sch. Dental Med., U. Pennsylvania, Phila.). *Eur J Cancer* 9(3):223-227, 1973.

B_{16} melanoma cells grown *in vitro* were used to study factors influencing experimental metastasis. Groups of C57 BL/6_J mice were given i.v. injections of varying numbers of viable B_{16} melanoma cells, with or without gamma-irradiated B_{16} cells; viable B_{16} cells with or without C_{57} embryo cells; and equal number of viable B_{16} cells suspended singly or in clumps. The data permitted the following conclusions: The number of lung metastases was proportional to the number of viable tumor cells injected i.v. Mice injected with clumps of B_{16} tumor cells had a higher number of lung metastases when compared to mice injected with an equal number of single B_{16} cells. Mice injected with viable B_{16} tumor cells together with dead B_{16} cells or viable C_{57} embryo cells had a statistically significant higher number of pulmonary tumors as compared to mice injected with viable B_{16} cells alone. The mechanism responsible for these phenomena might be the induction of pulmonary vasospasm by arrested emboli bringing about an increased trapping of tumor cells in the lung capillaries. These results point out the absolute necessity for a standardized and homogenous system for studies of metastasis. Embolic homogeneity, viability number and size must be considered to insure accurate and reproducible results. In addition, they call attention to the possible role of circulating dead tumor cells in the formation of secondary tumors.

2896 AN ULTRASTRUCTURAL STUDY OF THE NATURE AND ORIGIN OF THE BRENNER TUMOUR OF THE OVARY. *(E.)* Cummins, P. A. (Dept. Obstet. Gynaecol., U. Manchester, England), H. Fox and F. A. Langley. *J Pathol* 110(2):167-176, 1973.

The ultrastructure of the Brenner ovarian tumor was compared with that of various structures from which it has been thought to be derived or with which its epithelial component is thought to be homologous. These were normal bladder epithelium, a transitional-cell papilloma of the bladder, Walthard rests from the surface of normal fallopian tubes, ovarian surface epithelium, and an ovarian follicular cyst. The tissues were fixed in ice-cold cacodylate buffered glutaraldehyde, post-fixed in buffered osmium tetroxide, dehydrated with alcohol, and embedded in Araldite resin at 60 C. Ultrathin sections were mounted on naked copper grids, stained with uranyl acetate and lead acetate, and examined with an electron microscope. It was concluded that the epithelial cells of the Brenner tumor have a number of ultrastructural characteristics in common with those of most of the other tissues studied, but that they correspond most closely to uro-epithelial cells. The results suggest that Brenner tumors are derived either from the surface epithelium of the ovary by uro-epithelial metaplasia or from structures of urogenital ridge origin in the ovarian hilum.

2897 NASOPHARYNGEAL CARCINOMA. III. ULTRASTRUCTURE OF DIFFERENT GROWTHS LEADING TO LYMPHOBLASTOIC TRANSFORMATION *IN VITRO*. *(E.)* Vuillame, M. (Internatl. Agency Res. Cancer, Lyon, France) and G. de-The. *J Natl Cancer Inst* 51(1): 67-80, 1973.

The successive growth in culture of nasopharyngeal carcinoma (NPC) biopsy specimens was investigated by electron microscopy until lymphoblastoid transformation occurred. During early lymphocyte production, the cell population was heterogeneous, revealing some alterations of the nuclear membrane but no evidence of herpesvirus infection. In the Spongostan specimens, epithelial growth exhibiting fibroblastoid transformation was maintained up to two months. Lymphoid elements and both epithelial and fibroblastoid cells were regularly in close contact; and ultrastructural differences were observed between fibroblasts growing in Spongostan and those growing on the bottom of the tissue culture flasks. Whereas no fingerprint of herpesvirus infection was evident in the different growths before lymphoblastoid transformation, important nuclear alterations of fibroblasts in contact with lymphoblastoid cells were observed. The lymphoblastoid transformation could result from the stimulation of a few lymphocytes with the Epstein-Barr virus genome present but inactive in NPC cultures. Stimulation of lymphoblastoid transformation could be triggered by close contacts with epithelial or fibroblastoid cells and the possible exchange of cytoplasmic material between these lymphocytes and other cells (epithelial or fibroblastoid).

2898 ON THE MALIGNANT TRANSFORMATION OF CELLS DURING PROLONGED CULTURE UNDER HYPOXIC CONDITIONS *IN VITRO*. *(E.)* Goldblatt, H. (Mt. Sinai Hosp., Cleveland, Ohio), L. Friedman and R. L. Cechner. *Biochem Med* 7(2):241-252, 1973.

Four independent series of mixed cultures of epithelial cells and fibroblasts, from the skin (mainly epidermis) of Sprague-Dawley-derived Holtzmann strain rat embryos, grown under a layer of fluid medium in stationary T-60 flasks, with or without free access of ambient air to the surface of the medium, eventually yielded malignant tumors, when the cultures were injected into 4-5 wk old female rats of the same strain. These cultures were composed of both types of cells, and proved transplantable when injected s.c. or i.p., even into untreated rats of the same strain as the embryos. Exposure of the cells to a high concentration of readily available oxygen in the medium resulted in at least a delay, and probably the complete prevention, of the malignant transformation of the cells. Observations of the malignant transformation of cells grown for prolonged periods *in vitro* have become commonplace, but the cause of the phenomenon has remained obscure. Neither a virus nor any known physical or chemical carcinogenic agent has been proved to be the cause. The transformation of the cells was not reversed during 13 months of exposure of the cultures of malignant cells to a high concentration of oxygen in the medium. The experimental results of this investigation, as well as the theoretical considerations, suggest that intracellular hypoxia, due to the inadequate rate of diffusion of the oxygen in the ambient air through the stationary layer of liquid medium, acted at least as the initiating agent, with unknown (intrinsic or extrinsic) factors possible acting as promotors.

2899 COMPARATIVE MICROSPECTROPHOTOMETRIC STUDY OF THE DNA CONTENT IN THE DIAGNOSIS OF PRE-TUMOROUS PROCESSES AND CANCER. *(E.)* Avtandilov, G. G. (USSR Acad. Med. Sci., Moscow) and I. A. Kazantseva. *Virchows Arch* [*Pathol Anat*] 359(4): 289-297, 1973.

A comparative microspectrophotometric study of the nuclear Feulgen DNA content of various types of epithelial tissue (multi-layer flat epithelium of the larynx, glandular epithelium of the large intestine and endometrium) in different forms of dysplasia, hyperplasia and neoplastic growth was made, using histological sections of biopsy material (66 biopsies from 62 patients). The study showed that different forms of neoplastic transformation of epithelial tissues are marked by definite shifts in the nuclear Feulgen DNA content and revealed different levels of their cellular polyploidy and genetic heterogeneity. These levels of polyploidy attest to the beginning of neoplastic transformation and can be used as an objective diagnostic test. Transitional dysplastic and hyperplastic conditions of the epithelium, characterized by an increased degree of heterogeneity of cell populations and increased polyploidy, represent a much greater probability of progression of neoplastic transformation.

2900 GYNECOMASTIA. ENZYME-HISTOCHEMICAL AND HISTOLOGICAL INVESTIGATIONS WITH A CORRELATION OF ENZYME ACTIVITIES IN GYNECOMASTIA AND FIBROADENOMATOSIS. *(E.)* Jensen, H. (U. Pathol. Anat., Rigshospitalet, Copenhagen, Denmark). *Acta Pathol Microbiol Scand* [A] 81(8):543-551, 1973.

Histochemical reactions for NADH-tetrazolium reductase, NADPH-tetrazolium reductase, lactic acid dehydrogenase, succinic acid dehydrogenase, glucose-6-phosphate dehydrogenase, α-glycerophosphate dehydrogenase, non-specific alkaline phosphates and non-specific acid phosphatase were investigated on biopsy specimens from 20 cases of gynecomastia and compared with similar tests carried out on material obtained from cases of fibroadenomatosis of women. Identical results were obtained in the dehydrogenase reactions for the two diseases apart from that for glucose-6-phosphate dehydrogenase which revealed localized enzyme activity in the proliferating epithelium of the ducts in fibroadenomatosis, which contrasted with the negative findings in gynecomastia. The activity of the non-specific alkaline phosphatases was found to be increased in the myoepithelial cells in gynecomastia. This is presumably due to hormonal influence. The pathological changes and the etiological background are discussed.

2901 THE KARYOTYPE EVOLUTION IN CHRONIC GRANULOCYTIC LEUKAEMIA. I. THE CHROMOSOMES GAINED AND LOST DURING INITIATION OF THE EVOLUTION. *(E.)* Pedersen, B. (Cancer Res. Inst., Aarhus C, Denmark). *Eur J Cancer* 9(7):503-507, 1973.

Cytogenetically, the acute transformation of chronic granulocytic leukemia (CGL) is characterized by an evolutionary process leading to the establishment of a number of abnormal, mostly hyperdiploid Ph^{1}-positive hemopoietic clones. To determine whether the types of chromosomes gained and lost during the initial steps of the CGL evolution differed from those observed with normal cells, karyotype variants showing gain and/or loss of one chromosome were investigated in material consisting of 3633 Ph^{1}-positive metaphases from 48 patients with chronic myelogenous leukemia. The frequencies of missing as well as super-numerary chromosomes were found to be inversely related to the average chromosomal size in the various groups, a relation very similar to that of missing chromosomes observed in studies on normal cells. Further, each morphological chromosome group contributed much the same to chromosome gain as to chromosome loss. Together, these observations suggest that the mitotic aberrations responsible for simple abnormal leukemic karyotype variants are the same as those operating in normal cells, and, in addition, in leukemic cells the same mitotic aberrations appear to cause both chromosome loss and gain. Further, the observations may suggest that karyotype evolution is caused by defective elimination rather than excessive production of abnormal variants.

2902 THE KARYOTYPE EVOLUTION IN CHRONIC GRANULOCYTIC LEUKAEMIA. II. THE CHROMOSOME AND KARYOTYPE PATTERN OF ADVANCED EVOLUTION. *(E.)* Pedersen, B. (Cancer Res. Inst., Aarhus C. Denmark). *Eur J Cancer* 9(7):509-513, 1973.

While initial steps of karyotype evolution in chronic granulocytic leukemia (CGL) involve gain and loss of members of a variety of chromosome groups, later stages involve gain and loss of specific chromosome categories. To determine whether a few cytogenetic features in advanced, abnormal karyotypes might be found which represent conditions *sine qua non* for prolonged clonal survival in leukemic hemopoietic tissues, trends of karyotype evolution were investigated in 3633 karyotyped Ph^{1}-positive metaphases obtained from 48 patients with chronic granulocytic leukemia. In agreement with observations by others, the majority of the chromosomes gained during progressive evolution were observed to be Ph^{1}, F and, in particular, C members and most of those lost to be E(17-18) chromosomes. The vast majority of the hyperdiploid karyotypes fell in two distinct groups: one showing excess numbers of C chromosomes with or without supernumerary chromosomes (C variants) and another presenting the same cytogenetic characters except for presence of a second Ph^{1} (Ph^{1}C variants). With progressive evolution the Ph^{1}C variants covered increasing proportions of the variant population whereas the C variants decreased. Apparently, during evolutionary progress, gain of a second Ph^{1} confers selective advantage.

2903 PRELEUKEMIA. *(Ger.)* Heimpel, H. (Ulm U. Ctr. Int. Med., Germany). *Wein Klin Wochenschr* 85(21):378-381, 1973.

2904 ELECTRON MICROSCOPIC STUDIES OF THE HUMAN LARYNX IN PRECANCEROUS AND CANCEROUS CONDITIONS. *(Rus.)* Bordzhardze, A. K. (Georgian SSR Acad. Sci. Inst. Exp. Morphol., Tiflis, USSR), U. A. Gabuniya and Z. G. Tsagareli. *Soobshcheniya Akad Naur Gruz SSR* 69(3):717-720, 1973.

2905 ON PRETUMOR CHANGES IN THE ZIMBAL GLANDS IN RATS. *(Rus.)* Pliss, G. B. (USSR Ministry of Public Health) and N. I. Voljfson. *Vopr Onkol* 18(10):61-66, 1972.

2906 PRELEUKEMIA. THE HEMATOLOGIC SYNDROME PRECEDING ACUTE LEUKEMIA. *(E.)* Saarni, M. I. (Hatanpää Hosp., Tampere, Finland) and J. W. Linman. *Am J Med* 55(1):38-48, 1973.

2907 THE PRECANCEROUS STATE OF THE STOMACH. *(E.)* Elster, K. (City Hlth. Inst., Bayreuth, West Germany). *Acta Hepato Gastroenterol* 20(1):49-51, 1973.

2908 PROLIFERATION KINETICS OF NORMAL AND LEUKEMIC HEMOPOIESIS IN ACUTE LEUKEMIA. *(Ger.)* Hoelzer, D. (Ulm U. Clin. Res. Ctr., W. Germany) and T. M. Fliedner. *Wien Klin Wochenschr* 85(26):470-473, 1973.

2909 MALIGNANT TUMOR-LIKE RETICULOSIS DEVELOPED ON A BACKGROUND OF CHRONIC MYELOID LEUKEMIA. *(Rus.)* Petrov, H. S. (S. M. Kirov Military Med. Sci. Acad., USSR), V. P. Dygin and Y. I. Mazurov. *Vopr Onkol* 18(10):83-86, 1972.

2910 THE KINETICS OF PRECANCEROUS CONDITIONS AND CANCER OF THE UTERINE CERVIX. *(Pol.)* Kwoczynski, M. Z. (Inst. Obset. Gynecol., Wroclaw, Poland). *Pol Tyg Lek* 27(42):1652-1655, 1972.

2911 EARLY FINDINGS IN LEUKEMIC DISEASES. *(Ger.)* Huber, H. (Med. Clin. U. Innsbruck, Austria) and G. Michlmayr. *Dtsch Med Wochenschr* 98(18):945-947, 1973.

2912 PLASMA GONADOTROPIN LEVELS DURING EARLY STAGES OF OVARIAN TUMORIGENESIS IN MICE OF THE W^{X}/W^{v} GENOTYPE. *(E.)* Murphy, E. D. (Jackson Lab., Bar Harbor, Me.) and W. G. Beamer. *Cancer Res* 33(4):721-723, 1973.

2913 PATHOGENESIS OF HODGKIN'S DISEASE: SEPARATION AND CULTURE OF DIFFERENT KINDS OF CELLS FROM HODGKIN'S DISEASE IN A STERILE ISOKINETIC GRADIENT OF FICOLL IN TISSUE CULTURE MEDUIM. *(E.)* Pretlow, T. G. (Dept. Path., U. Alabama, Birmingham), D. E. Luberoff, L. J. Hamilton, P. C. Weinberger, W. A. Maddox and J. R. Durant. *Cancer* 31(5):1120-1126, 1973.

2914 DIVERTICULOSIS AND POLYPS OF THE LARGE INTESTINE. A NECROPSY STUDY OF HAWAII JAPANESE. *(E.)* Stemmermann, G. N. (Kuakini Hosp., Honolulu, Hawaii) and R. Yatani. *Cancer* 31(5):1260-1270, 1973.

See also:

* (Rev): 2448
* (Chem): 2480, 2533, 2538, 2577
* (Phys): 2602

2915 ASBESTOS AND MESOTHELIOMA IN THE NETHERLANDS. *(E.)* Planteydt, H. T. (Regional Lab. "Zeeland", Middelburg, Netherlands). *TNO Nieuws* 27(11):667-671, 1972.

The frequency and geographical distribution of mesothelioma in the Netherlands has been determined based on 140 cases of histologically diagnosed pleural and peritoneal tumors reported to the Mesothelioma Register from 1969 through 1971. Of the cases reported, 121 were in males and 19 were in females. The highest frequencies of mesothelioma were noted in the areas near harbors and shipyards, especially in the large cities. A detailed history obtained for 30 cases from the Rotterdam area revealed certain or probable professional exposure to asbestos in nearly all instances. Electron microscopic observation of 6μ section of lung tissue showed the presence of chrysotile asbestos in lungs of all of 28 mesothelioma patients tested and also in lungs of all of 10 controls. However, amphiboles were present in lungs of 18 of the mesothelioma patients but in only one of the controls. The findings were confirmed in all essential aspects by tests using 30μ sections stained for iron and also by tests in which asbestos was revealed by treatment of lung tissue with KOH.

2916 MALIGNANT MELANOMA OF THE SKIN IN CALIFORNIA, 1942-1969: EPIDEMIOLOGIC AND SURVIVAL CONSIDERATIONS. *(E.)* Krain, L. S. (Med. Ctr. U. California, Los Angeles). *Cutis* 11(3):350-357, 1973.

Factors said to influence the occurrence of malignant melanoma of the skin are trauma to normal skin and to pre-existing moles, exposure to sunlight, local heat, racial and phenotypic constitution, hormones, genes and occupations. In California, the prevalence of malignant melanoma of the skin increased about 135% between the period 1942 to 1954 and the period 1955 to 1969, and the incidence increased from 3.6 to 4.7/100,000 population from 1960 to 1969. The mortality rate rose in spite of improved 5 yr relative survival rates. The increase of the tumor was greater in southern hospitals, due, possibly, to increased sunlight and common failure to wear shoes. The tumor became increasingly prevalent in the 25 to 54 yr group, despite general aging of the population. Females had a greater incidence until menopause, when males had a greater incidence, but at age 70 and over, females again had a greater incidence. Whites had higher incidence than non-whites, apparently due to a protective effect of melanin against UV light. The disease was more common in private hospitals than county hospitals, indicating a socioeconomic differentiation. Improved survival rates were attributed to the fact that a greater percentage of cases were localized at the time of initial diagnosis and a greater percentage underwent surgery as the initial course of treatment. The survival rate was 67% for all stages of disease and 82% for localized cases in the 1955 to 1969 period. Better survival among women than men may be related to concern with cosmetic appearance, prompting earlier attention and surgery. The author suggests, in line with the Huebner-Todaro hypothesis, that trauma, sunlight, hormonal and local heat factors may interact with the genetic phenotype and allow a latent virus to become "switched on" and a malignant melanoma to develop in a pre-existing nevus or in normal skin.

2917 SEROEPIDEMIOLOGIC STUDIES OF HERPESVIRUS TYPE 2 AND CARCINOMA OF THE CERVIX. IV. DYSPLASIA AND CARCINOMA *IN SITU*. *(E.)* Adam, E. (Baylor Coll. Med., Houston, Tex.), R. H. Kaufman, J. L. Melnick, A. H. Levy and W. E. Rawls. *Am J Epidemiol* 98(2):77-87, 1973.

A study was undertaken to investigate in more detail than heretofore the distribution of antibodies to herpesvirus types 1 and 2 among 62 cases of carcinoma *in situ*, 137 cases of dysplasia, and 290 control women. Included in part of the study were 60 cases of invasive carcinoma from a previous investigation. Certain factors which appear to be measures of sexual promiscuousness, namely, age at first intercourse, age at first pregnancy, number of marriages, number of sex partners, and number of live births, also were evaluated, as well as race, age, and social level. The data were handled by direct comparisons of mean titers and by use of a threshold value for a II/I index, defined as the ratio of the titer to type 2 virus to the titer to type 1 virus, multiplied by 100. Direct comparisons of mean titers showed that titers were not significantly different between women with dysplasia and control women of comparable ages. For women 39 yr old or younger, the titers were significantly higher among cases of carcinoma *in situ* than among control women of the same age group; for older women, the titers did not differ between cases and controls. The different sexual factors showed no significant effect on the results, suggesting that the greater occurrence of antibodies among women with cervical lesions is not the result of greater promiscuity than among the controls. Threshold values for the II/I index showed that herpesvirus type 2 antibodies occurred at a frequency of 52% in cases of invasive carcinoma, 39% in cases of carcinoma *in situ*, 31% in cases of dysplasia, and 28% in controls. Statistically, differences between women with dysplasia, carcinoma *in situ*, and invasive cancer were not significantly different. However, the women with the various lesions did differ significantly from the controls, except for the carcinoma *in situ* group where the difference, as mentioned above, was significant only for the subgroups less than 40 yr of age. The observations are compatible with a virus infection initiating or promoting an oncogenic process which may lead to neoplastic changes of the epithelium; the changes may or may not subsequently develop into invasive cancer.

2918 AGE, TIME AND COHORT FACTORS IN MORTALITY FROM CANCER OF THE CERVIX. *(E.)* Barrett, J. C. (London Sch. Hygiene Tropical Med., England). *J Hyg (Camb)* 71(2):253-259, 1973.

Data for mortality from cancer of the cervix in England and Wales by 5-yr age groups and four quinquennia (1951-70) are analysed. The logarithms of the mortality rates are regressed on age group, epoch of death and epoch of birth. The factors obtained are considered in relation to particular features of the mortality pattern. The incidence of mortality rises steeply with increasing age up to an age between 60 and 65 yr and then falls very slightly. Approximately 90% of deaths from cancer of the cervix occur within 5 yr of the diagnosis.

2919 BIOLOGICAL EFFECTS OF ASBESTOS. REPORT OF THE ADVISORY COMMITTEE ON ASBESTOS CANCERS TO THE DIRECTOR OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER. *(E.)* IARC Advisory Committee (WHO Int. Agency Res. Cancer, Lyon, France). *Ann Occup Hyg* 16(1):9-17, 1973.

A Committee of three panels -- Epidemiology, Pathology, and Physics and Chemistry reported on evidence linking asbestos dust exposure to cancers and then made recommendations for further research and priorities. Evidence of a causal relationship has been increased by studies showing exposure-response relations for incidence. Risk depends on type of fiber and nature of exposure. Risk is greatest with crocidolite, less with amosite, apparently less with chrysotile, and absent with anthophyllite. There is evidence of an association of mesothelial tumors with air pollution in the neighborhood of crocidolite mines and of factories using mixtures of asbestos fiber types. There is no evidence of risk to the general public at present. Cigarette smoking enhances lung carcinoma risk in asbestos-exposed workers. Waxes, oils and trace elements are unlikely contributory factors. There is a relatively small excess risk for cancers of the gastrointestinal tract, none for ovarian tumors. Pleural plaques have been associated with past exposure to all commercial types of asbestos, but additional factors, other than asbestos itself, are involved. Among many recommendations for further research, the Epidemiology Panel listed the assessment of excess cancer risks following exposure to only one type of fiber, and studies of the amount and type of asbestos in lungs of mesothelioma cases; the Pathology Panel recommended an investigation of whether reduction of asbestos exposure to levels below those producing asbestosis also abolishes excess risk of carcinoma, and a program to ensure uniformity of diagnostic criteria for different histological types of diffuse mesotheliomas; the Physics and Chemistry Panel stressed the urgent need for quantitative assessment methods, size analysis and characterization of particles and fibers in lungs and other organs, together with improved methods for dispensing and collecting dusts for inhalation studies.

2920 ENVIRONMENTAL EXPOSURE AND BLADDER CANCER IN YOUNG MALES. *(E.)* Benton, B. (Dept. Pathol., U. Southern California, Los Angeles) and B. E. Henderson. *J Natl Cancer Inst* 51(1):269-270, 1973.

The occupational histories of nine young males with transitional cell carcinoma of the bladder were examined. Of these patients, six had occupational and environmental histories of unusual exposure to paints, solvents, and chemicals although none of these cases can be correlated with a particular carcinogen. One patient developed cancer of the same site and cell type as his father whose work included 20 years of welding and metal work. It is likely that the son's cancer reflected an environmental exposure factor acting on a genetically susceptible individual.

2921 MULTIPLE CASES OF FELINE LEUKEMIA AND FELINE INFECTIOUS PERITONITIS IN A HOUSEHOLD. *(E.)* Cotter, S. M. (Angell Mem. Animal Hosp., Boston, Mass.), C. E. Gilmore and C. Rollins. *J Am Vet Med Assoc* 162(12):1054-1058, 1973.

Although most studies of murine and avian leukemia indicate vertical transmission of the disease from mother to offspring, cases of leukemia in clusters of unrelated cats strongly implied, in earlier work, horizontal transmission in this species. The mode of transmission is speculative, but saliva and tracheal secretions of infected cats have been shown to contain virus, the disease can be induced by intranasal exposure to the virus, and contact controls of experimentally or naturally infected cats have developed leukemia. The present report is concerned with the clinical and pathologic features of leukemia and feline infectious peritonitis (FIP) in a household of 35 stray cats, presumably unrelated to one another. Ten cases of leukemia and four cases of FIP occurred in this group. The significantly high frequency of observed occurrence of leukemia compared to that expected by chance, together with the unrelatedness of the animals, indicated the likelihood of horizontal transmission of the disease. The clinical disease in leukemic cats was characterized first by fever and progressive anemia; later, by the presence of malignant cells of varying type in their blood. The leukemia in each cat was classified histologically as malignant lymphoma, reticulum-cell type. Treatment of leukemic cats with blood transfusions helped relieve weakness and lethargy associated with anemia; prednisone decreased fever and the number of abnormal circulating lymphocytes; cyclophosphamide was more effective than prednisone in decreasing the number of abnormal lymphocytes but was more toxic to bone marrow; vincristine and l-asparaginase did not cause noticeable improvement. Two of the cats with FIP developed abdominal fluid early in the course of illness and two others had early clinical signs similar to those seen in the leukemic cats before developing characteristic abdominal fluid.

2922 ESOPHAGEAL CANCER IN THE TRANSKEI: 1955-69. *(E.)* Rose, E. F. (Bantu Cancer Registry, East London, South Africa). *J Natl Cancer Inst* 51(1):7-16, 1973.

During a 15-year survey (1955-69) of esophageal cancer among the Bantu of the Transkei, 5095 cases were reported to the registry from a population of 1.7 million. Of these, 3281 were confirmed by doctors. The survey was divided into three five-year study periods. Tabulations for the first and last periods are presented and show an incidence rate for all cases of 35/100,000 for males and 17-19/100,000 for females for the whole area (African standard). Variation in incidence was marked among the 26 districts, ranging from 116 for males and 50 for females in the Butterworth area to 3 and 2, respectively, in Bizana during the first study period (1955-59). In the third period the pattern of variation between the districts followed that of the first period, and high- and low-incidence areas tended to remain as such. There was, however, a slight decrease in reported cases from the high incidence areas and an increase in the low incidence areas. More cases were confirmed in the second period, and the male to female ratio remained 2:1. It is evident that carcinoma of the esophagus has reached high proportion in the Transkei. The incidence rates are far above those of most other countries.

2923 ASBESTOS FIBRE IN THE LUNG AND MESOTHELIOMA. A RE-EXAMINATION OF THE MALMÖ MATERIAL. *(E.)* Pooley, F. D. (U. Dept. Pathology, General Hosp., Malmö, Sweden). *Acta Pathol Microbiol Scand* [A]81(8):390-400, 1973.

An examination of general lung tissue with an electron microscope for asbestos fibers is reported. The 65 cases examined consisted of 33 cases of pleural and peritoneal mesothelioma and 32 control cases, matched for age and sex. Of the mesothelioma cases 82% were positive for asbestos, while the total control cases were 53% positive for asbestos. Furthermore, the asbestos positive cases in the mesothelioma group contained a maximum of several hundred to a minimum of 50 fibers per case, while the control group positive cases ranged from 25 to 100 fibers per case. The most common fibers detected in the mesothelioma group were amphibole asbestos while chrysotile was the more predominant fiber detected in the controls. The difference in asbestos fiber content obtained between the mesothelioma group and controls is larger than the difference obtained in asbestos body counts made with an optical microscope on tissue from the same cases. In those cases found to be positive for asbestos bodies by light microscopy amphibole asbestos fibers were the predominant fiber type detected.

2924 CIRRHOSIS AND HEPATOMA OCCURRING AT BOSTON CITY HOSPITAL (1917-1968). *(E.)* Purtilo, D. T. (Armed Forces Inst. Pathol., Washington, D.C.) and L. S. Gottlieb. *Cancer* 32(2):458-462, 1973.

Cirrhosis and hepatoma at Boston City Hospital have progressively increased in frequency, especially cirrhosis associated with chronic alcoholism. Alcoholic cirrhosis or fatty nutritional cirrhosis (FNC) increased from 31% (1917-1926) to 61% (1955-1968) of all types of cirrhoses, and one-third more hepatomas occurred in this group. Although post-necrotic cirrhosis (PNC) and hemochromatosis decreased by half, the occurrence of hepatoma associated with PNC increased twofold and threefold in subjects with hemochromatosis. Chronic alcoholism is considered as a significant pathogenic agent in the production of FNC, hemochromatosis and PNC; 43 of the 98 subjects with hepatoma gave a history of alcohol abuse. More widespread alcohol abuse among this group is suspected since data was not available for the whole group. An average of 8 yr ensued from the onset of alcoholic cirrhosis to the appearance of hepatoma in 23 alcoholic patients.

2925 MALIGNANT MELANOMA IN EAST ANGLIA, ENGLAND. *(E.)* Bakos, L. (Addenbrooke's Hosp., Cambridge, England) and A. L. MacMillan. *Br J Dermatol* 88(6):551-556, 1973.

A series of 137 patients with primary malignant melanoma of skin occurring in the Cambridge area in 1961-71 is reviewed. The histological material has been classified into lentigo maligna melanoma, superficial spreading melanoma and nodular melanoma. Consideration of type, site and incidence of melanoma is compared with previously reported series from Boston and Sydney. An inverse relationship between latitude of residence and melanoma incidence or mortality in populations of European decent was found. The reports indicate that there is greater exposure to sunlight from occupation, habit, or hobbies in melanoma patients that in control groups. In the melanoma patients there is an excess of subjects with fair complexion and red hair who sunburn easily and sun tan with difficulty. In xeroderma pigmentosum, a hereditary skin disorder with greatly increased liability to skin carcinoma of exposed skin, there is an increased incidence of melanoma of exposed skin. Thus, it appears likely that melanomas are induced by exposure to ultraviolet light. The apparent induction of melanoma by ultraviolet light in unexposed skin could be explained by a hypothetical solar circulating factor that is produced in skin exposed to ultraviolet light and that stimulates malignant change in pigment cells in unexposed skin.

2926 AN EPIDEMIOLOGIC STUDY OF GONORRHEA AND CANCER OF THE PROSTATE GLAND. *(E.)* Heshmat, M. Y. (Howard U., Coll. Med., Washington, D.C.), J. Herson, J. Kovi and R. Niles. *Med Ann DC* 42(8): 378-383, 1973.

The relationship between gonorrheal infection and development of prostatic cancer was studied using the empiric (graphic) method and the time series analysis method (spectral analysis). Age specific death rates for cancer of the prostate, pancreas and lung were computed from vital statistics of the United States (1937-1967). Trends in age specific mortality rates from prostatic cancer could be explained by exposure to a carcinogenic condition that stopped sometime in the past. It is hypothesized that gonococcal infection in the pre-chemotherapy preantibiotic times and the resultant iatrogenic complications may be related to development of cancer of the prostate later in life. The mortality from prostatic cancer (1941-1970) and the incidence of gonorrhea (1891-1940) were obtained from the National Health Service of Denmark. Death rate curves for prostatic cancer and incidence curves for gonorrhea match well with a lag period of 45 yr. Spectral analysis gave a lag period of 42 yr and a coherence coefficient that differed from zero at level p<.05. If the demonstrated relationship is of causal nature, one could predict a continuing decline of mortality from prostatic cancer due to attrition of the cohort that received the old treatment.

2927 OVARIAN TUMORS IN PREGNANCY. A PRIVATE HOSPITAL TEN YEAR SURVEY. *(E.)* White, K. C. (St. Mary's Long Beach Hosp., Ca.). *Am J Obstet Gynecol* 116(4):544-550, 1973.

Of the 35,461 live births considered by this study, 38 patients were operated upon for ovarian tumors complicating pregnancy, giving an incidence of one ovarian tumor surgically treated for each 958 live births. Among these surgery patients the age range was 18-44 yr, the average being 27.2 yr. The parity is weighted toward those in their first or second pregnancy. Of the ovarian tumors reported, 70-80% were of these four types: benign cystic teratoma, serous cystadenoma, cystic corpus luteum and mucinous cystadenoma. Operations upon the group of 12 wk or less gestational age were done on an urgent or emergency basis. In five of twelve such patients the resected ovary or cyst included the corpus lutem. All of these infants survived, except one twin who was delivered at 34 wk. Only two of these five patients received progesterone. The remaining seven patients of this group had varying diagnoses and a fetal survival of only 17%, in part because of the surgical procedure employed. The 15 patients operated upon between the 12-15 wk of gestation represent the optimum management of ovarian tumors in pregnancy. Progesterone or progestins were more uniformly used in this group, but the therapy was so diverse as to be meaningless. The fetal salvage was 73%. The remaining 11 patients were subjected to operation after 20 wk gestation and had 100% fetal survival. Of the entire patient sample, there were four instances of gross error in diagnosis.

2928 ASBESTOS IN THE WORK PLACE AND THE COMMUNITY. *(E.)* Newhouse, M. L. (London Sch. Hyg. Tropical Med., England). *Ann Occup Hyg* 16(2):97-107, 1973.

The fibrogenic properties of asbestos dust were detected early; later, knowledge accumulated about the carcinogenic properties of the mineral and the hazard of bronchial carcinoma and mesothelial tumors was recognized. Mortality studies have measured the effect of exposure on working populations. Recent analysis of data from a cohort of asbestos factory workers shows that even with low to moderate exposure there is excess mortality from cancer of the lung and pleura and other cancers, after more than 25 years' observation. The mesothelioma rate increases both with severity and length of exposure. Occurrence of these and other tumors appears to be dose-related. The markers of a community effect of asbestos dust in the environment are the occurrence of mesothelial tumors in neighborhoods of a source of asbestos dust, and the presence of asbestos bodies or calcified asbestos pleural plaques in the general population. Conditions giving rise to neighborhood mesothelial tumors apparently no longer occur, but the importance of adequate control in all countries where asbestos is mined or manufactured is stressed.

2929 CANCER INCIDENCE IN FIJI. *(E.)* Boyd, J. T. (Scottish Home Hlth. Dept., Edinburgh, Scotland), R. Doll and C. H. Gurd. *Int J Epidemiol* 2(2):177-187, 1973.

A cancer registration scheme was started in the Fiji Islands in January 1965. The background to the scheme is described and cancer incidence rates derived from five years' registration are reported. Comparison between the two main ethnic groups, Fijian and Indian, shows that all cancers taken together were somewhat commoner in Fijian males than in Indian but almost equally common in both groups of females. Both island groups show rates substantially lower than those recorded in more industrialized societies, but it seems unlikely that incomplete registration accounts for more than a small proportion of the total deficiency. The cervix uteri is the most common site to be affected in both races and the present data do not support earlier suggestions that Fijian women are less often affected than Indian women. Both sets of rates were about double those recorded in England. Breast cancer ranked as the second most common site for cancer in women in both ethnic groups, the incidence being two or three times greater in Fijian women than in Indian, but relatively low in comparison with rates in Europe and North America. Thyroid cancer rates are notably high among Fijian women, as well as for women throughout the Central and South Pacific area. There is some evidence of excessive occurrence of liver cancer among Fijian men. Many of the cancers common to Europe and North America were rare in Fiji in both main ethnic groups. This held for cancers of the lung and stomach and particularly for cancers of the colon and rectum in Fijian men.

2930 TRENDS IN MORTALITY FROM CANCER OF THE UTERUS IN CANADA AND IN ENGLAND AND WALES. *(E.)* Kinlen, L. J. (Radcliffe Infirmary, Oxford, England) and R. Doll. *Br J Prev Soc Med* 27(3):146-149, 1973.

The evidence that carcinoma-in-situ of the cervix uteri commonly develops into invasive cancer is strong but inconclusive, and the prophylactic value of screening for carcinoma-in-situ in healthy women continues to be questioned. To determine whether screening programs are followed by any appreciable change in the mortality from cervix cancer, data obtained in British Columbia where an extensive screening program has been conducted since 1949 were compared with those from other areas of Canada and with those from England and Wales where screening was not carried out. Mortality rates were considered for three age groups, namely, 20-44 yr, 45-64 yr, and 65 and over. The results for Canada showed that mortality from cervical cancer declined materially in the last 10 yr, particularly under 45 yr of age, but that there is very little difference between the experience in British Columbia and other areas. Only at ages 45-64 does British Columbia appear to have any appreciable advantage. Mortality data for England and Wales showed decreases at ages 20-44 during the past decade, but in contrast to Canada, fairly constant rates at ages 45-64. The results for the 45-64 yr group in British Canada were consistent with the view that prophylactic screening can reduce mortality due to cervical cancer, but, since the findings were far from dramatic and were not seen at other ages, the reduced mortality can not yet be attributed to the screening program. Other information, such as prevalence of hysterectomy at different ages, is also needed.

2931 EPIDEMIOLOGIC EVIDENCE FOR TRANSMISSION OF HODGKIN'S DISEASE. *(E.)* Vianna, N. J. (Cancer Control Bureau, New York St. Dept. Hlth., Albany) and A. K. Polan. *N Engl J Med* 289(10):499-502, 1973.

The hypothesis that in schools where a case of Hodgkin's disease had occurred in a teacher or student, more than the expected number of additional cases would appear among teachers or students (or both) attending the same school was tested in order to evaluate the possible role of horizontal transmission in Hodgkin's disease. Two objective epidemiologic approaches were applied. In a two-time period approach it was found that five of eight secondary public schools with cases diagnosed during the 1960-1964 time period had other cases diagnosed during the years 1965-1969. In contrast none of the matched control schools had cases in the latter period. In an index-secondary approach, it was found that schools with index cases had a higher than expected number of secondary cases. The results of both analyses were statistically significant at high levels and support the concept that some form of transmission may be important in the occurrence of Hodgkin's disease.

2932 LUNG CANCER AMONG CIGAR AND PIPE SMOKERS. *(E.)* Wynder, E. L. (Div. Epidemiol., American Hlth. Fdn., New York, N.Y.) and K. Mabuchi. *Preventive Med* 2:529-542, 1973.

The smoking of cigars and pipes has been held relatively innocuous in terms of its etiological role in lung cancer even though the carcinogenicity of these forms of tobacco products has been well-established for cancer of the upper alimentary tract. The purpose of the present communication is to review the epidemiological literature linking cigar and pipe smoking to the development of lung cancer and to report on the association of smoking cigars and pipes to lung cancer based upon a 10 yr retrospective study carried out in New York City. The study included 30 male lung cancer patients who had smoked exclusively cigars and/or pipes. It was found that heavy cigar and pipe smoking is associated with development of lung cancer although cigar and pipe smokers have a lower risk than cigarette smokers, related possibly to differences in inhalation practices and to age at which smoking began. Furthermore, the lower risk for Jewish males with respect to cigarette smoking does not apply to cigar and pipe smoking, indicating that the lower risk related to differences in smoking habits rather than constitutional factors, since Jewish males smoke fewer cigarettes and more cigars and pipes than non-Jewish males. Also, the age of male lung cancer patients who have smoked only cigars and pipes is older than that for corresponding cigarette smokers, reflecting possibly an older age at start of smoking or a lesser deposition of smoke in the respiratory tract of cigar and pipe smokers due to less inhalation.

2933 INVASIVE MOLE AND GESTATIONAL CHORIOCARCINOMA IN DENMARK, 1940-1969. *(E.)* Mogensen, B. (Cancer Res. Inst., Radiumstationen, Aarhus, Denmark). *Acta Obstet Gynecol Scand* 52(3):211-220, 1973.

A survey has been made of all invasive moles and gestational choriocarcinomas registered during a 30 yr period in Denmark, a nation with a population of about 5 million (1971). All the diagnoses have been verified histologically by reappraisal. Based on clinical data the two groups of patients are described with reference to the following criteria: incidence and age; gravidity, parity and latency; dissemination and course of disease; and treatment. Some case histories are reported which contrast with the view generally prevailing before the era of chemotherapy, i.e. that choriocarcinoma was a highly malignant and rapidly progressive disease. All the 19 patients who had an invasive mole, including 5 metastases, became free from symptoms following treatment by surgery, irradiation or chemotherapy, and transition from this lesion to choriocarcinoma and did not occur in any of these cases. Among the 50 choriocarcinoma patients the over-all mortality was 64% with - excepting one case - an average survival time of five months; 18 patients (36%) - 10 of them with metastases - recovered. A decreasing mortality was observed during the period under investigation but much additional work has to be done before the prognosis of the disease approaches that of invasive mole. Surgery has now been abandoned as the primary treatment of trophoblastic tumors; methotrexate alone is used or combined with other cytostatics. The results of this study illustrate the many difficulties encountered in the diagnosis, treatment and follow-up of these rare trophoblastic tumors. The need for coordination of detection and treatment of choriocarcinoma is strongly indicated from scientific as well as therapeutic points of view.

2934 EPIDEMIOLOGIC SURVEILLANCE OF MESOTHELIOMA IN CANADA. *(E.)* McDonald, A. D. (Dept. Epidemiology Hlth., McGill U., Montreal, Quebec, Canada) and J. C. McDonald. *Can Med Assoc J* 109(5):359-362, 1973.

The number of fatal malignant mesotheliomas was ascertained for the period 1960-70 by contacting all pathologists in Canada. The annual incidence was steady between 1966 and 1970 at 1.4 per million population. Of 71 cases registered in 1968-70 and not previously reported, 66% were pleural, 24% peritoneal and the remainder in both sites; 45% of tumours were in women. The diagnosis of mesothelioma was approved by the Canadian Mesothelioma Panel in 59%. Sixty-nine cases were successfully investigated epidemiologically. A history of definite or probable occupational asbestos exposure was found in 30% of male cases compared with 11% of controls, but in none of the female cases or controls. However, among cases, four women and one man had had domestic exposure to dusty clothing of an asbestos worker. Most of the excess occupational exposure was in the manufacture of asbestos products or insulation and little in mining or milling. No case other than those occupationally or domestically exposed had lived within 20 miles of asbestos mines or mills. These results suggest that asbestos exposure is associated with a relatively small proportion of the tumors that Canadian pathologists call mesotheliomas. There was no evidence that cigarette smoking potentiates asbestos exposure in the production of mesothelioma. The main findings of the 1968-1970 survey were very similar to those from an earlier period, 1960-1968.

2935 A NOTE ON TRENDS IN MORTALITY FROM CANCER OF THE CERVIX, ONTARIO AND BRITISH COLUMBIA, 1950-1971. *(E.)* Sellers, A. H. (Ontario Cancer Treatment and Res. Fdn., Toronto, Ontario, Canada) and E. E. McLerie. *Can J Public Health* 64(4):364-367, 1973.

Mortality rates from cancer of the cervix uteri and total uterus at ages 25 and over for British Columbia, where a screening program has been carried out, and for Ontario, where a program has not been carried out, showed a continuing long-term tendency to decline from 1950, but no significant differences for the two different areas. There seemed, therefore, to be little evidence for the reduction in mortality in screened populations, as was anticipated by many observers. The observed data did show that the long-term decline affected most age groups, more particularly women under 65.

2936 GROWTH RATE INVESTIGATION AND TUMOR LETHAL DOSE IN EWING'S SARCOMA. *(E.)* Pearlman, A. W. (Hosp. Joint Dis., Med. Ctr., New York, N.Y.). *Acta Radiol* 12(1):57-70, 1973.

2937 FECAL CONSTITUENTS OF POPULATIONS WITH DIVERSE INCIDENCE RATES OF COLON CANCER. *(E.)* Reddy, B. S. (American Hlth. Fdn., New York, N.Y.) and E. L. Wynder. *Proc Am Assoc Cancer Res* 14(March):79, 1973.

2938 SMOKING AND CANCER. *(E.)* Smith, A. (Clin. Sci. Building, Manchester, England). *Lancet* (7817):1448, 1973.

2939 INCIDENCE OF GASTRIC MALIGNANT NEOPLASMS IN THE POPULATION OF THE CITY OF WARSAW IN THE CITY OF WARSAW IN THE YEARS 1963-1968. *(E.)* Gadomska. H. (Inst. Oncology, Warsaw, Poland), T. Koszarowski and Z. Karewicz. *Pol Med J* 11(6): 1523-1530, 1972.

2940 ORAL TUMOURS IN KENYA. *(E.)* Cameron, H. M. (Dept. Path., U. Nairobi, Kenya). *Pathol Microbiol (Basel)* 39(3-4):187-195, 1973.

2941 EPIDEMIOLOGY OF LEUKEMIA-LYMPHOMA IN MID-NEBRASKA. *(E.)* Caldwell, G. G. (Kansas City Lab., Kansas), R. C. Rosenlof, H. M. Lemon, R. E. Bailey and T. D. Y. Chin. *Nebr Med J* 58(7): 233-237, 1973.

2942 AN EPIDEMIOLOGIC ANALYSIS OF MORTALITY AND GASTRIC CANCER IN NEWFOUNDLAND. *(E.)* Pfeiffer, C. J. (Fac. Med., Mem. U. Newfoundland, St. John's), J. G. Fodor and E. Canning. *Can Med Assoc J* 108(11):1374-1380, 1973.

2943 THE RELATIVE FREQUENCY OF BRAIN TUMOURS IN EGYPT. *(E.)* Sorour, O. (Fac. Med., Cairo U., Egypt), M. Rifaat and M. Lofti. *Afr J Med Sci* 4(2):179-186, 1973.

2944 INTRACRANIAL TUMOUR PATTERN IN IBADAN, NIGERIA. *(E.)* Odeku, E. L. (Surg. Dept., U. Ibadan, Nigeria), A. Adeloye, B. O. Osuntokun and A. O. Williams. *Afr J Med Sci* 4(2):137-141, 1973.

2945 MALIGNANT TUMOURS OF THE CONTROL NERVOUS SYSTEM IN MIGRANT POPULATION OF EAST AFRICA. *(E.)* Chopra, S. A. (H. A. Aga Platinum Jubilee Hosp., Nairobi, Kenya). *Afr J Med Sci* 4(2): 215-221, 1973.

2946 A STATISTICAL TEST FOR MEASURING UNIMODAL CLUSTERING: A DESCRIPTION OF THE TEST AND OF ITS APPLICATION TO CASES OF ACUTE LEUKEMIA IN METROPOLITAN ATLANTA, GEORGIA. *(E.)* Larsen, R. J. (Vanderbilt U., Nashville, Tenn.), C. L. Holmes and C. W. Heath, Jr. *Biometrics* 29(2):301-309, 1973.

2947 TESTING OF DU PONT RUBBER CHEMICALS FOR HEALTH HAZARDS. *(E.)* Johnson, P. R. (Du Pont Rubber Chemicals Dept. Res. Station, Wilmington, Del.). *Rubber J* 155(4):37-38, 40, 42, 44, 1973.

2948 CYTOPHOTOMETRIC AND AUTORADIOGRAPHIC CELL PROLIFERATION STUDIES IN PARAPROTEINEMIC HAEMOBLASTOSIS WITH LEUKEMIC DIFFERENTIAL COUNT ALTERATIONS. *(Ger.)* Queisser, W. (Ulm U. Int. Med. Pediatrics Ctr., W. Germany), D. Hoelzer and U. Queisser. *Klin Wochenschr* 51(5):230-234, 1973.

2949 OSHA MOVES TO PROTECT PLANT WORKERS AGAINST "CANCER AGENTS". *(E.)* Anonymous. *Amer Paint J* 57(49):64-65, 68, 1973.

2950 THE CIRCADIAN RHYTHM OF TUMOR CELL DIVISION. *(Rus.)* Kharlampovich, S. I. (Sci. Res. Inst. Radiol., Obninsk, USSR) and T. P. Svinogeyeva. *Biull Eksp Biol Med* 74(2):66-70, 1973.

2951 MORTALITY FROM CANCER AND CARDIOVASCULAR DISEASES IN THE COUNTY BOROUGHS OF ENGLAND AND WALES CLASSIFIED ACCORDING TO THE SOURCES AND HARDNESS OF THE WATER SUPPLIES, 1958-1967. *(E.)* Stocks, P. (Colwyn Bay, Wales). *J Hyg (Camb)* 71 (2):237-252, 1973.

See also:

* (Rev): 2419, 2464, 2476,
* (Chem): 2498, 2580
* (Viral): 2643
* (Path): 2908

2952 CALCIUM METABOLISM IN TUMORS - ITS RELATIONSHIP WITH CHROMIUM COMPLEX ACCUMULATION: V. THE INTERACTION NUCLEIC ACID-CALCIUM AND THE CALCIFICATION OF TUMORS. *(E.)* Anghileri, L. J. (VA Hosp., Denver, Colo.). *Oncology* 27(1):30-44, 1973.

RNA from wheat germ and DNA from calf thymus were mixed in precipitation reactions with $CaCl_2$. In related experiments *in vivo*, tumor-bearing mice were injected with $^{45}CaCl_2$ and ^{45}Ca uptake by tumor cells was observed. About 25% of the RNA in the reaction mixture, but none of the DNA, was precipitated when mixed with $CaCl_2$. The presence of complexed chromium reduced by 50-20% the amount of RNA precipitated with $CaCl_2$ and affected the physiochemical properties of DNA. In studies *in vivo*, the highest uptake of ^{45}Ca by nuclear and mitochondrial cell fractions were found in mice bearing hepatomas and melanomas. The study of the cell fractions from experimental tumors indicated variations in the fraction distribution of calcium and nucleic acids related to the growth stage. The possible role of the cell membranes in the calcification process appeared emphasized by the accumulation of calcium and nucleic acids in the microsomal fraction.

2953 MALE BREAST CANCER. 3. BREAST CARCINOMA IN ASSOCIATION WITH THE KLINEFELTER SYNDROME. *(E.)* Scheike, O. (Frederiksberg Hosp., Copenhagen, Denmark), J. Visfeldt and B. Petersen. *Acta Pathol Microbiol Scand* [A]81(3):352-358, 1973.

Thirty male patients with breast carcinoma were examined for sex chromatin. One of them was found to be sex chromatin positive. Chromosome anlysis of peripheral blood revealed the chromosomal constitution normally found in the Klinefelter syndrome. By pooling series of cases of male breast cancer, in which systematical screening for sex chromatin was made, it was found that 9 out of 242 patients were sex chromatin positive, which is a higher number than that found in the normal male population. Calculated on the basis of the incidence of breast cancer in women and men, and of the incidence of sex chromatin positive individuals in the normal male population, the incidence of breast cancer among patients with the Klinefelter syndrome was found to be about one fifth of the incidence of breast cancer in women and about 20 times the incidence of breast cancer in normal men. The breast carcinoma found in the sex chromatin positive patient reported in this study presented no special histological features. Testicular histology showed a pattern different from that normally found in the Klinefelter syndrome, since no totally hyalinized acellular tubules, which are considered pathognomonic for the Klinefelter syndrome, could be demonstrated.

2954 MODIFICATION OF HEXOSAMINIDASE ISOZYMES IN RAT HEPATOMA. *(E.)* Weber, A. (Cochin U. Hosp. Ctr., Paris, France), L. Poenaru, C. Lafarge and F. Schapira. *Cancer Res* 33(8):1925-1930, 1973.

The kinetic and electrophoretic properties of different normal young albino Wistar rat tissues (liver; brain, muscle, lung, placenta, and red cells) were studied and compared with those of slow- and fast-growing hepatoma and the properties of rat regenerating liver after partial hepatectomy, which represents the best example of intense cellular multiplication in adult liver, were compared with those of normal liver and hepatoma. The existence of two molecular forms of hexosaminidase in rat tissues and in rat serum is demonstrated. These isozymes have been separated by electrophoresis on cellulose acetate membrane and compared with isozymes of slow- and fast-growing hepatomas. In normal liver, as in slow-growing hepatoma, there is a preponderant band with slow anodic migration and another faint more anodic band. In two kinds of fast-growing hepatomas, the fast anodic band was constantly very intense while the slow band was much weaker or even disappeared. This pattern was similar to that of fetal liver (16th day). In brain the two bands possessed not only different charges but also different thermostabilities. The isozyme found in rapidly growing hepatoma had the same thermostability as the fast band of brain. There was generally a rough correlation between the growth rate of the tumors and the degree of isozymic abnormalities. Experiments performed on regenerating liver after partial hepatectomy show that the pattern is almost normal; the findings indicate that the isozymic modifications are not a mere consequence of cellular multiplication. The modifications of hexosaminidase isozymes give a new example of the resurgence of fetal enzymes in cancer.

2955 CONCANAVALIN A BINDING SITES IN THE PLASMA MEMBRANE OF NORMAL CELLS, SPONTANEOUSLY TRANSFORMED CELLS AND TUMOR CELLS AS VISUALIZED BY ELECTRON MICROSCOPY. *(E.)* Roth, J. (Inst. Pathol., Friedrich Schiller U., Jena, E. Germany), H. W. Meyer, G. Neupert and F. Bolck. *Exp Pathol (Jena)* 8:19-30, 1973.

The concanavalin A binding sites on the plasma membrane of normal and spontaneously transformed fibroblasts and of ascites tumor cells were studied. The question of differences in the molecular surface architecture of these cells as shown by means of Con A-peroxidase reaction (Con A-POD reaction) was examined. It was concluded that using the Con A-POD method and thin-section technique, a clear distinction between normal and malignant transformed cells cannot be made.

2956 CELL DENSITY DEPENDENT GLYCOLIPIDS IN NIL_2 HAMSTER CELLS, DERIVED MALIGNANT AND TRANSFORMED CULTURES. *(E.)* Critchley, D. R. (Imperial Cancer Res. Fund Labs., London, England) and I. Macpherson. *Biochim Biophys Acta* 296(1):145-159, 1973.

Normal and hamster sarcoma virus-transformed NIL_2 hamster cells were grown in the presence of ^{14}C-palmitate and glycolipid synthesis was observed. Normal NIL_2 cells contained five neutral glycolipids from a ceramide monohexoside to pentahexoside. The major sialic acid-containing component was sialyl dihexosylceramide. In sparse cultures of NIL_2

cells, little label was incorporated in the ceramide hexoside pathway, while in dense cultures the amount of labeling was increased. In virus-transformed cells synthesis of these glycolipids was blocked, though small amounts of pentahexoside were sometimes seen. The kinetics of synthesis of these density dependent glycolipids suggested that their synthesis was correlated with cell contact, though in two cases noncontacting cells synthesized the three ceramide compounds. Tumors produced by injecting nontransformed NIL$_2$ cells into hamsters contained only trace amounts of ceramide tri- and tetra-hexoside in 10 of 11 cases; in one case the normal glycolipid pattern was seen in tumor cells.

2957 SPONTANEOUS CHROMOSOMAL ABERRATIONS OCCURRING IN HEp-2-DERIVED CLONES. *(Rus.)* Poryadkova, N. A. (Inst. Med. Radiol., Obninsk, USSR), Ye. G. Kuz'mina and N. V. Luchnik. *Vopr Onkol* 19(3):80-86, 1973.

A total of 22 clones derived from single cells of the original HEp-2 cell population were cytogenetically studied for chromosome aberrations. The average percentage of cells with chromosome aberrations was 13.5% in the original population. The 22 HEp-2-derived clones contained 8-35% aberrant cells. The number of aberrant cells was higher in young clones than in the original cell population. This indicates that the genetic apparatus of the young clones of cancer cells is more sensitive to chromosome aberrations and destruction. These findings also support the hypothesis that there is latent genetic damage in cancer cells. Culturing of the clones for three months or longer led to disappearance of differences in the percentage of aberrant cells and the distribution of destroyed cells in both clones and the initial population. In these cell cultures, at least 85% of the cells had latent injuries of the genetic apparatus, which became manifested in visible chromosome aberrations in subsequent cell divisions.

2958 THE ANALYSIS OF MALIGNANCY BY CELL FUSION. IV. HYBRIDS BETWEEN TUMOUR CELLS AND A MALIGNANT L CELL DERIVATIVE. *(E.)* Wiener, F. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), G. Klein and H. Harris. *J Cell Sci* 12(1):253-261, 1973.

A range of highly malignant mouse tumor cells (Ehrlich, SEWA, MSWBS, YAC and YACIR) were fused with A$_9$ cells, L cell derivatives of low tumorigenicity. The resulting hybrid cells, so long as they retained something close to the complete chromosome sets of both parent cells, had little or no ability to grow progressively *in vivo*. Tumors arose from the injection of such hybrids in only 5-40% of X-irradiated newborn C$_3$H mice. These tumors were produced not by progressive growth of the cells injected, but by selective overgrowth of cells from which certain specific, but as yet unidentified, chromosomes had been eliminated. One of the infrequent tumors produced was explanted *in vitro*. A highly malignant cell line, A$_9$HT, was derived by passage through the animal. A$_9$HT cells were then fused with tumor cells. In all cases the resulting hybrid cells were found to be highly malignant; and the chromosome consititutions of the tumors which arose from the infection of these hybrids were not significantly different from those of the cells injected. It appeared that the L cell derivatives of low tumorigenicity contribute to the hybrid cells some factor, linked to specific chromosomes, that suppresses the malignant character of the tumor cell, and this suppression is removed, with consequent reappearance of the malignant phenotype, when certain chromosomes are eliminated.

2959 MYELOMA IN MOTHER AND DAUGHTER. *(E.)* Goldstone, A. H. (Western General Hosp., Edinburgh, Scotland), J. K. Wood and M. K. Cook. *Acta Haematol* 49(3):176-181, 1973.

The occurrence of multiple myeloma in mother and daughter is described. IgA myeloma diagnosed in a 60 yr old woman, and 4 yr later IgG myeloma was diagnosed in her 84 yr old mother. Familial occurrence of multiple myeloma has not previously been reported with both family members available for detailed study. On the basis of available literature, it seems likely that cases occurring in relatives are due to chance.

2960 PHYSICOCHEMICAL AND FUNCTIONAL CHARACTERISTICS OF MALIGNANT AND NORMAL RAT CELL POLYRIBOSOMES. *(Rus.)* Berdinskikh, N. K. (Inst. Oncol. Probl. Kiev, USSR). *Biokhimiya* 37(6):1237-1244, 1972.

Polyribosomal preparations were obtained from transplanted leukemic, sarcoma, nephroblastoma and hepatoma cells and from normal rat liver and spleen cells by sodium desoxycholate-treated postmitochondrial supernate sucrose gradient centrifugation or by precipitation with high magnesium ion concentrations. Analytical ultracentrifugation revealed 8 to 12 fractions in leukemic and tumor cell and in normal liver cell preparations, 6 fractions in a dimethylaminoazobenzene-induced hepatoma preparation, and 4 to 6 fractions in normal spleen cell preparations. Sedimentation coefficients of the liver polyribosomal fractions ranged from 80 to 380 S. Heavy components with sedimentation coefficients ranging from 280 to 380S were not found in polyribosomal preparations from dimethylaminoazobenzene-induced hepatomas but were present in the leukemic cell preparations. Findings for the leukemic cell preparation could not be compared with those for the normal spleen preparations used as a normal hematopoietic reference because of the high RNAase activity of the spleen. Monoribosome and diribosome level in liver polyribosomal preparations amounted to 20 and 10%, resp., of the total ribosome content and reached a level of 50 to 60% in malignant cell preparations. Heavy polyribosomes amounted to 70% in normal liver and were below 40 to 50% in malignant cell preparations. Polyribosomal RNA (obtained through phenolic deproteinization) revealed 6 to 9 fractions upon polyacrylamide gel electrophoresis with qualitative and quantitative differences depending on their origin. Eighty-five percent of the malignant cell polyribosomes were found to be detached from the endoplasmic reticulum membranes and free polyribosomes prevailed in the spleen preparations. In the normal liver cell preparations, 40% of polyribosomes were found in the cytoplasm and 60% were attached to the endoplasmic reticulum membranes.

2961 THE FINE STRUCTURE OF THREE MALIGNANT MELANOMAS OF THE IRIS.

(E.) Lee, W. R. (Tennent Inst. Ophthal., U. Glasgow, Scotland). *Albrecht von Graefes Arch Klin Opthalmol* 188:91-108, 1973.

Three malignant melanomas of the iris, classified as spindle-B type by paraffin histology, were studied by electron microscopy. The cells from each tumor were similar at the level of fine structure, but there was some variation in appearances between the tumors and the pattern suggested stages of dedifferentiation. Thus, the first tumor was characterized as amelanotic; the second contained melanin granules and also virus-like particles similar to those previously seen in experimental mouse and hamster melanomas; the third was particularly aggressive and was extensively necrotic. The fine structural features of the tumors differed in no significant way from previously published descriptions of posterior uveal melanomas and indicated an origin from stromal melanocytes.

2962 ULTRASTRUCTURE OF PROSTATIC BENIGN HYPERPLASIA AND CARCINOMA.

(E.) Sinha, A. A. (VA Hosp., Minneapolis, Minn.) and C. E. Blackard. *Urology* 2(2):114-120, 1973.

Fine structural criteria for differentiating the ultrastructures of benign prostatic hyperplasia (BPH) and of carcinoma of the prostate were determined by electron microscopic examination of biopsy specimens from 39 patients. Either transperineal or transrectal needle biopsies of the posterior portion of the prostate was taken, and at least two specimens were obtained from the same area of induration in each patient. One specimen was prepared for examination by light microscopy; the other for examination by electron microscopy. Of 70 specimens taken, adenocarcinoma was demonstrated in 23, BPH in 9, and prostatitis in 5; the remaining contained muscle and connective tissue only and were not processed. Differences between BPH and prostatic carcinoma could be seen in the fine structures of the secretory cells, in defects in the basal lamina and cell membranes, in the presence or absence of invasive cells, and in the quantities of stroma between glands. In some sections, fine structural changes characteristic of carcinoma were noted even though histological changes of carcinoma were not observed. This suggested that ultrastructural changes may be detected even before histologic changes of malignant neoplasms. If histological carcinoma could be shown to develop in areas where ultrastructural changes alone were first observed, it might be proved that the ultrastructural changes described in this report are a harbinger of histologic carcinoma.

2963 DNA POLYMERASES I AND II IN REGENERATING RAT LIVER AND MORRIS HEPATOMAS.

(E.) Baril, E. F. (Duke U. Med. Center, Durham, N.C.), M. D. Jenkins, O. E. Brown, J. Laszlo and H. P. Morris. *Cancer Res* 33(6):1187-1193, 1973.

Two nonmitochondrial forms of DNA polymerase, designated DNA polymerase I and II, were demonstrated in regenerating rat liver and Morris hepatomas. The enzymes were purified by fractionation with ammonium sulfate and chromatography on diethylaminoethyl cellulose and on phosphocellulose. Both purified enzymes show a strict requirement for a polydeoxyribonucleotide template and primer. Differences between the two enzymes include the inhibition of DNA polymerase II activity, but not DNA polymerase activity, by thiol-blocking agents. Other dissimilarities include the fact that DNA polymerase I is present in the nuclei and with cytoplasmic ribosomes from normal and regenerating rat liver and hepatomas. In normal rat liver, DNA polymerase II is detectable only in a smooth membrane fraction. In regenerating rat liver and hepatomas, DNA polymerase II activity is present in the nucleus and well as the smooth membrane fraction. The molecular weights of DNA polymerases I and II, as estimated by gel filtration, were 25,000 and 250,000, resp.

2964 A COMPARATIVE STUDY OF SOME PROPERTIES OF CHROMATIN FROM TWO "MINIMAL DEVIATION" HEPATOMAS.

(E.) Arnold, E. A. (Johns Hopkins U. Sch. Med., Baltimore, Md.), M. M. Buksas and K. E. Young. *Cancer Res* 33(6):1169-1176, 1973.

Chromatin from normal liver and from fast-growing (5123C) and slow-growing (9618A) Morris hepatomas maintained by i.m. transplantation into male Buffalo rats were compared with respect to their compositional and functional properties. The chromatin of both hepatomas had a slightly greater amount of nonhistone protein and RNA, an increased rate of incorporation of amino acid into histone protein, and a reduced turnover of nonhistone protein compared with normal liver chromatin. The chromatin from hepatoma 9618A had the same *in vitro* template activity for RNA synthesis as chromatin from the liver and the electrophoretic pattern of the nonhistone proteins was qualitatively identical to the pattern obtained with liver nonhistone proteins. However, chromatin from hepatoma 5123C showed a significant increase in template activity compared with that from normal liver, and several changes in the electrophoretic pattern of the nonhistone proteins were evident. It is suggested that some of the differences between the chromatin of the fast-growing hepatoma and that of normal liver are a characteristic of malignant progression rather than the process of neoplastic transformation.

2965 ADRENAL MICROSOMAL C_{19}-STEROID 5α-REDUCTASE ACTIVITY IN THE SNELL TRANSPLANTABLE RAT ADENOCORTICAL TUMOUR 494 AND THE EFFECT OF OESTRADIOL, TESTOSTERONE PROPIONATE AND ADRENOCORTICOTROPHIN IN INTACT AND GONADECTOMIZED RATS.

(E.) Maynard, P. V. (Welsh Natl. Sch. Med., Heath, Cardiff, England) and

E. H. D. Cameron. *Biochem J* 132(2):293-300, 1973.

In previous work the authors found that the activity of C_{19}-steroid 5α-reductase was high in normal rat adrenal and suggested that its role may be to provide a means of controlling the concentrations of circulating androgens, such as testosterone, and of providing a source of biologically active 5α-reduced C_{19}-steroids. In examining mechanisms for control of the activity of the enzyme, the authors have now investigated the effect of gonadectomy in both sexes and the administration of estradiol (150 μg/day for 7 days), testosterone propionate (2 mg/day for 7 days) and adrenocorticotrophin (ACTH, 0.1 mg/day for 3 days). The procedure for testing enzyme activity was to prepare the adrenal microsomes, add tritiated 7α-androst-4-ene-3,17-dione, incubate and, following addition of carriers, extraction of the steroids and purification by thin layer chromatography, measure the amount of 5α-androstane-3,17-dione produced per mg of microsomal protein. It was found that gonadectomy caused a marked increase in enzyme activity. Treatment with estradiol or testosterone decreased the activity of enzyme in castrated animals to approximately the values for intact animals; it also decreased the activity in the intact animals. ACTH produced less effect than the steroid hormones. The activity of the reductase in Snell tumor 494, previously found to be very low when determined with whole homogenate, was unexpectedly found to be similar to that in intact control animals when determined on microsomal material of the tumor. The authors suggest that the most likely explanation is that material exists in other parts of the tumor cell, which inhibits the microsomal activity in whole homogenates.

2966 SPONTANEOUS MYXOSARCOMA IN A COTTONTAIL RABBIT (*SYLVILAGUS FLORIDANUS*): OBSERVATION OF TUBULAR STRUCTURES IN THE ENDOPLASMIC RETICULUM OF TUMOR CELLS. *(E.)* Schaff, Z. (Natl. Cancer Inst., Bethesda, Md.), P. M. Grimley, H. J. Michelitch and W. G. Banfield. *J Natl Cancer Inst* 51(1):293-297, 1973.

Ultrastructural examination of a spontaneous myxosarcoma in a 6-yr-old cottontail rabbit (*Sylvilagus floridanus*) disclosed geometrically packed tubules (250 Å diameter) in the endoplasmic reticulum of >10% of tumor cell sections. The tubules were indistinguishable from structures previously described in virus encephalitis of rabbits and in virus-induced neoplasms of other experimental animals. They were also similar in dimensions and location to structures described in the cells of patients with connective tissue diseases. Present observations and review of the literature suggest that the tubular structures appear in two general forms: a tightly packed geometric or "crystalline" form presently identified and a less compact "tubuloreticular" form. Although their significance in the endoplasmic reticulum remains obscure, these tubular structures appear to have a limited distribution in mammalian tissues and usually are found in neoplasia or conditions associated with virus infection. The "tubuloreticular" form is usually observed in human connective tissue disease or neoplasms.

2967 STABLE CHROMOSOME CHANGES IN A HUMAN MALIGNANT MELANOMA. *(E.)* Chen, T. R. (M. D. Anderson Hosp. Tumor Inst., Houston, Texas) and M. W. Shaw. *Cancer Res* 33(9):2042-2047, 1973.

A cell culture was established from a human malignant melanoma. Karyotype analysis using some of the newer staining techniques revealed a modal chromosome number of 45 with the presence of a ring, a larger marker chromosome with an interstitial C-band, and at least five other structural rearrangements resulting in marker chromosomes. Most of these changes occurred *in vivo* since they were identifiable in the biopsy material 4 hr after the culture was initiated. They persisted in early and later subcultures. Although the culture grew slowly, the ring remained stable while endoreduplication decreased and 2S stem cells increased with time. It is possible that the high frequency of anaphase bridges observed explains the slow rate of proliferation of the tumor line. If many of the daughter cells receive incomplete genomes which are selected against, then the high rate of cell mortality would contribute to a lengthening of the doubling time.

2968 URINARY EXCRETION BY CANCER PATIENTS OF THE NUCLEOSIDES N^2,N^2-DIMETHYLGUANOSINE, 1-METHYLINOSINE, AND PSEUDOURIDINE. *(E.)* Waalkes, T. P. (Natl. Cancer Inst., Bethesda, Maryland), S. R. Dinsmore and J. E. Mrochek. *J Natl Cancer Inst* 51(1):271-274, 1973.

Through high-pressure, high-resolution, liquid ion exchange chromatography, in association with such analytic tools as gas chromatography, gas chromatography-mass spectrometry, and high- and low-resolution mass spectrometry, the nucleosides N^2,N^2-dimethylguanosine, 1-methylinosine, and pseudouridine, were identified and quantitatively determined in the urine of normal control subjects and patients with cancer. These nucleosides were derived primarily from transfer RNA, and their excretion reflected the turnover of that macromolecule. In this preliminary study of several types of malignancies, increased excretion of these compounds in at least half of the patients with such solid tumors as malignant melanoma, ovarian carcinoma, bronchogenic carcinoma and Burkitt's lymphoma, as compared with normal controls was noted.

2969 AN APPLICATION OF ELECTRON MICROSCOPIC CYTOCHEMISTRY TO HUMAN TUMORS. *(E.)* Yokoyama, M. (Dept. Urology, Fac. Med., U.Tokyo, Japan). *Acta Histochem Cytochem* 6(1):27-34, 1973.

Three prostatic carcinomas, ten renal cell carcinomas and three melanomas were studied by electron microscopic (EM) enzyme cytochemistry. Acid phosphatase of prostatic cancer cells was found in lysosomes, secretory vacuoles and vesicles as well as some Golgi apparatuses. The enzyme localizations remained unchanged in tumor cells two wk after castration when cells were viable. Renal cell carcinomas revealed to have positive alkaline phosphatase (ALPase) activity at microvilli resembling brush border and lateral plasma membranes facing adjacent tumor cells. The enzyme was negative at basal plasma membrane of the tumor cells. Proximal tubule cells of human kidney showed ALPase activity only at apical membranes. This indicated functional dedifferentiation of plasma membrane in the tumor cells. These observations were verified and substantiated by thiamine pyrophosphatase, inosine diphosphatase, and glucose-6-phosphatase (G6Pase) localizations in tumor cells. The tumor cells also showed positive G6Pase in endoplasmic reticulum. DOPA oxidase was successfully detected in smooth endoplasmic reticulum close to Golgi apparatus, innermost Golgi cisternae and premelanosomes both in cultured melanoma cells and melanoma tissues, which may have diagnostic value. Thus, EM enzyme cytochemistry was considered to be applicable and useful for human cancer study concerning histogenesis, diagnosis and to assess the treatment.

2970 FOLATE DEFICIENCY IN RATS BEARING THE WALKER TUMOR 256 AND THE NOVIKOFF HEPATOMA. *(E.)* Poirier, L. A. (Natl. Cancer Inst., Bethesda, Md.). *Cancer Res* 33(9):2109-2113, 1973.

The causes of the folate deficiency observed in rats bearing the Novikoff hepatoma and the Walker 256 carcinosarcoma were examined. The deficiency was determined by an elevated urinary excretion of the histidine catabolite, formiminoglutamic acid, following a loading dose of histidine to the tumor-bearing rats. The folate deficiency of the tumor-bearing rats could not be attributed to altered patterns of dietary consumption or of hepatic histidine-catabolizing enzymes. Further, the oligoglutamate folate fraction of the livers of rats bearing the Novikoff hepatoma was essentially identical to that of normal rats. The elevated formiminoglutamic acid excretion by rats bearing the Novikoff hepatoma could be reversed by high dietary levels of methionine and choline. High dietary levels of folate and of vitamin B_{12}, alone or in combination, had no effect on the elevated formiminoglutamic acid excretion by the hepatoma bearing rats; similarly, the addition of glycine, serine, formate, thymine, adenine, and guanine to the diets of rats bearing the Novikoff hepatoma did not diminish the elevated urinary formiminoglutamic acid levels. The results indicate that the folate deficiency of tumor-bearing animals may be the consequence of an excessive requirement for methyl groups in the host-tumor system. The results suggest that there may be a causal relation between the abnormal methyl metabolism seen in a broad spectrum of tumors and the common clinical observation of folate deficiency in cancer patients.

2971 THE ROLE OF ACROCENTRIC CHROMOSOMES IN NUCLEOLAR ORGANIZATION. I. CORRELATION BETWEEN THE LOSS OF ACROCENTRIC CHROMOSOMES AND A DECREASE IN THE NUMBER OF NUCLEOLI IN MENINGIOMA CELL CULTURES. *(E.)* Zankl, H. (Max-Planck-Inst., Munich, West Germany) and K. D. Zang. *Virchows Arch* [*Zellpathol*] 11(3):251-256, 1972.

The number of nucleoli in cells of meningioma cultures which have lost none, one, two or more acrocentric chromosomes were compared. Between tumors with normal karyotype and those showing a G monosomy only a slight difference was found. However there was a highly significant decrease in the nucleolar count after the loss of two or more acrocentric chromosomes ($p<0.01$). The loss of non-acrocentric chromosomes did not influence the number of nucleoli. Furthermore it was observed that meningioma cells being cultured only for 2-3 days, contained less nucleoli than cells of cultures from the 7th to 9th day ($p<0.01$). Already in this early stage of proliferation a strongly diminished number of nucleoli appeared in tumors having lost more than one acrocentric chromosome. These results indicate that acrocentric chromosomes play the most important role in nucleolar organization. The numerical and morphological findings show that explanted cultures of meningiomas can be regarded in certain periods of time as more or less synchronized cultures.

2972 CHANGES IN DISTRIBUTION OF ELECTRIC CHARGES IN THE SURFACE MEMBRANE OF RAT ASCITES HEPATOCARCINOMA CELLS INDUCED BY CON A. *(E.)* Yamada T. (Natl Cancer Res. Inst., Tokyo, Japan) and M. Yamada. *Nature* 244(5414):297-299, 1973.

Cell electrophoresis was used to determine if any specific changes in the surface of rat ascites hepatocarcinoma cells (AH62F and AH66F) associated with agglutination can be induced by the action of concanavalin A (con A). Con A induced biphasic changes in the electrophoretic mobility of both of the two strains of cells used. Low concentration of con A (5-40 μg/ml) increased mobility whereas high concentration (100-300 μg/ml) decreased mobility. Agglutination occurred when the concentration of con A was sufficient to facilitate a decrease in mobility. Apparently, when low concentrations of con A became bound to specific sites on the cell surface, the distribution of glycoproteins (and/or glycolipids) was changed. Some of the sialic acid molecules attaching to the terminal position of saccharide chains and originally situated at deeper sites may have shifted reactively into the electrokinetic plane of shear of the surface membrane, resulting in increased mobility. When cells were coated with excess con A, bound con A molecules may have covered negative elementary charges of the cell surface. They would then be too far below the new electrokinetic surface and mobility would decrease.

2973 ISOLATION OF THE NONHISTONE PROTEINS OF RAT WALKER CARCINOMA 256. THEIR ASSOCIATION WITH TUMOR ANGIOGENESIS. *(E.)* Tuan, D. (Children's Hosp. Med. Ctr., Boston, Mass.), S. Smith, J. Folkman and E. Merler. *Biochemistry* 12(17):3159-3165, 1973.

A procedure is described to isolate nonhistone proteins of malignant cell nuclei for the purpose of testing certain of its biologic activities. Walker rat carcinoma 256 cells were disrupted by N_2 cavitation, the nuclei isolated by differential centrifugation, the chromatin was purified, and the DNA separated from chromatin protein by chromatography on Bio-Gel A 5m in the presence of 4 M guanidine-HCl. Further separation of histone and nonhistone proteins was achieved with CM-Sephadex C-50, eluting alkylated nonhistones with 0.4 M guanidine-HCl in 0.01 M phosphate buffer and subsequent elution of histones in 2 M guanidine-HCl in 0.1 M acetic acid. Nonhistone and histone proteins were obtained in good yield and satisfactory purity. The histone and nonhistone proteins were characterized by amino acid analysis, macromolecular composition, and gel electrophoresis. A fraction containing tumor nonhistone proteins was mitogenic to endothelial cells, and, when implanted into the rabbit cornea, caused proliferation of vascular endothelium and new blood vessel formation. Tumor angiogenesis factor was thus demonstrated to be associated with a fraction also containing nonhistone proteins of malignant cell nuclei. Histone fractions had no activity in this bioassay.

2974 DIFFERENCES IN NUCLEAR RNA LABELLING PATTERNS OF BURKITT LYMPHOMA, LEUKAEMIC LYMPHOSARCOMA AND VARIOUS HUMAN LEUKAEMIAS.

(E.) Seeber, S. (Essen Clin., W. Germany), Ch. Hertenstein and C. G. Schmidt. *Klin Wochenschr* 51(13):680-684, 1973.

Labelling properties were compared for nuclear RNA preparations derived from AML, CML, CLL, leukemic lymphosarcoma and Burkitt lymphoma cells after 6 hr incubation with ^{32}P-phosphate. Fractionation of the RNA preparations on sucrose density gradients showed that the 45S fractions, which contained the ribosomal precursor RNA, possessed the highest specific activity. The lowest specific activity of nuclear 45S RNA was found in nuclear RNA of CML, indicating heterogenous, non-ribosomal type RNA for these cells. Also, a marked synthesis of preribosomal nuclear RNA (35S, 45S) took place in the CCL cells, known to have almost no DNA synthesis. Further studies will be required to determine whether the observed differences in rate of synthesis of nuclear 45S RNA, or other differences, reflect the differences in cytomorphology, cytochemistry and individual drug sensitivity shown by the various forms of human leukemia. Possible differences in nuclear RNA from lymphocytic and granulocytic cell lines are presently being studied.

2975 DNA SYNTHESIS IN ISOLATED HeLa NUCLEI: A SYSTEM FOR CONTINUATION OF REPLICATION *IN VIVO*.

(E.) Hershey, H. V. (McArdle Lab., U. Wisconsin, Madison), J. P. Stieber and G. C. Mueller. *Eur J Biochem* 34(2):383-394, 1973.

DNA synthesis was studied in HeLa nuclei isolated from synchronized HeLa cells. In isolated S-phase nuclei, DNA synthesis required the four deoxynucleotide triphosphates, ATP and Mg^{2+}. Optimal synthesis also required proper pH, ionic conditions and the presence of a soluble macromolecular fraction from cytoplasm. In optimal conditions, initial rates of DNA replication in isolated nuclei range from 50-100% of the rate in the cells of origin; 1-2% of the HeLa DNA could be replicated *in vitro*. Cytosol proteins were essential for stabilization of the nuclear DNA replication system. Bromodeoxyuridine labeling of parental DNA strands showed that the ordered nature of *in vivo* DNA replication was preserved in isolated nuclei.

2976 HUMAN LEUKEMIC CELLS. FRACTIONATION AND CHARACTERIZATION OF RIBONUCLEIC ACIDS.

(E.) Wulff, U. C. (Children's Cancer Res. Fdn., Boston, Mass.), L. S. Desai and G. E. Foley. *Exp Cell Res* 75(1):39-46, 1972.

RNA extracted from log-phase human lymphocytic cells in large-scale cultures in Eagle minimal essential medium with 10% whole fetal calf serum was purified and subjected to long (2.5 x 300 cm) exclusion chromatography (Sephadex G-100 columns) by a novel method which provided greater separation of the four main fractions, as well as separation of lower molecular weight species of RNA with a high degree of resolution. The molecular weights of each fraction was determined by polyacrylamide gel electrophoresis. Similar chromatographic patterns were obtained with RNA isolated from lymphocytic cells derived from patients with acute lymphocytic leukemia or infectious mononucleosis, and from normal healthy donors. Although these overall RNA patterns appeared to be similar, some quantitative differences in percentage distribution of individual RNA fractions were evident, in particular: the lower yield of high molecular weight RNA (Peak 1); and the higher yield of low molecular weight species of RNA from human leukemic cells. In addition, nucleotide analyses revealed a higher ratio of A + U/G + C in the 5.5S RNA Peak II fraction isolated from human leukemic (CCRF-CEM) cells as compared with that of lymphocytic cells derived from patients with infectious mononucleosis, or from normal donors. Despite these minor differences, the rather extensive homology between the same RNA species obtained from cells deriving from patients with acute lymphoblastic leukemia or infectious mononucleosis, and normal donors may be of biological significance.

2977 STUDIES ON CERTAIN CYTOPLASMIC ENZYMES AND SPECIFIC ESTROGEN RECEPTORS IN HUMAN BREAST CANCER AND IN NONMALIGNANT DISEASES OF THE BREAST.

(E.) Hilf, R. (U. Rochester Sch. Med. Dentistry, New York), J. L. Wittliff, W. D. Rector, E. D. Savlov, T. C. Hall and R. A. Orlando. *Cancer Res* 33(9):2054-2062, 1973.

Samples of normal breast tissue, fibrocystic disease, fibroadenomas, and infiltrating ductal carcinomas were examined for several cytoplasmic enzymes and specific estrogen-binding capacity, as well as for the levels of RNA, DNA, and protein. Compared with normal breast tissue, the activities per mg DNA of pyruvate kinase, glucose 6-phosphate dehydrogenase, isocitrate dehydrogenase, hexokinase, glucose phosphate isomerase, and aspartate aminotransferase were significantly elevated in carcinomas. Furthermore, the activities of these enzymes were significantly higher in the carcinomas, compared with those of samples of fibrocystic disease and fibroadenoma. Neither the age of the patient at the time the tumor was removed nor the presence of metastatic lesions was apparently related to the biochemical characteristics, although the carcinomas from women 70 years or older showed somewhat higher activities of glucose phosphate isomerase, lactate dehydrogenase, and glucose 6-phosphate dehydrogenase. Carcinomas that possessed specific estrogen-binding capacity, <7 fmoles (10^{-15} moles)/mg protein as estimated by sucrose gradient centrifugation of tumor cytosols, had lower activities of glucose 6-phosphate dehydrogenase, isocitrate dehydrogenase, and pyruvate kinase and slightly higher levels of DNA. The data reported here indicate that infiltrating ductal carcinoma of the breast presents with certain metabolic alterations that are not shared by nonmalignant breast diseases. Carcinomas that are potentially hormone responsive demonstrate a different enzyme activity profile from that of tumors that are potentially hormone independent.

2978 "PROPHASING" AS A POSSIBLE CAUSE OF CHROMOSOME TRANSLOCATION IN VIRUS-FUSED CELLS.

(E.) Matsui, S. I. (Chromosome Res. Unit, Hakkaido U., Sapporo, Japan). *Nature* [*New Biol*] 243(128): 208-209, 1973.

G_1 and G_2 Chinese hamster DON cells grown for two generations in the presence of ^{3}H-thymidine and synchronized by Colcemid block and mitotic harvest were fused with unlabeled metaphase cells with the aid of UV-inactivated Sendai virus. Under these conditions, autoradiographic observations revealed the presence of a low frequency of chromosomal translocations between the labeled interphase and unlabeled mitotic karyons. These translocations occurred only when the labeled chromatin first underwent the phenomenon variously called "prophasing", "premature chromosomal condensation", or "chromosome pulverization." Such "prophasing" was evident 30 min after fusion, and translocations appeared 14-18 hr later following synchronization of the original metaphase and interphase nuclei in mitosis. Additional experimental results implied that the translocations were not due to nuclear fusion *per se*, but that other factors were apparently involved.

2979 ACCEPTOR CONTROL RATIO OF MITOCHONDRIA. FACTORS AFFECTING IT IN MORRIS HEPATOMA 5123 AND YOSHIDA HEPATOMA AH-130.

(E.) Feo, F. (Inst. Gen. Pathol., U. Torino, Italy), R. A. Canuto and R. Garcea. *Eur J Cancer* 9(3):203-214, 1973.

Several conditions affecting the acceptor control ratios of tumor mitochondria *in vitro* were studied. Mitochondria from AH-130 Yoshida ascites hepatoma and 5123 Morris hepatoma showed high acceptor control and ADP:O ratios approaching the theoretical values when suspended in a reaction mixture containing EDTA. Bovine serum albumin or NaF did not completely substitute for EDTA in achieving high acceptor control. The ATPase activity of these mitochondria was stimulated by Mg^{2+}; this metal ion also had a harmful effect on acceptor control and ADP:O ratios. EDTA induced a strong inhibition of the ATPase basal activity (unstimulated by dinitrophenol or added Mg^{2+}). Hepatoma mitochondria, incubated in the presence of EDTA, responded well to different regulatory mechanisms such as: changes in mitochondrial concentration, phosphate potential, and endogenous substrate content. The phosphorylative apparatus of hepatoma mitochondria exhibited, even in the presence of protective agents, a higher lability than that of mitochondria from neonatal and adult rat livers. If is suggested that, although a slight degree of functional and perhaps a structural damage occurs in hepatoma mitochondria *in vitro*, it is possible to obtain a high degree of coupling if protective agents are added to the suspensions and appropriate experimental conditions are selected.

2980 PROPERTIES OF RNA POLYMERASES FROM B16 MELANOMA.

(E.) Menon, I. A. (Clin. Sci. Division, U. Toronto, Canada) and H. F. Haberman. *J Invest Dermatol* 60(3):153-159, 1973.

Some of the properties of nuclear and mitochondrial RNA polymerases from melanotic and amelanotic B16 melanomas have been studied. Chromatin-bound RNA polymerase is activated by Mg^{++} more than by Mn^{++}. Addition of ammonium sulfate was found to activate the enzyme. Preincubation with phospholipase A or C as well as extraction with ether or iso-octane decreased the RNA polymerase activity in the presence of Mg^{++} but not that in the presence of Mn^{++}. The polymerase activity in the presence of high salt concentration (ammonium sulfate) was not affected by the phospholipases or by solvent extraction. Preincubation with trypsin was found to activate enzymatic activity in the presence of Mn^{++} to a greater extent than in the presence of Mg^{++}. In contrast to chromatin-bound RNA polymerase, RNA polymerase of melanoma mitochondria was decreased in the presence of either Mg^{++} or Mn^{++} by treatment with phospholipase A or C or by solvent extraction. Phospholipase D and wheat-germ lipase had no effect on either nuclear or mitochondrial RNA polymerase activity. Increasing concentrations of α-amanitin inhibited the nuclear RNA polymerase activity in the presence of Mn^{++} at high salt concentration but had no effect in the presence of either Mn^{++} or Mg^{++} at low salt concentrations. The RNA polymerase activity in melanoma mitochondria on the other hand was not inhibited by α-amanitin under any of these conditions. Actinomycin D inhibited RNA polymerase activity in melanoma mitochondria but rifamycin and rifampicin were ineffective. Of the various normal tissues studied (liver, brain, heart, lung, spleen, and kidney), the properties of the RNA polymerase from melanoma most closely resembled those of the RNA polymerases from liver. Similar results were obtained with melanotic and amelanotic tumors.

2981 PROTON SPIN-LATTICE RELAXATION STUDIES OF NONMALIGNANT TISSUES OF TUMOROUS MICE.

(E.) Frey, H. E. (Dept. Physics, U. Waterloo, Ontario, Canada), R. R. Knispel, J. Kruuv, A. R. Sharp, R. T. Thompson and M. M. Pintar. *J Natl Cancer Inst* 49(3):903-906, 1972.

C3H/HeJ mice were given implants of a mammary adenocarcinoma or of a 3-methylcholanthrene-induced fibrosarcoma in the muscles of one hind leg. At various times, beginning two days after transplantation, the water proton spin-lattice relaxation time in the laboratory frame (T_1) was measured by pulsed NMR experiments on tissue and tumor samples from these animals. $T_{1\rho}$, the spin-lattice relaxation time in the rotating frame, and T_2, the spin-spin relaxation time, were also tested. All tumors had significantly higher T_1 and $T_{1\rho}$ values than any other tissue. Nonmalignant tissues (spleen, kidney, liver and heart) in animals with tumors had higher T_1 values than in those without tumors, indicating that water is less ordered inside the former tissues. Some chemical or biologic intermediate may be introduced into the blood stream during neoplasm growth which brings about structural changes in water of nonmalignant tissue. T_2 showed no systematic variations among tissues of tumor bearing and tumor-free mice. T_1 and $T_{1\rho}$ values for tissues of mice injected with tumor cells two days before testing, and for mice infected with *Salmonella*, were not significantly different from these values in healthy mice.

2982 SOLUBLE PLASMA DNA POLYMERASE AND GUINEA PIG LEUKEMIA.

(E.) Nayak, D. P. (Sch. Med., U. California, Los Angeles) and M. Shoyab. *Life Sci* 13(5):575-584, 1973.

A soluble DNA polymerase activity was shown to be present in plasma of leukemic strain-2 guinea pigs, and absent in plasma of normal animals. A good correlation existed between the amount of the polymerase activity in plasma and the progression of disease as indicated by total number of white blood cells and by the appearance of leukemia lymphoblasts in the blood. Previous study of the properties of DNA polymerase in leukemic lymphoblasts together with the present results, suggest that soluble plasma polymerase is released from leukemic lymphoblasts. Even though DNA polymerase has previously been reported in the plasma pellet and leukemic cells of human and experimental animal leukemia, the presence of the enzyme in leukemic plasma has not heretofore been demonstrated. If the presence of soluble DNA polymerase should prove to be widespread among leukemias of viral and nonviral origin, it may be a useful tool in diagnosis.

2983 *IN VIVO* EFFECT OF ADENOSINE 3',5'-MONOPHOSPHATE ON EHRLICH ASCITES TUMOUR CELLS.

(E.) Seller, M. J. (Guy's Hosp. Med. Sch., London, England) and P. F. Benson. *Eur J Cancer* 9(7):525-526, 1973.

Since the concentration of cyclic AMP increases in untransformed cells as the population increases but decreases in transformed cells, and since addition of cyclic AMP to transformed or malignant cell cultures can inhibit cell division and restore morphological characteristics to those of untransformed cells, it appeared that density-dependent cessation of growth is controlled by intracellular cyclic AMP and that in some malignant cells this mechanism is defective. To determine whether cyclic AMP might be effective as a carcinostatic agent *in vivo*, young adult female A strain mice were injected with 10^7 Ehrlich ascites tumor cells, either s.c. to produce a solid tumor, or i.p. and, 3 days later, were given AMP i.p. twice daily for 4-5 days. The test group received the cyclic AMP, 10 mg/kg body weight, immediately preceeded by aminophylline, 50 mg/kg, each in 0.1 ml saline. The aminophylline group received aminophylline without AMP. The control group received saline alone. The tumor weights and numbers of ascitic cells in the test groups were half those in the control group, while those in the aminophylline group also were significantly, but less markedly, lower than those in the control group. Half the test mice had no ascitic fluid and in all of them the abdominal wall was normal, while all control mice had abundant ascitic fluid and distorted, infiltrated abdominal walls. The drug treatment also altered the morphology of the tumor cells and appeared to have no toxic properties. The impeding of growth by aminophylline alone suggested that inhibition of phosphodiesterase allowed enough native cyclic AMP to accumulate to limit cell growth.

2984 THE EFFECT OF ENVIRONMENTAL pH ON THE GROWTH OF NORMAL AND MALIGNANT CELLS.

(E.) Eagle, H. (Dept. Cell Biol., Albert Einstein Coll. Med., Bronx, N.Y.). *J Cell Physiol* 82(1): 1-8, 1973.

The cyclical pH fluctuations observed in bicarbonate-buffered media, involving an initial alkalinization to approximately pH 7.8-8.0 due to loss of CO_2, and a subsequent metabolic acidification to as low as pH 6.9, conspire to limit the growth of many cell types and contribute in large part to the early "contact" inhibition of growth observed in many normal cell cultures. The present work describes the effect of environmental pH on the growth of a number of human, mouse, monkey, rat, and hamster cells, as measured by plating efficiency, growth rate, and maximum population density. The effect on serum requirement and long-term viability were also examined. Medium for the cultures was replaced daily, and amount of cell growth was determined by measurement of protein after 6-11 days. Five strains of normal human fibroblasts had a well-defined pH optimum at 7.6-7.8, while two virus transformants and two cancer lines grew over a significantly broader range with a significantly more acid pH optimum (e.g., 7.3-7.6 to 7.0-7.9). There was somewhat less variation in the pH optimum for the growth of the various mouse and rat cells tested. A mouse-human hybrid grew well over a broad pH range, encompassing that optimal for both parental cells. Monkey or hamster cells grew over a considerably broader range of pH than did either human or mouse cells, centering at about pH 7.2-7.4. The optimum for cloning coincided with that for mass culture, and the pH effects on plating efficiency and initial growth rates were paralleled by those on maximum population density. Environmental pH had no significant effect, however, on the widely varying serum requirements of normal and malignant cells, and the optimum pH in turn was independent of serum concentration. The limited life expectancy of normal human fibroblasts in culture also was unrelated to the environmental pH observed with the usual bicarbonate-buffered media.

2985 NEW HUMAN CANCER CELL CULTURE LINES. I. SW-13, SMALL-CELL CARCINOMA OF THE ADRENAL CORTEX. *(E.)* Leibovitz, A. (Scott White Clinic, Temple, Tex.), W. B. McCombs III, D. Johnston, C. E. McCoy and J. C. Stinson. *J Natl Cancer Inst* 51(2):691-697, 1973.

This is the first of a series of papers documenting cell lines with unique characteristics or derived from sites where cell lines have not previously been reported. A permanent human tissue culture cell line SW-13 was established from a small-cell carcinoma originating in the adrenal cortex. The line was derived from viable "spillout" cells prepared by the technique of Lasfargues and Ozzello; 1-2 mm cubical explants of tumor tissue yielded only fibroblast-like cells and were discarded. The first colonies from spillout cells required 60 day incubation in primary culture before growth was sufficient to necessitate harvesting and subculturing. After that, the generation time became shorter. Electron microscopy revealed many bulb gap junctions (BGJ) in the cell line and occasional paired cisternae of the endoplasmic reticulum. The BGJ phenomenon appeared related to the capability of this cell line to form domes.

2986 PROLIFERATION KINETICS OF SHAY CHLOROLEUKAEMIA CELLS GROWN IN DIFFUSION CHAMBERS *IN VIVO*. *(E.)* Vilpo, J. A. (Second Dept. Path., U. Helsinki, Finland) and T. Rytömaa. *Cell Tissue Kinet* 6(5):489-498, 1973.

The proliferation kinetics of Shay chloroleukemia cells of rats was studied by the labeled mitoses technique using ^{3}H-thymidine and by other methods. Detailed estimates of the phase durations of the cell cycle for proliferating cells were obtained using young (3-4 day old) exponentially growing cultures in diffusion chambers *in vivo*; estimates of kinetic parameters were also obtained for "old" (10-12 day old) cultures and for local subcutaneous chloroma tumors. The data permitted calculation of the growth fraction and the cell loss factor. The results showed that chloroleukemia cells proliferate at a faster rate in diffusion chambers implanted in the peritoneal cavity than in local s.c. chloroma tumors. The mean cycle time for young cultures was 13.1 hr with durations for G_1, S and G_2 phases of 3.3, 7.7 and 2.1 hr, resp. This cycle time appeared somewhat longer than that for normal hematopoietic cells. The chloroleukemic cells behave "normally" in the sense that 46% of new cells produced at mitosis enter a non-cycling population. The non-cycling cells appeared to be mainly, if not solely, responsible for the incomplete morphological and enzymatic maturation which occurs in diffusion chamber culture of the cells and, with a limited life span, may also account for a large part of the cell loss which occurs in such cultures. While the calculated potential doubling time (20.5 hr) was very constant for different experiments, the doubling times actually observed varied considerably (24-58 hr). This meant that it is the rate of cell loss which is different from case to case and, owing to the closed nature of the diffusion chambers, that the mechanism of cell loss is cell death. It thus follows that cell loss is the dominant factor determining the growth of chloroleukemia cell populations; that the sensitivity of the cells to killing factors is "innate" to the tumor (or the host) donating the cells, as revealed by constant cell loss within an experiment but variation between experiments; and that donor tumor does not similarly determine proliferative activity of the cells when transferred to a new environment, as revealed by constant potential doubling time between experiments. Variable cell death and constant cell production may also bear relevance to effects of tissue-specific inhibitors where the outcome of treatment with tumor inhibitors may be dictated by the "innate" death rate of the cells, thus explaining complete regression in some treated leukemia animals, small prolongation of life in others.

2987 CLEAR-CELL ADENOCARCINOMA OF THE VAGINA IN A GIRL WHOSE MOTHER HAD TAKEN DIETHYLSTILBESTROL. *(E.)* Williams, R. R. (Peralta Hosp., Oakland, Calif.) and R. J. Schweitzer. *Calif Med* 118(6):53-55, 1973.

2988 *IN VITRO* THROMBOPLASTIN AND INTRAVASCULAR COAGULATION ACTIVITIES OF LEUKOCYTES IN LEUKEMIA. *(Fr.)* Gouault-Heilmann, M. (H. Mondor Hosp. Central Hematol. Immunol. Service, Créteil, France), B. Varet, Cl. Romano and F. Fosso. *Pathol Biol* 20 (Suppl):56-64, 1972.

2989 NEOPLASIA AND G MONOSOMY. *(Fr.)* Allard, S. (Hotel-Dieu Dept. Path., Montreal, Canada) and M. Cadotte. *Union Med Can* 101(10): 2116-2120, 1972.

2990 MORPHOLOGICAL ANOMALIES OF POLYNUCLEAR NEUTROPHILIC LEUKOCYTES OCCURRING IN PROLIFERATIVE DISEASES OF THE BONE MARROW. *(Fr.)* Muratore, R. (No affiliation), R. Follana, A. Khayati and J. Olmer. *Nouv Rev Fr Hematol* 13(3): 376-380, 1973.

2991 THE DIAGNOSIS OF PRELEUKEMIC CONDITIONS AND OF MYELO-MONOCYTIC AND ATYPICAL LEUKEMIAS. *(Fr.)* Zittoun, R. (No affiliation), J. Diebold, A. Bernadou, C. Dao, J. Zittoun, M. Cadiou, J. Prost-Dvojakovic, G. Bilski-Pasquier and J. Bousser. *Nouv Rev Fr Hematol* 13(3):380-390, 1973.

2992 CERTAIN ASPECTS OF PROGRESSION IN THE BIOLOGICAL PROPERTIES OF CELLS CULTIVATED *IN VITRO*. *(Rus.)* Dobrynin, Ya. V. (Moscow, USSR). *Vopr Onkol* 19(4):98-99, 1973.

2993 FEATURES OF GLYCOLYSIS REGULATION OCCURRING IN VARIOUS STAGES OF LIVER CARCINOGENESIS. *(Rus.)* Rubenchuk, B. L. (Kiev, USSR). *Vopr Onkol* 19(4):97-98, 1973.

2994 SHIFTS IN THE LACTATE DEHYDROGENASE ISOENZYME SPECTRUM OCCURRING IN MALIGNANT TUMORS AS A RESULT OF THEIR PROGRESSION. *(Rus.)* Gorozhanskaya, E. G. (Moscow, USSR). *Vopr Onkol* 19(4):96, 1973.

2995 CERTAIN ASPECTS OF TUMOR PROGRESSION VIEWED IN TERMS OF HISTOCHEMICAL AND ELECTRON MICROSCOPIC STUDIES. *(Rus.)* Raykhlin, H. T. (Moscow, USSR). *Vopr Onkol* 19(4):95, 1973.

2996 THE SELECTION PROCESS OF TUMOR CELL POPULATIONS. *(Rus.)* Vakhtin, Yu. B. (Leningrad, USSR). *Vopr Onkol* 19(4):93-94, 1973.

2997 A CONTRIBUTION TO THE CONCEPT OF TUMOR "PROGRESSION." *(Rus.)* Neyman, I. M. (Moscow, USSR). *Vopr Onkol* 19(4):92-93, 1973.

2998 SEX CHROMATIN (BARR BODY) AND INFLAMMATORY CARCINOMA OF THE BREAST. *(E.)* Gros, Ch. (Ctr. Hosp.-U. Strasbourg, France), C. Colin and P. Haehnel. *Biomedicine* 19(2):65-67, 1973.

2999 ULTRASTRUCTURAL AND FUNCTIONAL FEATURES OF THE BLOOD PLATELETS IN PATIENTS WITH ACUTE LEUKEMIA. *(Fr.)* Dobréva, A. (Inst. Sci. Res. Hematol. Blood Transfusions, Sofia, Bulgaria) and V. Ghénova. *Folia Haematol (Leipz)* 99(1):65-75, 1973.

3000 COINCIDENCE OF HIGH INCIDENCE PLASMOCYTOMA, CHRONIC LYMPHOID LEUKEMIA, AND RECTAL CARCINOMA IN ONE FAMILY. *(Ger.)* Lohrmann, H.-P. (Ctr. Internal Med. Pediatrics, U. Ulm, W. Germany), G. Schneider, K. Merten and A. Tenbaum. *Blut* 24:356-364, 1972.

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

AARONOV, A.
2855*
ABANOBI, S.E.
2500
ABLASHI, D.V.
2710
ABRAMSON, J.H.
2418
ADAM, E.
2917
ADELOYE, A.
2944*
AHLERS, P.
2796*
AKHMETELI, M.A.
2518
AL'PERT, A.E.
2568*
ALASTAIR, D.
2747
ALBERT, S.
2689*
ALBRECHT, T.
2658
ALCOCK, N.W.
2752
ALEXANDER, M.
2541*
ALEXANDER, P.
2705, 2736, 2890*
ALLARD, S.
2989*
ALLEN, P.T.
2612
ALTER, B.J.
2787*
ALTHOFF, J.
2465, 2528
ALTSHTEYN, A.D.
2684*
AMANO, K.
2619
AMAROSE, A.P.
2520
AMBUS, U.
2773*
AMLACHER, E.
2534
AMOUDRUE, K.
2558*
ANDERSON, B.
2673*
ANDERSSON, B.
2799*
ANDJARGHOLI, M.
2488
ANDRE-SCHWARTZ, J.
2656
ANGHILERI, L.J.
2952
ANKERST, J.
2666*
ARKHANGEL'SKII, A.V.
2568*
ARLON, R.
2427*
ARNOLD, E.A.
2964
ASHKAR, F.S.
2588
ASOFSKY, R.
2708
AUGENER, W.
2844*
AUGUSTI-TOCCO, G.
2846*
AUNGST, C.W.
2849*
AUSLANDER, H.P.
2803*
AUSSEL, C.
2706
AUST, J.C.
2724
AVIV, H.
2816*
AVTANDILOV, G.G.
2899
AWANO, I.
2619
AXELRAD, M.
2807*
AYANABA, A.
2541*
BACH, F.H.
2787*
BACH, M.L.
2787*
BAGLIONI, C.
2766
BAILEY, R.E.
2941*
BAKOS, L.
2925
BALDWIN, R.W.
2756, 2852*
BALK, S.D.
2640
BAND, P.R.
2851*
BANFIELD, W.G.
2966
BANNIKOV, G.A.
2564*
BANSAL, S.C.
2780*
BARBER, H.R.K.
2471*
BARBU, E.
2768*
BARENDSEN, G.W.
2590*
BARIL, E.F.
2963
BARKER, C.R.
2856*
BARKUN, N.A.
2603*
BARRETT, J.C.
2918
BARSKI, G.
2713
BARTH, R.F.
2796*
BARTSCH, H.
2479
BASTIEN, H.
2560*
BAUER, H.
2800*
BAUMAL, R.
2697
BAUMGARTENER, L.E.
2637
BAUR, F.J.
2569*
BAXT, W.
2632
BEAMER, W.G.
2912*
BEATHARD, G.A.
2742
BECKER, Y.
2634
BEDFORD, J.S.
2608*
BELLELLI, L.
2818*
BEN-EFRAIM, S.
2795*
BENEACERRAF, B.
2774*, 2842*
BENSON, P.F.
2983
BENTON, B.
2920
BENTVELZEN, P.
2657
BENTWICH, Z.
2841*
BENVENISTE, R.E.
2859*
BERDINSKIKH, N.K.
2960
BERENYI, E.
2838*
BERNADOU, A.
2991*
BERNHARD, M.
2850*
BEVERLEY, P.C.L.
2811*
BHATTACHARYYA, J.
2622
BIANCO, C.
2797*, 2798*
BIBERFELD, P.
2825*
BIERFELD, J.L.
2789*
BILSKI-PASQUIER, G.
2991*
BIRCKBICHLER, P.J.
2721
BIRKHEAD, B.M.
2824*
BLACK, H.S.
2583
BLACK, P.H.
2656, 2674*, 2731, 2733
BLACKARD, C.E.
2962
BLAU, H.-J.
2880*

BLEJER, H.P.
2427*
BLOCH, K.J.
2774*
BLOIS, M.
2570*
BLOIS, M.S.
2813*
BLOMGREN, H.
2799*
BLOOM, B.R.
2843*
BLOOM, E.T.
2850*
BLUNCK, J.M.
2540*
BOGART, B.
2797*
BOGOVSKY, S.P.
2579*
BOIRON, M.
2472*, 2661
BOIVIN, P.
2810*
BOLCK, F.
2955
BOLEK, F.
2534
BOLTON, P.M.
2527, 2531
BONE, G.
2848*
BONISSOL, C.
2678*
BOOT, L.M.
2757
BOQUOI, E.
2480
BORDES, M.
2560*
BORDZHARDZE, A.K.
2904*
BOSENBERG, H.
2465*
BOSS, J.H.
2865*
BOUILLON, D.
2706
BOURGAUX, P.
2650
BOURGAUX-RAMOISY, D.
2650
BOUSSER, J.
2991*
BOWEN, J.G.
2756
BOWEN, J.M.
2612
BOYD, J.T.
2929
BOYER, J.F.
2577*
BOYLSTON, A.W.
2820*
BOYNS, A.R.
2512
BOYSE, E.A.
2790*
BRACONI-ROMEI, L.
2667*
BRANDIS, H.
2886*
BRAUN, A.C.
2640
BRENNAN, M.
2689*
BREWEN, J.G.
2609*
BRILL, E.
2510
BRITTINGER, G.
2844*
BRITTON, S.
2833*
BROCKHAUS, A.
2557*
BROOME, J.D.
2692
BROUET, J.C.
2420*
BROVET, J.C.
2814*
BROWN, O.E.
2963
BRUSH, M.G.
2432*, 2433*
BRYAN, G.T.
2503, 2582*
BUCHAN, R.
2512
BUCHMAN, V.M.
2633
BUCHSBAUM, H.J.
2407
BUFFE, D.
2867*
BUKSAS, M.M.
2964
BULFONE, L.M.
2649
BUNTO, T.V.
2559*
BURCH, W.M.
2598*
BURTIN, P.
2867*
BUTTERWORTH, A.E.
2794*
BUXBAUM, J.
2734
BYFIELD, J.E.
2722
BYSTRYN, J.-C.
2776*
CABANA, V.
2828*
CADIOU, M.
2991*
CADOTTE, M.
2989*
CALDWELL, G.G.
2941*
CALVIN, M.
2537*
CAMERON, E.H.D.
2965
CAMERON, H.M.
2940*
CANIVET, M.
2661
CANNING, E.
2942*
CANUTO, R.A.
2979
CARDESA, A.
2528
CARRUTHERS, C.
2703
CARSON, C.A.
2672*
CARTER, D.M.
2593*, 2594*
CASPARY, W.
2542*
CATER, D.B.
2836*
CAWLEY, J.C.
2856*
CECHNER, R.L.
2898
CEGLOWSKI, W.S.
2717
CERNOV, K.S.
2518
CEVIK, N.
2772*
CHAHINIAN, P.
2580*
CHAKRABARTY, M.
2625
CHAN, E.L.
2693
CHANDLER, J.A.
2459*
CHANG, L.O.
2720
CHANG, S.-Y.
2625
CHEDD, G.
2404
CHEN, T.R.
2967
CHEPULIS, G.-K.S.
2626
CHERMANN, J.C.
2678*
CHILLER, J.M.
2777*
CHIN, T.D.Y.
2941*
CHIPYSHEVA, T.A.
2564*
CHIRIGOS, M.A.
2674*
CHOI, Y.S.
2715
CHOPRA, H.
2689*
CHOPRA, S.A.
2945*
CHOU, C.T.
2808*
CHOWANIEC, J.
2545*

CHUNG, E.B.
2522
CHURCHILL, W.H.
2699
CINADER, B.
2808*
CLAPP, W.
2802*
CLEMMESEN, J.
2498
CLEVE, H.
2857*
CLIFFORD, P.
2727, 2754, 2822*
CLIFTON, K.H.
2591*
COGGLE, J.E.
2605*
COHEN, B.
2542*
COHEN, I.R.
2775*
COHEN, J.A.
2453*
COHEN, S.M.
2582*
COHN, Z.A.
2711
COHNEN, G.
2844*
COLE, E.N.
2512
COLEMAN, D.V.
2638
COLIN, C.
2998*
COLLINS, J.J.
2731, 2733
COLNAGHI, M.I.
2562*
COMBS, J.W.
2517
CONDIT, E.S.
2593*, 2594*
CONSTANTINI-SOUROJON, M.
2795*
CONTRACTOR, S.F.
2866*
CONZELMAN, G.M.
2510
COOK, M.K.
2959
COOK, M.L.
2652
COON, F.B.
2569*
COSENZA, H.
2760
COSTANZA, M.E.
2707
COTTER, S.M.
2921
COTTIER, H.
2804*
COTTOM, D.L.
2742
COTTON, J.R.
2742
COX, H.W.
2642
COX, K.O.
2628
COX, R.
2500, 2506
CRITCHLEY, D.R.
2956
CROCKER, J.
2836*
CULP, D.
2407
CUMMINS, P.A.
2896
CURRIE, G.A.
2702
CURTIS, K.
2847*
CURTIS, S.B.
2590*
CUSTER, R.S.
2764
CUTLER, M.G.
2492
CUTTS, J.H.
2551*
CUZIN, F.
2463*
DABROWSKI, J.
2621
DALE, M.M.
2488, 2761
DALES, S.
2406
DALY, J.W.
2405
DAMJANOV, I.
2500, 2506
DAMJANOVICH, S.
2584
DANDEU, J.P.
2768*
DANZ, M.
2534
DAO, C.
2991*
DAUNE, M.
2524
DAVIES, H.
2866*
DAVIES, J.N.P.
2456*
DAWSON, P.J.
2663*
DE CASTRO, A.F.
2864*
DE-GROOT, L.
2586
DE NECHAUD, B.
2706
DE PETRIS, S.
2698
DE SEZE, S.
2888*
DE-THE, G.
2710, 2897
DE VRIES, A.
2893
DE VRIES, M.J.
2688*
DECARVALHO, S.
2415
DECKER, B.
2643, 2759
DECOSSE, J.J.
2782*
DEFENDI, V.
2858*
DEGRE, M.
2735
DEICHER, H.
2874*
DEICHMAN, G.I.
2879*
DEINHARDT, F.
2442*, 2653
DEL VILLANO, B.C.
2858*
DELLA PORTA, G.
2812*
DEMISSIC, A.
2727
DEMISSIE, A.
2822*
DEOREO, G.
2631
DERGE, J.G.
2625
DESAI, H.
2488
DESAI, L.S.
2976
DESMUKES, B.
2502
DESPLACES, A.
2814*
DESTREE, A.
2737
DIAMOND, E.L.
2604*
DIAMOND, L.H.
2830*
DICKSON, C.
2660
DIEBOLD, J.
2991*
DIJSKTRA, J.
2489
DINSMORE, S.R.
2968
DIPAOLO, J.A.
2497, 2578*
DISHON, T.
2865*
DIXON, F.J.
2712
DMOCHOWSKI, L.
2402, 2612, 2615
DOBREVA, A.
2999*
DOBRYNIN, YA.V.
2992*
DOENHOFF, M.J.
2765
DOLL, R.
2929, 2930

DONNER, L.
2680*
DONOVAN, P.J.
2497
DORSETT, B.H.
2827*
DOUGLAS, D.R.
2583
DOUGLAS, S.D.
2841*
DOWBEN, R.M.
2721
DREWINKO, B.
2881*
DRYLL, A.
2888*
DU TOIS, R.
2558*
DUBBS, D.R.
2647
DUBININ, N.I.
2596*
DUBISKI, S.
2808*
DUKOR, P.
2793*
DURANT, J.R.
2913*
DVORAK, H.F.
2699
DVORAK, K.
2782*
DWORKIN, E.C.
2479
DYACHENKO, N.S.
2679*
DYGIN, V.P.
2909*
DZHIOYEV, F.K.
2565*
EAGLE, H.
2984
EALES, L.
2830*
EAST, J.L.
2612
EASTON, T.G.
2644
EASTY, G.C.
2488
EATON, G.J.
2764
EBBESEN, P.
2620, 2635
EBERT, P.
2689*
EBNER, H.
2480
ECCLES, S.
2705
EDEN, A.
2797*, 2798*
EDWARDS, A.J.
2769*
EL-FIKY, S.
2664*
ELGER, W.
2470*
ELGJO, K.
2735
ELLIS, D.A.
2674*
ELSTER, K.
2907*
EMANUEL, N.M.
2559*
EMBLETON, M.J.
2852*
EMMERLING, P.
2875*
ENGERS, H.D.
2728, 2786*
EPSTEIN, W.
2813*
ERIKSSON, K.H.
2755
ERLANDSON, R.A.
2752
ESSEX, M.
2730
EVANS, C.H.
2497, 2578*
EVGEN'EVA, T.P.
2572*
EYER, G.
2558*
FAANES, R.B.
2715
FAHEY, J.L.
2850*
FAKHRI, O.
2750
FALK, L.A.
2653
FALK, R.E.
2817*
FARBER, E.
2506
FARRUGIA, R.
2643
FASSKE, E.
2623
FELDMAN, J.D.
2843*
FEO, F.
2979
FERON, V.J.
2573*
FERRERO, A.
2505
FETTING, R.
2623
FIALKOW, P.J.
2754
FIDLER, I.J.
2895
FIELD, A.M.
2638
FIELDSTEEL, A.H.
2663*
FINE, J.M.
2870*
FINGER, H.
2875*
FINOGENOVA, M.A.
2538*, 2574*
FISH, J.C.
2742
FITCH, F.W.
2760
FLIEDNER, T.M.
2908*
FODOR, J.G.
2942*
FOLEY, G.E.
2976
FOLKMAN, J.
2973
FOLLANA, R.
2990*
FOMBEUR, J.-P.
2575*
FORMAN, J.
2833*
FORREST, A.P.M.
2512
FOSSO, F.
2988*
FOWLER, N.A.
2610*
FOX, H.
2896
FRADELIZI, D.P.
2808*
FRANK, H.
2665*
FRANKS, C.R.
2847*
FREY, H.E.
2981
FRIEDMAN, H.
2717
FRIEDMAN, L.
2532, 2898
FRITSCH, W.
2407
FROMMEL, D.
2870*
FUCHS, R.
2524
FURTH, J.
2853*
GABALDON, M.
2523
GABRIEL, L.
2567*
GABUNIYA, U.A.
2904*
GADOMSKA, H.
2939*
GAINER, J.H.
2536*
GALLO, R.C.
2412, 2622
GARATUN-TJELDSTO, O.
2721
GARCEA, R.
2979
GARCIA, H.
2513, 2516, 2521
GARDNER, S.D.
2638
GARNIER-EXPERT, D.
2668*

GAVOSTO, F.
2863*
GEERING, G.
2850*
GELENDER, H.
2453*
GELSHTEIN, V.I.
2564*
GEORGIADES, J.
2612
GERGELY, J.
2845*
GERGELY, P.
2838*
GHENOVA, V.
2999*
GIACOMONI, D.
2828*
GIBBS, G.
2558*
GILMORE, C.E.
2921
GIMOVSKY, M.L.
2690*
GLASER, R.
2643, 2759
GLEICH, G.J.
2779*
GLEICHMANN, H.
2726
GLOVER, E.L.
2556*
GOLDBLATT, H.
2898
GOLDMAN, M.
2585
GOLDSCHMIDT, B.M.
2535
GOLDSTEIN, A.L.
2723
GOLDSTEIN, I.J.
2860*
GOLDSTONE, A.H.
2959
GOLESKI-REILLY, C.
2826*
GOLLERKERI, M.P.
2460*
GOOD, R.A.
2696, 2715
GOODHEART, C.R.
2417, 2440*, 2458*
GORODILOVA, V.V.
2873*
GOROSHKINA, G.I.
2596*
GOROZHANSKAYA, E.G.
2994*
GOTTLIEB, L.S.
2924
GOUAULT-HEILMANN, M.
2988*
GRABER, E.A.
2471*
GRACE, M.
2851*
GRAF, T.
2646
GRAHAM, C.E.
2529
GRANOFF, A.
2630
GRASSO, A.
2846*
GRAVELA, E.
2567*
GREAVES, M.F.
2765
GREEN, I.
2548*
GREEN, M.
2616
GREENWALD, P.
2456*
GRIFFEN, W.O., JR.
2802*
GRIFFITHS, K.
2459*, 2512
GRIMLEY, P.M.
2966
GROBBELAAR, B.G.
2803*
GROENEN, P.J.
2553*
GROHSMAN, J.
2835*
GROS, CH.
2998*
GROSSMANN, H.
2719
GROUCHY, J.
2473*
GRUNWALD, H.
2546*
GUERRA, A.
2822*
GUNVEN, P.
2727, 2822*
GUPTA, R.K.
2729
GURD, C.H.
2929
GUSHCHA, K.P.
2679*
GUTTERMAN, J.U.
2773*
HAAPALA, D.
2678*
HABERMAN, H.F.
2980
HADJI-AZIMI, I.
2834*
HAEHNEL, P.
2998*
HAGEMAN, P.
2657
HAKIM, J.
2810*
HALES, I.B.
2610*
HALL, C.F.
2672*
HALL, T.C.
2977
HAMAOKA, T.
2714, 2774*, 2842*
HAMILTON, L.J.
2913*
HAMMER, R.H.
2636
HAMMERLING, G.J.
2791*
HAMMOND, M.G.
2803*
HAMPAR, B.
2625
HAN, T.
2849*
HARCUS, A.W.
2424*
HAREWOOD, K.R.
2649
HARRIS, H.
2958
HARRIS, R.
2409
HARTLEY, J.W.
2620, 2641
HARTMANN, K.U.
2793*
HARVAN, D.
2570*
HASPEL, O.
2634
HAYES, H.M.
2892
HEADLEY, D.B.
2582*
HEATH, C.W., JR.
2946*
HEDGES, J.R.
2761
HEHLMANN, R.
2632, 2657
HEILPORN, V.
2606*
HEIMPEL, H.
2903*
HELLER, P.
2815*, 2828*
HELLSTROM, I.
2780*
HELLSTROM, K.E.
2780*
HENDERSON, B.
2885*
HENDERSON, B.E.
2920
HENDERSON, J.S.
2491
HENDERSON, W.J.
2459*
HENLE, G.
2727, 2822*
HENLE, W.
2727, 2822*
HERBERMAN, R.B.
2414
HERMENS, A.F.
2590*
HERSHEY, H.V.
2975
HERSON, J.
2926

HERTENSTEIN, CH.
2974
HERZENBERG, L.A.
2693
HESHMAT, M.Y.
2926
HESTON, W.E.
2502
HEUBNER, R.J.
2655
HEWETSON, J.F.
2822*
HICKS, R.M.
2545*
HIGDON, C.R.
2649
HILF, R.
2977
HILFRICH, J.
2494
HINES, J.E.
2746
HINUMA, Y.
2619
HIRAI, H.
2749
HIRSCH, J.G.
2778*
HIRSCH, M.S.
2656, 2674*
HIRSHAUT, Y.
2704
HLOZANEK, I.
2614, 2682*
HOBBS, J.R.
2750
HOELZER, D.
2908*, 2948*
HOFFMAN, M.
2743
HOLMES, C.L.
2946*
HOLMES, S.
2558*
HOLSTEIN, B.A.
2869*
HOLYOKE, E.D.
2744
HORTON, B.J.
2501
HORTON, J.D.
2501
HOSS, H.E.
2637
HOWK, R.S.
2617
HSIEH, D.P.H.
2544*
HUBER, H.
2911*
HUEBNER, R.J.
2641
HUGHES, A.M.
2537*
HUGHES, N.R.
2862*
HULL, E.V.
2688*
HUMPHREY, J.H.
2806*
HUNSMANN, G.
2665*, 2767
HUNT, P.S.
2744
HUNTER, E.
2725
HUNTER, L.
2802*
HURDLE, D.F.
2716
HUTH, F.
2557*
HUTTEROTH, T.H.
2857*
HWANG, K.M.
2495
HYDE, H.A.
2851*
HYMAN, R.
2737
ILBERY, P.L.T.
2408
IMBENOTTE, J.
2613
INBAR, M.
2758
INGRAM, A.J.
2543*
IOACHIM, H.L.
2690*, 2827*
ISAKA, H.
2749
ITO, N.
2486
IWAHARA, S.
2555*
JABLONSKA, S.
2621
JAKUBOWICZ, K.
2621
JANOSSY, G.
2765
JARRETT, O.
2730
JARRETT, W.
2730
JASMIN, C.
2678*
JEGASOTHY, B.V.
2593*
JENG, M.W.
2692
JENKINS, M.D.
2963
JENSEN, H.
2900
JERINA, D.M.
2405
JESSEN, C.
2636
JIRTLE, R.
2591*
JOHNSON, P.R.
2947*
JOHNSTON, D.
2985
JOLLOW, D.J.
2548*
JOSE, D.G.
2696
JOSEY, W.E.
2443*
JOSLIN, C.A.F.
2459*
JOSS, U.R.
2537*
JU, D.M.C.
2416
JULLIEN, P.
2627
JURAJDA, V.
2682*
JURIN, M.
2805*
JUSTRABO, E.
2560*
KAHN, A.
2810*
KAISERLING, E.
2771*
KAL, H.B.
2599*
KAMMER, K.
2624
KANO, S.
2843*
KAPPLER, J.W.
2743
KARA, J.
2618
KAREWICZ, Z.
2939*
KARNOVSKY, M.J.
2728, 2786*
KAROLYI, G.
2584
KASHMIRI, R.
2802*
KATZ, C.
2525, 2535
KATZ, D.H.
2714, 2774*, 2842*
KAUFMAN, R.H.
2917
KAUFMANN, H.
2611*
KAVANAH, M.
2726
KAWAMURA, Y.
2514
KAWASE, A.
2514
KAWAZOE, Y.
2481
KAZANTSEVA, I.A.
2899
KEAST, D.
2454*, 2628
KEDAR, E.
2854*, 2855*
KEEFER, L.K.
2513
KELLEN, J.A.
2519

KELLER, R.
2694
KELLER, S.E.
2690*
KELLOFF, G.J.
2655
KEMPFF, P.G.
2489
KERKVLIET, N.L.
2821*
KHARLAMPOVICH, S.I.
2950*
KHAYATI, A.
2990*
KILLEEN, A.A.
2617
KIMELDORF, D.J.
2821*
KING, R.J.B.
2433*
KINLEN, L.J.
2930
KIRSCHSTEIN, R.L.
2746
KISELIEVA, J.G.
2626
KISHIMOTO, S.
2868*
KIT, S.
2647
KITAGAWA, M.
2515
KJELLEN, L.
2666*
KJEMS, E.
2871*
KLAVIUS, J.V.
2530
KLEIN, D.S.
2504
KLEIN, E.
2673*, 2754
KLEIN, G.
2437*, 2695, 2727, 2754,
2822*, 2958
KLEIN, J.
2787*
KLIETMANN, W.
2687*
KLISAK, I.
2722
KLYUCHAREVA, T.E.
2879*
KNEZEVIC, S.
2462*
KNIGHT, S.C.
2831*
KNISPEL, R.R.
2981
KNOPF, P.M.
2737
KODAMA, H.
2861*
KOGA, H.
2669*
KOHAN, S.S.
2869*
KOHLER, H.
2760
KOHOUT, J.
2809*
KONA, P.
2678*
KONIG, E.
2844*
KOSLOVA, L.N.
2559*
KOSZAROWSKI, T.
2939*
KOTLER, M.
2634
KOURI, R.E.
2483
KOVI, J.
2926
KOZLOVA, M.D.
2633
KRAIN, L.S.
2419*, 2916
KRELLI, L.
2558*
KRUEGER, F.W.
2485
KRUEGER, G.R.F.
2701
KRUGER, F.W.
2511
KRULL, P.
2874*
KRUSE, H.
2880*
KRUUV, J.
2981
KUFE, D.
2632, 2657
KUMAR, S.
2762
KUNKEL, H.G.
2841*
KURAHARA, C.
2663*
KURTH, R.
2800*
KUSTER, G.G.R.
2539*
KUZ'MINA, YE.G.
2957
KUZNETSOV, O.K.
2683*
KVEDAR, J.
2689*
KWA, H.G.
2757
KWAPINSKI, J.3.G.
2739, 2832*
KWOCZYNSKI, M.Z.
2910*
LACOMBA, T.
2523
LAFARGE, C.
2954
LAFFARGUE, P.
2577*
LAGASSE, L.D.
2722
LAGEROF, B.
2589
LAHAM, S.
2526
LAIRD, H.
2730
LAISHES, B.A.
2499
LAMBIN, P.
2870*
LAMERTON, L.F.
2464*
LAMON, E.W.
2673*
LANGE, J.
2665*
LANGER, B.
2817*
LANGLEY, F.A.
2896
LARSEN, R.J.
2946*
LASZLO, J.
2963
LAUDER, I.
2848*
LE FRANCOIS, D.
2713
LE MEVEL, B.P.
2745
LEBEDEVA, G.A.
2600*
LEDER, P.
2816*
LEE, M.R.
2769*
LEE, W.R.
2961
LEIBOVITZ, A.
2985
LEMON, H.M.
2941*
LENNERT, K.
2771*
LENNETTE, E.H.
2662
LESKO, S.
2542*
LEVIN, V.J.
2633
LEVY, A.H.
2917
LEVY, J.A.
2641
LIABEUF, A.
2710
LIBERTI, P.
2766
LIEBERMAN, M.W.
2482
LIEBERMAN, P.H.
2752
LIEVENS, A.
2606*
LIJINSKY, W.
2513, 2521
LILIENFELD, A.M.
2604*
LILLE, I.

—-. 2814*
LIMBOSCH, S.
2606*
LIN, M.T.
2544*
LINDBERG, L.
2695
LINDGREN, J.
2819*
LINDLEY, J.D.
2742
LINDOP, P.J.
2605*
LING, N.R.
2831*
LINMAN, J.W.
2906*
LINNEMANN, R.
2709
LINNIK, A.B.
2518
LITVINOV, N.N.
2475*
LITWIN, S.D.
2857*
LIVINGSTON, D.M.
2859*
LOEGERING, D.A.
2779*
LOFTI, M.
2943*
LOHR, E.
2611*
LOHRMANN, H.-P.
3000*
LOISILLIER, F.
2706
LOPEZ, D.M.
2753
LOPEZ-REVILLA, R.
2686*
LOUIS, C.J.
2540*
LOUIS, J.
2777*
LOUSTAN, C.B.
2678*
LOWENTHAL, R.M.
2823*
LOWER, G.M.
2503
LUBEROFF, D.E.
2913*
LUCHNIK, N.V.
2957
LUDFORD, C.
2642
LUDWIG, J.
2539*
LUNTS, A.M.
2568*
LUX, M.
2768*
MABUCHI, K.
2932
MAC GREGOR, G.A.
2770*
MAC MILLAN, A.L.
2925
MAC PHERSON, I.
2956
MACDONALD, E.J.
2476*
MACH, J.-P.
2700
MACH, O.
2682*
MACIEJEWSKI, W.
2621
MACKEY, L.
2730
MACRAE, K.D.
2423*
MADDOX, W.A.
2913*
MADISON, R.M.
2504
MAGEE, P.N.
2543*
MAILE, A.
2588
MAKIDONO, R.
2829*
MALDONADO, J.E.
2779*
MALENCHENKO, A.F.
2603*
MANDEL, M.A.
2782*
MANN, P.
2817*
MANNERING, G.J.
2549*
MANSA, B.
2871*
MARES, I.
2589
MARESCOTTI, G.
2803*
MARTIN, D.P.
2746
MARTIN, F.
2560*
MARTIN, M.S.
2560*
MARTOS, L.M.
2625
MARUYAMA, K.
2612, 2615
MASUDA, T.
2791*, 2861*
MATEEVA, V.A.
2879*
MATHEWS, J.
2561*
MATHEWS, K.P.
2860*
MATSUI, S.I.
2978
MATSUOKA, Y.
2515
MATUSEVICH, L.L.
2596*
MAURER, L.H.
2748
MAVLIGHT, G.M.
2773*
MAY, E.
2645
MAY, P.
2645
MAYNARD, P.V.
2965
MAYR, W.R.
2872*
MAYYASI, S.A.
2649
MAZIERE, J.-CL.
2768*
MAZURENKO, N.P.
2626
MAZUROV, Y.I.
2909*
MC BRIDE, C.M.
2773*
MC CLURE, H.M.
2529
MC COMBS, W.B. III
2985
MC COY, C.E.
2985
MC DEVITT, H.O.
2712, 2791*
MC DONALD, A.D.
2934
MC DONALD, J.C.
2934
MC INTYRE, O.R.
2748
MC KHANN, C.F.
2724
MC LAUCHLAN, S.L.
2823*
MC LAUGHLIN, H.
2750
MC LERIE, E.E.
2935
MC MACKEN, M.DEL.V.
2594*
MC PHERSON, T.A.
2851*
MCLAUGHLIN, C.L.
2421*
MEDGYESI, G.A.
2845*
MEEKER, W.R., JR.
2802*
MEEVS, L.
2814*
MEHER-HOMJI, D.R.
2461*
MELNICK, J.L.
2917
MELSOM, H.
2785*
MENARD, S.
2562*
MENCK, H.
2885*
MENON, I.A.
2980
MERLER, E.
2973
MERTEN, K.

3000*
METCALF, D.D.
2474*
MEYER, H.W.
2955
MEYERS, P.
2753
MICHALIDES, R.
2657
MICHELITCH, H.J.
2966
MICHLMAYR, G.
2911*
MICKEY, M.R.
2885*
MICKLEM, H.S.
2763
MIKAELIAN, S.E.
2633
MILLER, E.C.
2479
MILLER, J.A.
2479
MILLER, J.M.
2637
MINTZ, U.
2893
MIRVISH, S.S.
2516
MITCHELL, G.F.
2712
MITCHELL, J.B.
2608*
MITCHELL, J.R.
2548*
MITCHELL, M.S.
2887*
MITSUHASHI, S.
2691
MIZUTANI, S.
2639
MOENNIG, V.
2665*, 2767
MOGENSEN, B.
2933
MOHR, U.
2485, 2528
MOLE, R.H.
2403
MONTESANO, R.
2505, 2543*
MONTGOMERY, C.T.
2881*
MOORE, V.L.
2876*
MORAL, M.D.
2789*
MOREL, G.
2668*
MORONI, C.
2670*
MORRIS, H.P.
2720, 2963
MORROW, J.
2595*
MORTENSEN, R.F.
2717
MOSKALEV, YU.I.
2597*
MOURALI, N.
2710
MOY, P.
2853*
MROCHEK, J.E.
2968
MUELLER, G.C.
2975
MUKHERJI, B.
2704
MULLER, M.
2719
MULLER, T.P.
2520
MUNTZING, J.
2891
MURAMATSU, T.
2790*
MURATORE, R.
2990*
MURPHREE, S.A.
2495
MURPHY, E.D.
2912*
MURRAY, A.W.
2490
MUSTEA, I.
2601*
MYRVIK, Q.N.
2876*
NAEGELE, R.F.
2630
NAGI, S.A.
2672*
NAGY, M.P.-C.
2845*
NAHMIAS, A.J.
2446*
NAIMY, N.K.
2550*
NAKAHARA, W.
2883*
NAKAJIMA, T.
2555*
NAKAKUKI, K.
2718
NAKANO, M.
2514
NASS, M.M.K.
2687*
NATHANSON, L.
2707
NATHENSON, S.G.
2790*
NAYAK, D.P.
2982
NAZERIAN, K.
2642
NECKERS, D.C.
2452*
NEEL, H.B., III
2837*
NEGRONI, G.
2725
NELKEN, D.
2781*
NELSON, R.L.
2497
NEUMANN, F.
2470*
NEUMANN, H.-G.
2493
NEUPERT, G.
2955
NEWEY, B.
2831*
NEWHOUSE, M.L.
2928
NEWTON, W.A. JR.
2612
NEYMAN, I.M.
2997*
NICHOLS, W.W.
2428*
NIELSEN, A.
2498
NILES, R.
2926
NILSONNE, U.
2738
NILSSON, A.
2587, 2755
NILSSON, T.
2891
NISHINO, Y.
2470*
NISHIZUKA, Y.
2718
NKRUMAH, F.K.
2675*
NOBUYUKI, I.
2576*
NOMOTO, K.
2829*
NONOYAMA, M.
2759
NOSACH, L.N.
2679*
NOWAKOWSKI, M.
2843*
NOWOTNY, A.
2835*
NUGENT, R.P.
2429*
NUSSENZWEIG, V.
2797*, 2798*
NUTTER, R.L.
2422*
O'CONOR, G.T.
2701
OCCELLA, E.
2558*
ODAKA, T.
2651
ODEKU, E.L.
2944*
OKON, E.
2865*
OLD, L.J.
2790*
OLDSTONE, M.B.A.
2712
OLIVIE, M.
2661
OLMER, J.

2990*
OLSEN, R.G.
2671*
OLSHEVSKY, U.
2634
OLSON, C.
2637
ONYANGO, J.
2727
ORENSTEIN, J.M.
2509
ORLANDO, R.A.
2977
ORTH, R.
2468*
ORTON, C.
2598*, 2889*
OSBORNE, D.P. JR.
2714
OSHIRO, L.S.
2662
OSTERKAMP, R.W.
2436*
OSTHER, K.
2709
OSUNTOKUN, B.O.
2944*
OUTZEN, H.C.
2764
PACKMAN, S.
2816*
PAKES, S.P.
2671*
PALOYAN, E.
2586
PAN, P.M.
2860*
PANI, P.
2567*
PANI, P.K.
2681*
PANINA, N.P.
2568*
PARKS, W.P.
2617, 2859*
PARMIANI, G.
2812*
PARWARESCH, M.R.
2771*
PATWARDHAN, V.C.
2851*
PAVLENKOMIKNAYLOV, YU.N.
2602*
PAYNE, L.N.
2681*
PEARLMAN, A.W.
2936*
PEDERSEN, B.
2894, 2901, 2902
PEGORARO, L.
2863*
PEGRUMC G.D.
2425*
PEREVOZCHIKOV, A.P.
2683*
PERIES, J.
2661
PERKINS, F.T.
2847*
PERKINS, I.V.
2675*
PERLMANN, P.
2747
PERO, R.W.
2570*
PETERS, R.L.
2655
PETERSEN, B.
2953
PETERSON, I.A.
2850*
PETO, R.
2561*
PETRANYI, G.
2838*
PETRICCIANI, J.C.
2746
PETROV, H.S.
2909*
PFEIFFER, C.J.
2942*
PHILLIPS, B.
2840*
PHILLIPS, W.M.
2656
PICK, A.I.
2893
PIERCE, C.W.
2708
PIHKO, H.
2819*
PINKHAS, J.
2893
PINN, V.
2707
PINTAR, M.M.
2981
PIRNAR, A.
2772*
PIROSKY, R.R.
2869*
PISTER, L.
2767
PITOT, H.C.
2501
PITOUT, M.J.
2489, 2547*
PITT, J.I.
2410
PLANTEYDT, H.T.
2915
PLISE, G.B.
2563*
PLISS, G.B.
2565*, 2905*
PLUMMER, G.
2445*
PLUZHNIKOVA, G.F.
2683*
POENARU, L.
2954
POIRIER, L.A.
2970
POIRIER, M.C.
2482
POLAN, A.K.
2931
POLLACK, R.
2659
POOL, R.R.
2585
POOLEY, F.D.
2923
PORTA, G.D.
2562*
PORYADKOVA, N.A.
2957
POSNER, H.
2570*
POTT, F.
2557*
POTTER, M.
2469*
POULIK, M.D.
2857*
POUR, P.
2528
PREHN, R.T.
2764
PRESSMAN, D.
2784*
PRESTON, R.J.
2609*
PRETLOW, T.G.
2913*
PRICE, L.A.
2455*
PRICE, M.R.
2756, 2852*
PRIESTER, W.A.
2892
PRIORI, E.S.
2612
PRITCHARD, H.
2763
PROCTOR, J.
2736
PROFFITT, W.R.
2674*
PROST-DVOJAKOVIC, J.
2991*
PROUKAKIS, C.
2605*
PRYME, I.F.
2721
PURCHASE, H.G.
2642
PURIFOY, D.
2630
PURNELL, D.M.
2517
PURTILO, D.T.
2924
PUSKAS, E.
2845*
PUSZTASZERI, G.
2700
QUEISSER, U.
2948*
QUEISSER, W.
2948*
RABKIN, R.
2830*
RABSTEIN, L.S.

2655
RADA, B.
2677*
RADOMSKI, J.L.
2510
RADZICHOVSKAYA, R.M.
2633
RAFF, M.C.
2698
RAFLO, C.P.
2671*
RAJU, M.V.
2571*
RAMULU, C.
2571*
RANGAN, S.R.S.
2629
RAO, P.R.
2654
RAPP, F.
2643, 2658, 2759
RAPP, H.J.
2522
RASK-NIELSEN, R.
2620
RASSIGA PIDOT, A.L.
2748
RATRIE, H.
2483
RAVITCH, M.M.
2693
RAWLS, W.E.
2441*, 2917
RAYBAUD, N.
2678*
RAYKHLIN, H.T.
2995*
RAYNAUD, M.
2678*
RECTOR, W.D.
2977
REDDY, B.S.
2937*
REDDY, C.R.
2571*
REED, W.B.
2813*
REEVE, T.S.
2610*
REICH, E.
2644
REICHERT, C.F.
2860*
REITZ, M.
2622
REMMERS, A.R. JR.
2742
REUBER, M.D.
2556*
REVESZ, L.
2755
REY, A.A.
2510
RIBLET, R.J.
2693
RICHMOND, L.M.
2540*
RICKINSON, A.B.
2408
RIFAAT, M.
2943*
RIGGS, J.L.
2662
RISDALL, R.J.
2724
RITTS, R.E., JR.
2837*
RITZMANN, S.E.
2742
RIVLIN, R.S.
2413
ROBIN, J.
2650
ROCCHI, G.
2822*
ROE, F.J.C.
2426*
ROGALSKY, V.Y.
2783*
ROGERS, A.I.
2789*
ROITT, I.M.
2840*
ROLLINGHOFF, M.
2741
ROLLINS, C.
2921
ROMANO, CL.
2988*
ROMER WILLIAMS, R.J.
2585
RONNBACK, C.
2587
ROPCKE, G.
2757
ROSE, E.F.
2922
ROSEMANN, E.
2865*
ROSENLOF, H.M.
2941*
ROSNER, F.
2546*
ROSS, J.
2816*
ROSSI, N.
2407
ROTH, J.
2955
ROWE, W.P.
2620, 2641
ROWLEY, D.A.
2760
RUBENCHUK, B.L.
2993*
RUDDERS, R.A.
2815*
RUDENSTAM, C.M.
2736
RUHLAND, D.
2623
RULE, A.H.
2826*
RUOSLAHTI, E.
2819*
RUSSELL, W.C.
2648
RYE, L.A.
2617
RYTOMAA, T.
2986
SAARNI, M.I.
2906*
SABIN, A.B.
2751
SACHS, L.
2758
SAGEBEIL, R.
2813*
SAINEROVA, H.
2680*
SAITO, K.
2691
SALLE, M.
2661
SALMON, S.E.
2478*
SAN, R.H.C.
2481
SAPRIN, A.N.
2559*
SARACINO, R.T.
2814*
SARIN, P.S.
2622
SARLES, H.E.
2742
SARMA, D.S.
2500
SARMA, S.R.
2506
SARTORELLI, A.C.
2495
SASAME, H.A.
2548*
SATO, N.
2687*
SAUER, G.
2624
SAVLOV, E.D.
2977
SAY, B.
2772*
SCHABORT, J.C.
2489, 2547*
SCHAFER, W.
2665*, 2767
SCHAFF, Z.
2966
SCHAPIRA, F.
2954
SCHARFF, M.D.
2697, 2734
SCHEIKE, O.
2953
SCHEINER, M.I.
2789*
SCHENDEL, D.J.
2787*
SCHLOM, J.
2657
SCHMERLER, H.
2604*
SCHMIDT, C.G.

2974
SCHMITT, G.
2611*
SCHNEIDER, G.
3000*
SCHNEIDER, R.
2492
SCHRADER, J.W.
2801*
SCHULTZ, D.R.
2789*
SCHULZE, B.
2487
SCHUSTER, C.R.
2520
SCHUSTER, R.
2729
SCHWARTZ, R.S.
2656, 2707, 2726
SCHWEITZER, R.J.
2987*
SCOLNICK, E.M.
2617, 2859*
SCOTT, R.M.
2824*
SCRIBNER, J.D.
2550*
SEEBER, S.
2974
SEGAL, A.
2525
SEIDMAN, I.
2525
SELIGMANN, M.
2420*
SELJELID, R.
2785*
SELLER, M.J.
2983
SELLERS, A.H.
2935
SETHI, K.K.
2886*
SEYTS, I.F.
2683*
SHABAD, L.M.
2467*
SHAMBERGER, R.J.
2431*
SHAPOT, V.S.
2466*
SHARP, A.R.
2981
SHARPINGTON, C.
2598*, 2889*
SHAW, M.W.
2967
SHEPHERD, S.
2690*
SHEVLYAGHIN, V.YA.
2626
SHIGEMOTO, S.
2868*
SHIGETA, S.
2619
SHILS, M.E.
2752
SHLYANKEVICH, M.A.
2685*
SHOEMAN, D.W.
2549*
SHOWS, T.
2643
SHOYAB, M.
2982
SHUBIK, P.
2505
SHULYAKOVSKAYA, T.S.
2559*
SIGEL, M.M.
2753
SIMMONS, R.
2747
SIMPSON, E.
2811*
SIMU, G.
2788*
SINGH, S.
2727, 2754, 2822*
SINGLA, O.
2796*
SINHA, A.A.
2962
SINOUSSI, F.
2678*
SIRSI, M.
2430*
SISKIND, G.W.
2776*
SIVAK, A.
2525
SJOGREN, H.O.
2780*
SKAPINYECZ, J.
2584
SKEHEL, J.J.
2648
SKUTELSKY, E.
2841*
SMITH, A.
2938*
SMITH, J.L.
2856*
SMITH, K.V.
2610*
SMITH, S.
2973
SMOAK, W.M.III
2588
SNAJDR, J.
2765
SOBIN, L.
2804*
SOKAL, J.E.
2849*
SOLLIDAY, S.M.
2708
SOLNIK, C.
2726
SOLOMON, A.
2421*
SOMOGYI, B.
2584
SOREN, L.
2825*
SOROF, S.
2484
SOROKINA, YU.D.
2579*
SOROUR, O.
2943*
SPAHN,G.J.
2655
SPALDING, J.W.
2570*
SPIEGELMAN, S.
2632, 2657
SPIRA, G.
2854*
SPITLER, L.
2813*
SRIVATANAKUL, P.
2884*
STEELE, K.
2890*
STEIN, H.
2771*
STEINER, L.
2695
STEINERT, G.
2606*
STEINMAN, R.M.
2711
STEMMERMANN, G.N.
2914*
STENBACK, F.
2505
STERN, E.
2448*
STEVENS, J.G.
2652
STEVENSON, D.E.
2507
STICH, H.F.
2499
STIEBER, J.P.
2975
STIMPFLING, J.H.
2787*
STINSON, J.C.
2985
STITCH, H.F.
2481
STJERNSWARD, J.
2695, 2738
STOCKS, P.
2951*
STORTS, R.W.
2672*
STRAKS, W.
2573*
STRONG, L.C.
2477*
STUART, F.P.
2760
STUTMAN, O.
2732
SUIT, H.D.
2805*
SULITZEANU, D.
2807*, 2854*, 2855*
SUNDBLAD, R.
2738
SUSSMAN, E.

2701
SUTOU, S.
2554*
SVEDMYR, A.
2727, 2822*
SVET-MOLDAVSKY, G.J.
2633
SVINOGEYEVA, T.P.
2950*
SVOBODA, J.
2614, 2680*
SWATSKE, C.
2533
SYMMES, L.R.L.
2569*
SZEDEDI, G.
2838*
TACHIBANA, T.
2740
TAGAMETS, M.A.
2625
TAKASUGI, M.
2885*
TAKEYA, K.
2829*
TAKIZAWS, S.
2514
TAMBOURIN, P.E.
2627
TANIOKU, K.
2861*
TARRO, G.
2751
TAYLOR, D.O.N.
2662
TAYLOR, G.
2762
TAYLOR, R.W.
2433*
TCHAO, R.
2488
TENBAUM, A.
3000*
TERASAKI, P.I.
2885*
TEYSSIE, A.R.
2869*
THATCHER, G.N.
2830*
THEMANN, H.
2623
THOMAS, W.R.
2454*
THOMPSON, R.
2885*
THOMPSCN, R.T.
2981
THOMSON, D.M.P.
2705, 2890*
THOMSON, S.
2884*
THORPE, E.
2507, 2508
TIHON, C.
2616
TODASO, G.J.
2859*
TOKUZEN, R.
2883*
TOMA, V.
2788*
TORRIELLI, M.V.
2567*
TORRIOLI, M.
2667*
TORRIOLI-RIGGIO, G.
2667*
TOTO, P.D.
2533
TOURNEUR, J.
2668*
TOWNSEND, C.M. JR.
2742
TRALKA, T.S.
2701
TREMPE, G.
2850*
TRUJILLO, J.M.
2881*
TS'S, P.O.P.
2542*
TSAGARELI, Z.G.
2904*
TSUKADA, E.
2486
TSUKADA, Y.
2749
TSUKIMOTO, I.
2740
TUAN, D.
2973
TURK, J.
2804*
TYRRELL, D.A.J.
2823*
TYTKO, S.
2431*
UEBERHORST, P.J.
2629
UEDA, G.
2853*
UHR, J.W.
2776*
UMEHARA, S.
2749
UNANUE, E.R.
2728, 2786*
URBAN, H.
2534
URIEL, J.
2706
USATEGUI-GOMEZ, M.
2864*
VAAGE, J.
2839*, 2882*
VAKHTIN,YU.B.
2996*
VAN DEN BRENK, H.A.S.
2598*, 2889*
VAN DER WATT, J.J.
2489
VAN DUUREN, B.L.
2525, 2535
VAN GRIENSVEN, L.J.L.D.
2688*
VANDERMEERSCH, B.
2457*
VANE, F.M.
2549*
VANKY, F.
2695, 2738
VANTSAK, N.P.
2679*
VARET, B.
2988*
VENKATARATHANAM, G.
2571*
VERGER, C.
2613
VERMA, A.K.
2490
VERNICK, D.
2595*
VIANNA, N.J.
2456*, 2931
VIDEBACK, A.
2871*
VIDRINE, J.G.
2649
VILPO, J.A.
2986
VIRY, B.
2560*
VISFELDT, J.
2953
VLAHAKIS, G.
2502
VLASOV, N.N.
2565*
VOGEL, A.
2659
VOLFSCN, N.I.
2563*
VOLJFSON, N.I.
2905*
VON KLEIST, S.
2867*
VOS, D.
2803*
VOS, G.H.
2803*
VUILLAME, M.
2897
WAALKES, T.P.
2968
WAGENER, D.J.T.
2792*
WAGNER, H.
2741
WAKEFIELD, J.ST.J.
2545*
WALDECK, W.
2624
WALKER, A.E.
2607*
WALKER, A.I.
2508
WALKER, A.I.T.
2507
WALLACE, R.E.
2746
WALTER, G.
2686*
WATABA, H.

2749
WEBER, A.
2636, 2954
WEBSTER, W.S.
2671*
WEEKS, J.L.
2491
WEIGLE, W.O.
2777*
WEIL, R.
2645
WEINBERG, E.
2634
WEINBERGER, P.C.
2913*
WEINSTEIN, I.B.
2509
WEINSTEIN, M.B.
2588
WEINTRAUB, I.
2722
WEISBURGER, E.K.
2504
WEISBURGER, J.H.
2504
WEISSBERG, M.
2496
WEISSMAN, C.
2434*
WEKERLE, H.
2775*
WEKSLER, M.E.
2435*
WELLS, S.A. JR.
2745
WELTMAN, J.K.
2721
WENDLING, F.
2627
WENK, M.L.
2504
WESSELY, Z.
2530
WHITE, A.
2723
WHITE, K.C.
2927
WHITFIELD, J.F.
2640
WHITMIRE, C.E.
2483
WHITTEN, J.B.
2436*
WIENER, F.
2958
WILBANKS, G.
2411
WILBANKS, G.D.
2438*, 2439*
WILDY, P.
2444*
WILLIAMS, A.O.
2944*
WILLIAMS, R.R.
2987*
WILLIS, C.E.
2431*
WILSON, J.D.
2716
WITKOP, B.
2405
WITTLIFF, J.L.
2977
WOLFE, L.G.
2653
WOLLMANN, R.
2665*
WONG, M.C.
2629
WOOD, J.K.
2959
WOODS, J.E.
2539*
WOODSIDE, N.
2689*
WROKLANS, M.
2810*
WULFF, U.C.
2976
WUNDER, W.
2566*
WYBRAN, J.
2813*
WYNDER, E.L.
2932, 2937*
XUMA, M.
2622
YAGI, Y.
2784*
YAKULIS, V.
2815*, 2828*
YAMADA, M.
2972
YAMADA, T.
2972
YAMAMURA, Y.
2868*
YAMASHITA, U.
2515
YAO, R.C.
2544*
YATA, J.
2740
YATANI, R.
2914*
YEAGER, F.M.
2864*
YIN, L.
2532
YOKORO, K.
2514
YOKOYAMA, M.
2969
YONG, D.C.T.
2832*
YOUDALE, T.
2640
YOUN, J.K.
2713
YOUNG, E.M.
2484
YOUNG, K.E.
2964
YOYAMA, U.
2486
YUHAS, J.M.
2607*
ZAMPETTI-BOSSELER, F.
2606*
ZANG, K.D.
2971
ZANKL, H.
2971
ZASUKHINA, G.D.
2596*
ZATZ, M.M.
2723
ZBAR, B.
2522
ZEMLA, J.
2677*
ZEROV, YU.P.
2683*
ZHDANOV, V.M.
2626
ZIGMOND, S.H.
2778*
ZITTCUN, J.
2991*
ZITTOUN, R.
2991*
ZOTTER, S.
2719
ZUEVA, J.N.
2626

- ACETAMIDE
  - N-OXIDATION
    - URINARY BLADDER, DOG, MONKEY, 2510

- ACETANILIDE
  - N-2-FLUORENYLACETAMIDE CARCINOGENESIS
    - MOUSE, HAMSTER, RAT, 2504
  - N-HYDROXY-N-2-FLUORENYLACETAMIDE CARCINOGENESIS
    - MOUSE, HAMSTER, RAT, 2504

- N-ACETOXY-2-ACETYLAMINOFLUORENE
  - DNA DAMAGE
    - HUMAN SKIN FIBROBLASTS, 2499
  - DNA REPAIR SYNTHESIS
    - HUMAN DIPLOID FIBROBLASTS, 2482

- N-ACETOXY-N-ARYLACETAMIDE
  - DECOMPOSITION
    - FORMATION OF FREE RADICALS, 2550*

- 2-ACETYLAMINOFLUORENE
  - ADRENAL CORTEX CELLS
    - STIMULATION, RAT, 2534
  - CHOLESTEROL SYNTHESIS
    - LIVER, RAT, 2501
  - DNA DAMAGE
    - HUMAN SKIN FIBROBLASTS, 2499
  - N-HYDROXYLATION
    - RULE OF CYTOCHROME P-450, 2548*

- N-ACETYLATION
  - 4-AMINO BIPHENYL
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503
  - 2-AMINOFLUORENE
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503
  - 2-AMINONAPHTHALENE
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503

- ACID PHOSPHATASE
  - ACTIVITY
    - REGRESSION, ESTRONE-INDUCED MAMMARY TUMOR, RAT, 2551*

- ADENOCARCINOMA
  - CARCINOEMBRYONIC ANTIGEN
    - CHANGES IN BLOOD GROUP SUBSTANCES, 2747
  - MAMMARY GLAND
    - LACTATE AND MALATE DEHYDROGENASE ISOZYMES, MOUSE, 2664*
  - 3-METHYL-4-DIMETHYLAMINOAZOBENZENE
    - CARCINOGEN-BINDING PROTEIN, RAT, 2564*
  - PROSTATE
    - ULTRASTRUCTURE, HUMAN, 2962

- ADENOCARCINOMA, CLEAR CELL
  - DIETHYLSTILBESTROL
    - MOTHER-DAUGHTER, CASE REPORT, 2987*

- ADENOCARCINOMA, PULMONARY
  - DIBUTYLNITROSOAMINE
    - RAT, 2565*

- ADENOMA
  - KIDNEY
    - 1-NITROSO-5,6-DIHYDROURACIL, RAT, 2516

- ADENOMA, PULMONARY
  - DIETHYLNITROSAMINE
    - ULTRASTRUCTURE, HAMSTER, 2573*

- ADHERENCE
  - POLYAMINE EFFECT
    - LYMPH-NODE CELLS ALLERGISED TO BP8 ASCITES TUMOR CELLS, 2836*
  - QUARTERNARY AMMONIUM COMPOUND EFFECT
    - LYMPH-NODE CELLS ALLERGISED TO BP8 ASCITES TUMOR CELLS, 2836*

- ADRENOCORTICAL CARCINOMA
  - SMALL-CELL LINE
    - HUMAN, 2985
  - STEROID REDUCTASE
    - RAT, 2965

- AFLATOXIN
  - EFFECT IN ANIMALS AND MAN
    - REVIEW, 2465*
  - INTERNATIONAL CHECK SAMPLE PROGRAM
    - 1972 STUDY, 2569*
  - LIVER CANCER
    - RAINBOW TROUT, 2566*
  - PRODUCTION
    - BIOLOGICAL FACTORS, REVIEW, 2468*

- AFLATOXIN B1
  - BIOSYNTHESIS OF STERIGMATOCYSTIN
    - ASPERGILLUS PARASITICUS, 2544*
  - DEOXYRIBONUCLEASE
    - LYSOSOMES, RAT LIVER, 2547*
  - DNASE
    - LIVER, RAT, MOUSE, 2489
    - LYSOSOMES, RAT LIVER, 2547*
  - LYSOSOME PERMEABILITY
    - LIVER, RAT, 2547*
  - METABOLISM
    - BIOCHEMICAL EFFECTS, HYPOPHYSECTOMIZED RAT, 2532
  - TUMOR INDUCTION
    - LIVER, RAT, MOUSE, 2489

- AFLATOXIN B2
  - DEOXYRIBONUCLEASE
    - LYSOSOMES, RAT LIVER, 2547*
  - DNASE
    - LYSOSOMES, RAT LIVER, 2547*
  - LYSOSOME PERMEABILITY
    - LIVER, RAT, 2547*

- AGE
  - CELL-MEDIATED IMMUNE RESPONSE
    - MOUSE, 2868*
  - TRANSPLANTATION
    - INTRAVENOUSLY INJECTED ALLOGENEIC TUMOR CELLS, RAT, 2889*
    - WALKER 256 CARCINOSARCOMA, RAT, 2821*

- AMINES, AROMATIC
  - N-OXIDATION
    - URINARY BLADDER, DOG, MONKEY, 2510

- 4-AMINO BIPHENYL
  - N-ACETYLATION
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503

- 2-AMINOFLUORENE
  - N-ACETYLATION
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503

- AMINOHYDROXYTHYMINE HYDRAZINE
  - RADIATION LETHALITY ENHANCEMENT
    - EHRLICH ASCITES TUMOR, MOUSE, 2601*

- 2-AMINONAPHTHALENE
  - N-ACETYLATION
    - LIVER CYTOSOL, HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503

- ANGIOGENESIS
  - WALKER CARCINOMA
    - NON-HISTONE PROTEIN, RAT, 2973

- ANTIBODY
  - ACTIVITY
    - MYELOMA GLOBULINS, HUMAN, REVIEW, 2420*
  - ANTI-TUMOR
    - MACROPHAGE TUMOR CELL INTERACTION, MOUSE, RAT, 2750
  - ANTIMEMBRANE
    - SV40-TRANSFORMED CELLS, HAMSTER, 2854*
  - BLOOD-GROUP-A-LIKE ANTIGEN
    - CULTURED TUMOR CELLS, HUMAN, 2850*
  - CELL MEMBRANE ALTERATION
    - METABOLIZING TARGET CELL, MOUSE, 2715
  - CERVICAL CARCINOMA
    - HUMAN, 2722
  - IMMUNOSUPPRESSION
    - REVIEW, 2760
  - ONCORNAVIRUS-ASSOCIATED ANTIGEN
    - LEUKEMIC CAT, 2730
  - PAUL-BUNNELL
    - REACTIVE HUMAN ANTIGEN, 2823*
  - RESPONSE
    - CERVICAL CARCINOMA PATIENTS, 2803*
  - TUMOR-SPECIFIC TRANSPLANTATION ANTIGEN
    - RAT, 2705
  - VIRUS, EPSTEIN-BARR
    - BACILLUS CALMETTE-GUERIN INNOCULATION, 2727
    - BURKITT'S LYMPHOMA PATIENTS, 2727
    - IRRADIATED TUMOR CELL TREATMENT, 2727
  - VIRUS, HERPES 1
    - HUMAN SERA, REVIEW, 2445*
  - VIRUS, HERPES 2
    - HUMAN SERA, REVIEW, 2445*
  - VIRUS, MAMMARY TUMOR
    - OCCURRENCE, NATURALLY INFECTED MICE, 2719
  - VIRUS, POLYOMA
    - TUMOR REJECTION, MOUSE, 2725

- ANTIGEN
  - ABH(O)
    - BENIGN AND MALIGNANT BREAST LESIONS, 2729
  - BLOOD-GROUP-A-LIKE
    - CULTURED TUMOR CELLS, HUMAN, 2850*
  - CARCINOEMBRYONIC
    - BLOOD GROUP SUBSTANCES, ADENOCARCINOMA, 2747
    - COMPARISON OF METHODS, 2851*
    - GASTRIC CARCINOMA, LIVER METASTASIS, HUMAN, 2789*
    - HUMAN, REVIEW, 2867*
    - MUCUS-PRODUCING CELLS, 2783*
    - NON-DIGESTIVE CARCINOMAS, HUMAN, 2700
    - TEST, CLINICAL EVALUATION, 2802*
    - TUMOR, FETAL GUT, MECONIUM, NORMAL COLON, 2826*
  - CARCINOGMBRYONIC
    - PH-DEPENDENT CHANGES IN COMPOSITION, 2862*
  - CELL-SURFACE
    - CERVICAL CARCINOMA, HUMAN, 2722
    - DIBUTYRYL CYCLIC-AMP EFFECT, CULTURED CELLS, MOUSE, 2800*
    - LYMPHOCYTE, MOUSE, 2698
    - SV40-TRANSFORMED CELL LINE, HAMSTER, 2733
    - SV40-TRANSFORMED CELL LINE, RABBIT, HAMSTER, 2731
    - THEOPHYLLINE EFFECT, CULTURED CELLS, MOUSE, 2800*
  - EMBRYONAL
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED FIBROSARCOMA, MOUSE, 2562*
  - FETAL
    - CARCINOMA PROPENSITY, HUMAN, 2704
    - CROSS-REACTIVITY WITH TUMOR-SPECIFIC TRANSPLANTATION ANTIGEN, 2745
    - MOUSE, 2745
  - GROSS VIRUS-SPECIFIC
    - BIOCHEMICAL ANALYSIS, 2670*
  - H-2
    - CELL-MEDIATED LYMPHOLYSIS, MOUSE LYMPHOCYTE, 2787*
  - HEPATITIS-ASSOCIATED
    - BURKITT'S LYMPHOMA, 2772*
  - HL-A
    - SOLID TUMORS, HUMAN, 2885*
  - HL-A SYSTEM
    - CLINICAL SIGNIFICANCE, 2872*
  - HUMAN THYMUS-LYMPHOID TISSUE
    - ROSETTE FORMATION, HUMAN, 2740
  - LEUKEMIA
    - HUMAN, REVIEW, 2409
    - ONCORNAVIRUS-ASSOCIATED ANTIBODY, CAT, 2730
    - REVIEW, 2425*
  - LEUKEMIA-ASSOCIATED
    - HUMAN, 2832*
  - MEMBRANE
    - SERUM, SV40 TUMOR-BEARING HAMSTER, 2854*
    - SV40-TRANSFORMED CELLS, HAMSTER, 2855*
    - THYMUS CELLS, RABBIT, 2808*
  - MEMBRANE, TUMOR-SPECIFIC
    - SERUM, CHEMICALLY-INDUCED SARCOMA,

    - RAT, 2890*
- NON-H-2
    - ERYTHROPOIESIS INHIBITION TECHNIQUE, 2799*
- NONVIRION
    - HERPES SIMPLEX VIRUS, CELL CULTURE, 2751
- OVARIAN CARCINOMA
    - HUMAN, 2827*
- PAUL-BUNNELL ANTIBODY-REACTIVE
    - HUMAN, 2823*
- RECEPTORS
    - B AND T LYMPHOCYTES, 2728
    - IMMUNOGLOBULIN CLASS SHIFT, MOUSE, 2708
- RECOGNITION SPECIFICITY
    - CELLULAR AND HUMORAL IMMUNE RESPONSE, 2743
- SPECIES-SPECIFIC
    - INTERSPECIFIC SOMATIC HYBRID CELLS, 2879*
- SQUAMOUS CELL CARCINOMA
    - UREA, MOUSE, 2703
- T
    - PROPERTIES, SV40-INFECTED AND TRANSFORMED CELLS, ANIMAL, HUMAN, 2858*
- TL
    - PAPAIN-DIGESTED MEMBRANE, THYMOCYTE, MOUSE, 2790*
- TRANSPLANTATION
    - CELLULAR IMMUNITY, REVIEW, 2430*
    - SARCOMA, SYNGENEIC, MOUSE, 2724
- TUMOR-ASSOCIATED
    - CELLULAR IMMUNITY, HUMAN, REVIEW, 2414
    - NEUROGENIC SARCOMA CELL LINE, HUMAN, 2881*
- TUMOR-SPECIFIC
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED FIBROSARCOMA, MOUSE, 2562*
    - INTERSPECIFIC SOMATIC HYBRID CELLS, 2879*
    - SERUM, AMINOAZO DYE-INDUCED HEPATOMA, RAT, 2756
- TUMOR-SPECIFIC TRANSPLANTATION
    - BLOOD, LYMPH, RAT, 2705
    - LYMPHOMA, FRIEND VIRUS COMPLEX, 2663*
    - RETICULUM CELL SARCOMA, FRIEND VIRUS COMPLEX, 2663*
- VIRUS, C-TYPE
    - NORMAL CELLS AND TISSUES, MOUSE, 2859*
- VIRUS, EPSTEIN-BARR
    - LYMPHOBLASTOID CELL LINES, HUMAN, 2759
    - NASOPHARYNGEAL CARCINOMA, BIOPSIES, 2710
- VIRUS, HERPES SIMPLEX
    - ORAL AND GENITAL ORIGEN, REVIEW, 2444*

ANTIGEN BINDING
- LYMPHOID CELLS
    - HIGH, LOW RESPONDER MICE, 2791*
    - IMMUNE MICE, 2777*
- PLASMACYTOMA CELLS
    - MOUSE, 2776*

ANTIGEN, CELLULAR
- SV40 VIRUS-TRANSFORMED FIBROBLASTS
    - SERUM, HAMSTER, 2768*

ANTIGEN, SOLUBLE
- TUMOR, HUMAN
    - HYPERSENSITIVITY, DELAYED CUTANEOUS, 2773*
    - LYMPHOCYTE STIMULATION, 2773*

ANTIGEN, THETA
- CULTURED LYMPHOMA CELLS
    - MOUSE, 2820*

ANTIGENIC COMPETITION
- EFFECT OF BACTERIAL ADJUVANT
    - MOUSE, 2875*

ANTIOXIDANTS
- STOMACH CANCER MORTALITY
    - REVIEW, 2431*

ANTISERUM
- LYMPHOCYTE
    - HELA TUMOR SURVIVAL, MOUSE, 2847*
- VIRUS, POLYOMA
    - ANTIGEN, MOUSE, 2725

ANTITHYMOCYTE GLOBULIN
- IN VITRO TEST
    - TUMORIGENIC POTENTIAL OF CELLS FOR MAN, 2746

ARENE OXIDE
- METABOLISM
    - NIH SHIFT, REVIEW, 2405

ARSENIC
- BRONCHIAL CARCINOMA
    - TUMOR DOUBLING TIME, HUMAN, 2580*
- CARCINOGENESIS
    - TUMOR DOUBLING TIME, HUMAN, 2580*

ARYL HYDROCARBON HYDROXYLASE
- 3-METHYLCHOLANTHRENE-INDUCED TUMORS
    - MOUSE, 2483

ARYLAMINE ACETYL-TRANSFERASE
- LIVER
    - HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503

ASBESTOS
- ENVIRONMENTAL HAZARD
    - REVIEW, 2427*
- LUNG CANCER
    - REVIEW, 2919
- MESOTHELIOMA
    - OCCURRENCE, NETHERLANDS, 2915
    - REVIEW, 2919
    - ULTRASTRUCTURE, 2923
- OCCUPATIONAL HAZARD
    - ASSESSMENT AND RECOMMENDATIONS, 2558*
    - MESOTHELIOMA, MORTALITY STUDY, 2928
    - REVIEW, 2427*

- AUTOSENSITIZATION
  - REGULATION
    - LYMPHOCYTE, T CELL, MOUSE, RAT, 2775*
- AZATHIOPRINE
  - IMMUNOSUPPRESSION
    - NEPHROTIC SYNDROME, MALIGNANCY, CASE REPORT, 2830*
- BACILLUS CALMETTE-GUERIN
  - HYPERSENSITIVITY
    - EFFECT OF CORTISONE ACETATE, LUNG, RABBIT, 2876*
    - GRANULA FORMATION, LUNG, RABBIT, 2876*
  - IMMUNE RESPONSE
    - BURKITT'S LYMPHOMA, EPSTEIN-BARR VIRUS, 2727
    - CANCER PATIENTS, 2849*
    - SARCOMA, MOUSE, 2522
  - IMMUNITY, CELL-MEDIATED
    - HUMAN TUMORS, 2817*
  - IMMUNOGLOBULIN IGA
    - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
  - IMMUNOGLOBULIN IGG
    - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
  - IMMUNOGLOBULIN IGM
    - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
  - TUMOR REGRESSION
    - LYMPHOCYTE ROLE, MOUSE, 2522
- BASIC LEAD ACETATE
  - KIDNEY TUMOR
    - RAT, 2576*
  - URINARY BLADDER TUMOR
    - RAT, 2576*
- BENZENE
  - SKIN
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
  - SPLEEN
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
- BENZO(A)PYRENE
  - CYCLIC AMP ACCUMULATION
    - SKIN, MOUSE, 2490
  - IODINE-INDUCED FREE RADICALS
    - ELECTRON PARAMAGNETIC RESONANCE STUDIES, 2542*
- BONE
  - STRONTIUM 90-INDUCED TUMORS
    - BEAGLE, 2585
- BONE MARROW
  - NEOPLASIA
    - MONOSOMY, CLINICAL STUDY, 2989*
  - PROLIFERATIVE DISEASES
    - LEUKOCYTE ANOMALIES, HUMAN, 2990*
  - RADIATION, X-RAY
    - REPOPULATING ABILITY, MOUSE, 2605*
- BONE TUMOR
  - STRONTIUM 90
    - ESTROGEN EFFECT MOUSE, 2587
- BRAIN TUMOR
  - INCIDENCE
    - EGYPT, 2943*
    - IBADAN, NIGERIA, 2944*
- BREAST
  - BENIGN AND MALIGNANT LESIONS
    - ABH(O) ANTIGENS, 2729
  - CARCINOMA, MALE
    - SEX CHROMATIN, CLINICAL STUDY, 2953
- 5-BROMO-2-DEOXYURIDINE
  - HEPATOMA
    - FILAMENTOUS A PARTICLES, RAT, 2509
- 7-BROMOMETHYLBENZ(A)ANTHRACENE
  - DNA REPAIR SYNTHESIS
    - HUMAN DIPLOID FIBROBLASTS, 2482
- BRONCHIAL CARCINOMA
  - ARSENIC
    - TUMOR DOUBLING TIME, HUMAN, 2580*
  - CARCINOEMBRYONIC ANTIGEN
    - HUMAN, 2700
- BRONCHIAL MUCOSA
  - PROTEIN
    - AGAROSE-GEL ELECTROPHORESIS, 2540*
- BRONCHOGENIC CARCINOMA
  - PROTEIN
    - AGAROSE-GEL ELECTROPHORESIS, 2540*
- BRONCHOPULMONARY CARCINOMA
  - IMMUNOGLOBULIN IGA
    - SALIVA, HUMAN, 2782*
- BURKITT'S LYMPHOMA
  - ANTIGEN, HEPATITIS-ASSOCIATED, 2772*
  - GLUCOSE-6-PHOSPHATE DEHYDROGENASE
    - CELL ORIGIN MARKER, 2754
  - IMMUNE RESPONSE
    - IMMUNE STIMULANT TREATMENT, 2822*
  - IMMUNOGLOBULIN IGM
    - CELL ORIGIN MARKER, 2754
  - INCIDENCE
    - KENYA, 2940*
  - NEUROLOGICAL MANIFESTATIONS
    - GHANA, 2675*
- N-BUTYL-N(4-HYDROXYBUTYL)NITROSAMINE
  - KIDNEY TUMOR
    - RAT, 2576*
  - URINARY BLADDER TUMOR
    - RAT, 2576*
- BUTYLATED HYDROXYANISOLE
  - STOMACH CANCER MORTALITY
    - REVIEW, 2431*
- BUTYLATED HYDROXYTOLUENE
  - STOMACH CANCER MORTALITY
    - REVIEW, 2431*
- CALCIUM

- METABOLISM
  - TUMOR, MOUSE, 2952
- ROUS SARCOMA VIRUS PROLIFERATION
  - FIBROBLASTS, CHICKEN, 2640

- CANCER
  - EVOLUTION OF SPECIES
    - HYPOTHESIS, REVIEW, 2473*
  - INCIDENCE
    - FIJI, 2929

- CARBON TETRACHLORIDE
  - HEPATOTOXICITY
    - EFFECT OF 3-METHYLCHOLANTHRENE, RAT, 2567*
    - EFFECT OF PHENOBARBITOL, RAT, 2567*
  - MAMMARY CARCINOMA
    - GLUCURONIC ACID LEVELS, RAT, 2568*

- CARCINOGEN, CHEMICAL
  - CANCER PROPHYLAXIS
    - USSR, REVIEW, 2467*
  - HOST-MEDIATED ASSAY
    - HAMSTER, 2497
  - HOST-MEDIATED IN VIVO-IN VITRO ASSAY
    - REVIEW, 2552*
  - IDENTIFICATION
    - MAMMALIAN CELLS, 2578*
  - IRRITANTS
    - REVIEW, 2447*
  - METABOLISM
    - NIH SHIFT, REVIEW, 2405
  - PRESENCE IN FOOD
    - REVIEW, 2426*
  - STANDARDIZATION
    - REVIEW, 2475*

- CATHEPSIN
  - ACTIVITY
    - REGRESSION, ESTRONE-INDUCED MAMMARY TUMOR, RAT, 2551*

- CELL
  - ADRENAL CORTEX
    - STIMULATION, 2-ACETYLAMINOFLUORENE, RAT, 2534
    - STIMULATION, NITROSAMINES, RAT, 2534
  - B-CELL
    - ANTITUMOR ACTIVITY, 2673*
  - GROWTH
    - MEMBRANE SITE MOBILITY, 2758
  - MEMORY
    - ENRICHMENT, IMMUNE SPLEEN CELLS, MOUSE, 2807*
  - ROSETTE-FORMING
    - NEURAMINADASE EFFECT ON T, B CELLS, HUMAN, 2841*
  - SURFACE C1 INACTIVATOR ACTIVITY
    - CARCINOMA, HUMAN, 2709
  - SURFACE CHANGE
    - HEPATOCARCINOMA, RAT, 2972
  - -VIRUS INTERACTIONS
    - REVIEW, 2406

- CELL CULTURE
  - PROGRESSION
    - BIOLOGIC PROPERTIES, 2992*

- CELL DENSITY
  - GLYCOLIPID
    - TRANSFORMED CELLS, HAMSTER, 2956

- CELL DIVISION
  - TUMOR
    - CIRCADIAN RHYTHM, 2950*

- CELL MEMBRANE
  - ALTERATION BY ANTIBODY
    - METABOLIZING TARGET CELL, MOUSE, 2715
  - IMMUNOGLOBULIN SURFACE ANTIGEN
    - MYELOMA, MOUSE, 2737
  - LESION, COMPLEMENT-INDUCED
    - CHARACTERIZATION, 2806*
  - SOLUBLE TL ANTIGEN
    - THYMOCYTE, MOUSE, 2790*

- CELL PROLIFERATION
  - ENVIRONMENTAL PH EFFECT
    - MAMMALIAN, HUMAN, 2984
  - KINETICS
    - CHLOROLEUKEMIA, RAT, 2986

- CELLOPHANE
  - -INDUCED PIGMENTED TUMORS
    - EYE, RAT, 2572*

- CELLULAR ANTIGEN, SERUM, HAMSTER
  - VIRUS, SV40
    - TRANSFORMED FIBROBLASTS, 2768*

- CERVICAL CARCINOMA
  - AGE, TIME, COHORT FACTORS
    - ENGLAND, WALES, 2918
  - ANTIBODY RESPONSE, 2803*
  - CYTOGENETIC STUDIES
    - REVIEW, 2453*
  - CYTOHISTOPATHOLOGY
    - REVIEW, 2448*
  - EPIDEMIOLOGY
    - HUMAN, REVIEW, 2407
  - EXPERIMENTAL STUDIES
    - REVIEW, 2438*
  - GENERAL ASPECTS
    - REVIEW, 2439*
  - PRECANCEROUS CONDITIONS
    - KINETICS, HUMAN, 2910*
  - SCREENING PROGRAM
    - CANADA, ENGLAND, WALES, 2930
    - ONTARIO, BRITISH COLUMBIA, 2935
  - TUMOR-SPECIFIC ANTIBODIES
    - SERUM, HUMAN, 2722
  - VIRUS, HERPES
    - REVIEW, 2437*
  - VIRUS, HERPES 2
    - HUMAN, REVIEW, 2411
    - NUCLEIC ACID HYBRIDIZATION, HUMAN, REVIEW, 2417
    - SEROEPIDEMIOLOGIC STUDIES, 2917

- CERVIX
  - ESTROGEN
    - 3H-THYMIDINE UPTAKE, MOUSE, 2530
  - 20-METHYCHOLANTHRENE
    - ULTRASTRUCTURE, CASTRATION, RAT, 2577*
  - 20-METHYLCHOLANTHRENE

    - 3H-THYMIDINE UPTAKE, MOUSE, 2530

- CHALONE
  - CANCER
    - REVIEW, 2457*

- CHEMOTACTIC FACTOR
  - MEASUREMENT
    - POLYMORPHONUCLEAR LEUKOCYTES, HORSE, HUMAN, RABBIT, 2778*

- CHILDREN
  - CANCER
    - GENETICS, REVIEW, 2477*

- CHLOROTRIANISENE
  - SUPPRESSION OF DMBA-TUMOR INDUCTION
    - RAT, 2519

- CHOLESTEROL SYNTHESIS
  - LIVER
    - 2-ACEYLAMINOFLUORENE-FED RATS, 2501

- CHORIOCARCINOMA
  - OCCURRENCE, TREATMENT
    - DENMARK, 2933

- CHROMATIN
  - HEPATOMA
    - RAT, 2964

- CHROMATIN, SEX
  - BREAST CARCINOMA
    - CLINICAL STUDY, MALES, 2953
  - MAMMARY CARCINOMA
    - CLINICAL STUDY, 2998*

- CHROMOSOME
  - ACROCENTRIC
    - MENINGIOMA, HUMAN, 2971
  - MALIGNANCY
    - L CELL-TUMOR CELL HYBRID, MOUSE, 2958
  - TRANSLOCATION
    - VIRUS-FUSED CELLS, HAMSTER, 2978
  - VIRUS-INDUCED CYTOGENETIC CHANGES
    - REVIEW, 2428*

- CHROMOSOME ABERRATIONS
  - BONE MARROW NEOPLASIA
    - CLINICAL STUDY, 2989*
  - CANCER
    - CELL CULTURE, 2957
  - EPSTEIN-BARR VIRUS INFECTION
    - LYMPHOBLASTOID CELL LINE, 2619
  - ETHYLENIMINE
    - REPAIR ACTIVITY, MAMMALS, 2596*
  - LEUKEMIA, CHRONIC MYELOGENOUS
    - CLINICAL STUDY, 2902
  - LSD
    - RABBIT, 2520
  - D-METHAMPHETAMINE-HCL
    - RABBIT, 2520
  - RADIATION, UV
    - REPAIR ACTIVITY, MAMMALS, 2596*
  - RADIATION, X-RAY
    - LEUKOCYTES, SPERMATOGONIAL CELLS, MOUSE, HAMSTER, 2609*

- COCARCINOGENICITY
  - TOBACCO SMOKE CONDENSATE
    - SKIN, MOUSE, 2535

- COLON CARCINOMA
  - 1,2-DIMETHYLHYDRAZINE
    - EXPERIMENTAL MODEL, RAT, 2560*
  - EPIDEMIOLOGY
    - FECAL CONSTITUENTS OF POPULATIONS, 2937*
    - REVIEW, 2476*
  - INCIDENCE
    - FECAL CONSTITUENTS OF POPULATIONS, 2937*
  - NEPHROSIS
    - CARCINOEMBRYONIC ANTIGEN-ANTIBODY COMPLEXES, CASE REPORT, 2707

- COMPLEMENT
  - C3 RECEPTOR
    - LYMPHOCYTE B-CELL ACTIVATION, 2793*

- CONCANAVALIN A
  - AGGLUTINATION, TRANSFORMED CELL
    - ENHANCED BY CYCLIC AMP, OVARY CELLS, HAMSTER, 2616
  - BINDING SITES, PLASMA MEMBRANE
    - NORMAL, TUMOR, TRANSFORMED CELLS, 2955
  - EFFECT ON PERIPHERAL LYMPHOCYTES
    - CHRONIC LYMPHOCYTIC LEUKEMIA, 2856*
  - LYMPHOCYTE TRANSFORMATION
    - HUMAN, 2860*

- CORTISONE ACETATE
  - BCG-INDUCED DELAYED HYPERSENSITIVITY
    - GRANULA FORMATION, LUNG, RABBIT, 2876*

- CYCLIC AMP
  - EHRLICH ASCITES TUMOR
    - CARCINOSTATIC ACTIVITY, MOUSE, 2983
  - ISOPROTERENOL-DEPENDENT ACCUMULATION
    - BENZO(A)PYRENE, SKIN, MOUSE, 2490

- CYCLOPHOSPHAMIDE
  - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMOR
    - IMMUNOSUPPRESSION, RAT, 2531

- CYTOCHROME P-420
  - PROPERTIES
    - 3-METHYLCHOLANTHRENE-TREATED RATS, 2549*

- CYTOCHROME P-450
  - N-HYDROXYLATION OF 2-ACETYLAMINOFLUORENE
    - MOUSE, HAMSTER, 2548*

- CYTOTOXICITY
  - "ALTERNARIA" METABOLITES
    - MOUSE, 2570*
  - ANTIGEN-TRANSFORMED LYMPHOCYTE
    - NONSPECIFIC EFFECT, 2794*
  - CELL-MEDIATED

    - ALLOGENEIC LYMPHOMA CELLS, MOUSE, 2833*
  - LYMPHOCYTE
    - SOLUBLE TUMOR MEMBRANE FRACTION INHIBITION, 2852*
  - MACROPHAGE
    - SYNGENEIC, ALLOGENEIC ERYTHROCYTES, MOUSE, 2785*

- DDT
  - TOXICITY
    - LONG TERM STUDIES, MOUSE, 2508

- DEOXYRIBONUCLEASE
  - ACTIVITY
    - DIETHYLNITROSAMINE CARCINOGENESIS, RAT LIVER, 2487
  - AFLATOXIN B1
    - LYSOSOMES, RAT LIVER, 2547*
  - AFLATOXIN B2
    - LYSOSOMES, RAT LIVER, 2547*

- DEUTERIUM
  - ISOTOPE EFFECT
    - DIMETHYLNITROSAMINE CARCINOGENESIS, LIVER, RAT, 2513

- 2,6-DI-TERT-BUTYL-4-METHYLPHENOL(IONOL)
  - ANTICARCINOGENESIS
    - FREE RADICAL INHIBITION, 2559*

- N-DIBUTYLNITROSAMINE
  - CARCINOGENESIS
    - HAMSTER, 2528

- DIBUTYLNITROSOAMINE
  - CARCINOGENESIS
    - RAT, 2565*
  - URINARY BLADDER TUMOR
    - RAT, 2565*

- DIBUTYRYL CYCLIC AMP
  - CELL-SURFACE ANTIGEN
    - CULTURED CELLS, MOUSE, 2800*

- DIELDRIN
  - TOXICITY
    - LONG TERM STUDIES, MOUSE, 2507, 2508

- DIETHYLAMINOETHYL-DEXTRAN
  - VIRUS, MURINE LEUKEMIA
    - POTENTIATION, MOUSE, 2635
  - VIRUS, RAUSCHER LEUKEMIA
    - POTENTIATION, MOUSE, 2635

- DIETHYLNITROSAMINE
  - CARCINOGENESIS
    - DEOXYRIBONUCLEASE ACTIVITY, RAT LIVER, 2487
    - BETA-D-GALACTOSIDASE ACTIVITY, RAT LIVER, 2487
    - INHIBITION BY HOMOLOGOUS THYMUS, RAT LIVER, 2496
  - IMMUNOSUPPRESSION
    - GUINEA PIG, 2761
  - INDUCED HEPATOMAS
    - IMMUNOLOGIC REJECTION, GUINEA PIG, 2699

  - PULMONARY ADENOMA
    - ULTRASTRUCTURE, HAMSTER, 2573*
  - TUMOR INDUCTION
    - GUINEA PIG, 2488

- DIETHYLSTILBESTROL
  - ADENOCARCINOMA, CLEAR CELL
    - MOTHER-DAUGHTER, CASE REPORT, 2987*
  - GLUCURONYLTRANSFERASE ACTIVITY
    - LIVER, HAMSTER, RAT, 2523
  - MESOTHELIOMA INDUCTION
    - UTERUS, MONKEY, 2529

- 9,10-DIMETHYL-1,2-BENZANTHRACENE
  - DNA SYNTHESIS
    - BUCCAL POUCH, HAMSTER, 2533

- DIMETHYL SULFOXIDE
  - HEPATOMA
    - FILAMENTOUS A PARTICLES, RAT, 2509

- 4-DIMETHYLAMINOBIBENZYL
  - METABOLISM
    - RAT, 2493

- TRANS-4-DIMETHYLAMINOSTILBENE
  - METABOLISM
    - RAT, 2493

- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - CARCINOGENIC RISK DETECTION
    - SENSITIVITY OF FEEDING TESTS, RAT, 2492
  - FIBROSARCOMA
    - EMBRYONAL ANTIGENS, MOUSE, 2562*
    - TUMOR-SPECIFIC ANTIGENS, MOUSE, 2562*
  - IMMUNOSUPPRESSION
    - MECHANISM, MOUSE, 2525
  - MAMMARY TUMOR
    - EFFECT OF DIMETHYLBENZYLDESMETHYLRIFAMPICIN, RAT, 2537*
    - IMMUNOSUPPRESSION, ANTI-LYMPHOCYTE SERUM, RAT, 2527
    - IMMUNOSUPPRESSION, CYCLOPHOSPHAMIDE, RAT, 2531
    - MAST CELLS, RAT, 2517

- 7,12 DIMETHYLBENZ(A)ANTHRACENE
  - MAMMARY TUMOR
    - PROLACTIN LEVEL, PLASMA, RAT, 2512

- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - MAMMARY TUMOR
    - RAT, 2744
    - SUPPERSSION BY PREGNANCY, RAT, 2519
    - SUPPRESSION BY CHLOROTRIANISENE, RAT, 2519
  - OVARIAN TUMOR, MALIGNANT
    - RAT, 2494

- DIMETHYLBENZYLDESMETHYLRIFAMPICIN
  - EFFECT ON CHEMICALLY INDUCED MAMMARY TUMORS
    - RAT, 2537*

- 1,2-DIMETHYLHYDRAZINE
  - COLON CARCINOMA
    - EXPERIMENTAL MODEL, RAT, 2560*
  - RECTAL CARCINOMA
    - EXPERIMENTAL MODEL, RAT, 2560*

- DIMETHYLNITROSAMINE
  - CARCINOGENESIS
    - DEUTERIUM ISOTOPE EFFECT, LIVER, RAT, 2513
    - FERRIC OXIDE SYNERGISM, LIVER, HAMSTER, 2505
  - HEPATOTOXICITY
    - DOG, 2539*
  - KIDNEY
    - EMBRYONAL TISSUE, MOUSE, 2579*
  - METABOLISM
    - AMPHIBIANS, FISH, IN VITRO, 2543*
  - MUTAGENESIS
    - METABOLISM, RAT LIVER MICROSOMES, 2555*

- DIMETHYNITROSAMINE
  - DNA DAMAGE
    - LIVER, RAT, 2506

- 4,4-DINITROBIPHENYL
  - METABOLISM
    - GUINEA PIG, RAT, 2526

- DIPROPYLNITROSAMINE
  - RESPIRATORY SYSTEM TUMORS
    - HAMSTER, 2485

- DIVERTICULOSIS
  - INTESTINE, LARGE
    - HAWAII JAPANESE, AUTOPSY STUDY, 2914*

- DNA
  - BINDING, COVALENT
    - HOST CELL-VIRUS INTEGRATION, 2624
  - CONTENT
    - IRRADIATED RHABDOMYOSARCOMA, RAT, 2599*
  - DAMAGE
    - N-ACETOXY-2-ACETYLAMINOFLUORENE, HUMAN SKIN FIBROBLASTS, 2499
    - 2-ACETYLAMINOFLUORENE, HUMAN SKIN FIBROBLASTS, 2499
    - DIMETHYLNITROSAMINE, LIVER, RAT, 2506
    - N-HYDROXY-2-ACETYLAMINOFLUORENE, HUMAN SKIN FIBROBLASTS, 2499
    - MEASUREMENT IN VIVO, RAT LIVER, 2500
    - N-METHYL-N-NITROSOUREA, LIVER, RAT, 2506
    - N-METHYL-N-NITROSOURETHAN, LIVER, RAT, 2506
    - METHYLAZOXYMETHANOL ACETATE, LIVER, RAT, 2506
  - EPITHELIUM, TRANSFORMATION
    - QUANTITATIVE STUDY, 2899
  - RADIATION, GAMMA
    - INFRARED SPECTROSCOPY, CALF, 2584
  - SARCOMA, ROUS CHICKEN
    - INTERFERON INDUCTION, ALLANTOIC FLUID, CHICK EMBRYO, 2869*
  - SV40-TRANSFORMATION
    - REVERTANT CELL LINES, MOUSE, 2659
  - VIRUS, EPSTEIN-BARR
    - LYMPHOBLASTOID CELL LINES, HUMAN, 2759
  - VIRUS, SV40
    - HOST CELL-VIRUS INTEGRATION, 2624

- DNA POLYMERASE
  - FORMATION
    - ENHANCED BY CYCLIC AMP, OVARY CELLS, HAMSTER, 2616
  - HEPATOMA, MORRIS
    - RAT, 2963
  - LEUKEMIA
    - PLASMA, GUINEA PIG, 2982
  - RNA-DEPENDENT
    - MAMMARY TUMOR VIRUS, MOUSE, 2660
  - RNA TRANSCRIPTION
    - LEUKEMIC CELLS, HUMAN, 2622
  - VIRUS, B-TYPE
    - MAMMARY TUMOR, MOUSE, 2617
  - VIRUS, MAMMARY TUMOR
    - MOUSE, 2617
  - VIRUS, RNA TUMOR
    - TEMPLATE PRIMER, CHICKEN, 2639

- DNA REPAIR SYNTHESIS
  - N-ACETOXY-2-ACETYLAMINOFLUORENE
    - HUMAN DIPLOID FIBROBLASTS, 2482
  - 7-BROMOMETHYLBENZ(A)ANTHRACENE
    - HUMAN DIPLOID FIBROBLASTS, 2482

- DNA REPLICATION
  - VIRUS, ADENO 2
    - KB CELLS, 2650

- DNA SYNTHESIS
  - 9,10-DIMETHYL-1,2-BENZANTHRACENE
    - BUCCAL POUCH, HAMSTER, 2533
  - EPSTEIN-BARR VIRUS ACTIVATION
    - LYMPHOBLASTOID CELLS, HUMAN, 2625
  - ERYTHROPOIETIC SERUM
    - 20-METHYLCHOLANTHRENE, BENZENE, SKIN, SPLEEN, MOUSE, 2538*, 2574*
  - HELA CELLS
    - ISOLATED NUCLEI, 2975
  - INHIBITION
    - INTERFERON, CHICK, 2618
  - LEUKEMIA, PLASMA CELL
    - CYTOPHOTOMETRY, AUTORADIOGRAPHY, 2948*
  - LYMPHOCYTES
    - INHIBITION BY TUMOR CELLS, HUMAN, 2695
  - RADIATION, X-RAY
    - LYMPHOMA, RAT, 2611*
  - RNA-DIRECTED
    - LEUKEMIC CELLS, HUMAN, 2622
    - LIGHT CHAIN-PRODUCING MYELOMA TUMOR, 2816*
  - TEMPERATURE SENSITIVE VIRUS
    - FROG, 2630

- DNASE
  - AFLATOXIN B1
    - LIVER, RAT, MOUSE, 2489
    - LYSOSOMES, RAT LIVER, 2547*

- AFLATOXIN B2
    - LYSOSOMES, RAT LIVER, 2547*
- 2-METHYL-N,N-DIMETHYLAMINOAZOBENZENE
    - LIVER, RAT, MOUSE, 2489
- 3'-METHYL-N,N-DIMETHYLAMINOAZOBENZENE
    - LIVER, RAT, MOUSE, 2489

EHRLICH ASCITES TUMOR
- CYCLIC AMP
    - CARCINOSTATIC ACTIVITY, MOUSE, 2983
- RADIATION, X-RAY
    - LETHALITY ENHANCED BY AMINOHYDROXYTHYMINE HYDRAZINE, MOUSE, 2601*

EMPYEMA
- LUNG CANCER
    - REVIEW, 2449*

ENDOMETRIAL CARCINOMA
- CYTOGENETIC STUDIES
    - REVIEW, 2453*

ENDOREDUPLICATION
- 4-NITROQUINOLINE-1-OXIDE
    - CULTURED CELLS, HAMSTER, 2554*

ENOVID
- CARCINOGENESIS
    - MOUSE, 2502

ENVIRONMENTAL HAZARD
- ASBESTOS
    - REVIEW, 2427*
- TALC
    - REVIEW, 2427*

ENZYME
- CYTOCHEMISTRY
    - TUMORS, HUMAN, 2969
- CYTOPLASMIC
    - MAMMARY TUMOR, HUMAN, 2977
- HEXOSAMINIDASE ISOZYMES
    - HEPATOMA, RAT, 2954
- LACTATE DEHYDROGENASE ISOENZYME
    - MALIGNANT TUMORS, HUMAN, 2994*
- MUSCLE DIFFERENTIATION
    - CELL CULTURE, CHICKEN, 2644
- STEROID REDUCTASE
    - ADRENOCORTICAL CARCINOMA, RAT, 2965

EPIDEMIOLOGY
- CERVICAL CARCINOMA
    - HUMAN, REVIEW, 2407
- COLON CARCINOMA
    - REVIEW, 2476*
- LUNG CANCER
    - HUMAN, REVIEW, 2407
- PENILE CANCER
    - HUMAN, REVIEW, 2407
- RECTAL CARCINOMA
    - REVIEW, 2476*
- SKIN CANCER
    - HUMAN, REVIEW, 2407

EPIDERMOID CARCINOMA
- INCIDENCE
    - KENYA, 2940*

EPITHELIAL TUMOR
- DNA CONTENT
    - DIAGNOSTIC TOOL, 2899
- VIRUS-LIKE CYTOPLASMIC INCLUSIONS
    - ULTRASTRUCTURE, HUMAN, 2621

ERYTHROLEUKEMIA
- ERYTHROCYTE POPULATIONS
    - ADENYLATE KINASE DEFICIT, HEMOGLOBIN A2 INCREASE, HUMAN, 2810*

ERYTHROPOIESIS
- INHIBITION
    - NON-H-2 ANTIGEN-SENSITIZED LYMPHOCYTE-INJECTED, IRRADIATED MICE, 2799*

ERYTHROPOIETIC SERUM
- DNA SYNTHESIS
    - 20-METHYLCHOLANTHRENE, BENZENE, SKIN, SPLEEN, MOUSE, 2538*, 2574*

ESOPHAGEAL CANCER
- INCIDENCE
    - SOUTH AFRICA, BANTER, 2922

ESOPHAGUS
- SQUAMOUS CELL PAPILLOMA
    - DIBUTYLNITROSOAMINE, RAT, 2565*

ESTROGEN
- BONE TUMOR FORMATION
    - STRONTIUM 90 TREATED MOUSE, 2567
- CERVIX
    - 3H-THYMIDINE UPTAKE, MOUSE, 2530
- RECEPTORS, MAMMARY TUMOR
    - HUMAN, 2977

ESTRONE
- MAMMARY TUMOR
    - REGRESSION, ENZYME ACTIVITIES, RAT, 2551*

ETHIDIUM BROMIDE
- RESISTANCE
    - SV40-TRANSFORMED HAMSTER CELLS, 2687*

ETHMOID TUMOR
- OCCUPATIONAL HAZARD
    - WOOD WORKERS, 2575*

ETHYLENIMINE
- CHROMOSOME ABERRATIONS
    - REPAIR ACTIVITY, MAMMALS, 2596*

EWING'S SARCOMA
- GROWTH, TUMOR
    - POTENTIAL, CLINICAL STUDY, 2936*

EYE
- IRIS, MALIGNANT MYELOMA
    - ULTRASTRUCTURE, HUMAN, 2961
- PIGMENTED TUMORS
    - PLATINUM AND CELLOPHANE FILM-

        INDUCED, RAT, 2572*

FERRIC OXIDE
    DIMETHYLNITROSAMINE CARCINOGENESIS
        SYNERGISTIC EFFECT, LIVER, HAMSTER, 2505

ALPHA-FETOPROTEIN
    EXCRETION IN URINE
        PREGNANT RATS, HEPATOMA-BEARING RATS, 2865*
    GASTRIC CARCINOMA
        LIVER METASTASIS EXTRACT, HUMAN, 2789*
    HEPATOCARCINOGENESIS
        MOUSE, 2518
    HEPATOMA
        RAT, 2706
    PURIFICATION
        RABBIT, 2819*
    YOSHIDA ASCITES SARCOMA
        RAT, 2749

FIBROADENOMATOSIS
    GYNECOMASTASIA
        HISTOCHEMISTRY, HUMAN, 2900

FIBROBLAST
    TRANSFORMATION
        INHIBITION BY CHICK INTERFERON, 2618

FIBROSARCOMA
    7,12-DIMETHYLBENZ(A)ANTHRACENE
        EMBRYONAL ANTIGENS, MOUSE, 2562*
        TUMOR-SPECIFIC ANTIGENS, MOUSE, 2562*
    METHYLCHOLANTHRENE
        CONCOMITANT IMMUNITY, MOUSE, 2839*
    3-METHYLCHOLANTHRENE
        ARYL HYDROCARBON HYDROXYLASE, MOUSE, 2483
    PROTON SPIN-LATTICE RELAXATION
        MOUSE, 2981

N-2-FLUORENYLACETAMIDE
    CARCINOGENESIS
        ACETANILIDE, MOUSE, HAMSTER, RAT, 2504
        P-HYDROXYACETANILIDE, MOUSE, HAMSTER, RAT, 2504
    DERIVATIVES AND ANALOGS
        CARCINOGENESIS, PHYSICAL BASIS, 2524

N-2-FLUORENYLDIACETAMIDE
    THYROIDITIS, CHRONIC
        RAT, 2556*

FOLIC ACID
    ROUS SARCOMA VIRUS PROLIFERATION
        FIBROBLASTS, CHICKEN, 2640

FOOD
    CARCINOGENS, CHEMICAL
        REVIEW, 2426*

FREE RADICALS
    FORMATION
        DECOMPOSITION OF N-ACETOXY-N-ARYLACETAMIDES, 2550*
    INHIBITION
        2,6-DI-TERT-BUTYL-4-METHYLPHENOL (IONOL), ANTICARCINOGENESIS, 2559*
    IODINE-INDUCED
        BENZO(A)PYRENE, ELECTRON PARAMAGNETIC RESONANCE STUDIES, 2542*

BETA-D-GALACTOSIDASE
    ACTIVITY
        DIETHYLNITROSAMINE CARCINOGENESIS, RAT LIVER, 2487

GAMMAGLOBULIN
    MYELOMA
        MOUSE, 2697

GASTRIC ADENOCARCINOMA
    TRANSPLANTATION SITE PREIRRADIATION
        VASCULAR SPACE, MICE, 2591*

GASTRIC CARCINOMA
    ALPHA-FETOPROTEIN
        LIVER METASTASIS EXTRACT, HUMAN, 2789*

GASTROINTESTINAL TRACT
    CANCER
        EPIDEMIOLOGY, NEWFOUNDLAND, 2942*
        INCIDENCE, YUGOSLAVIA, REVIEW, 2462*
    MALIGNANT NEOPLASMS
        OCCURRENCE, WARSAW, POLAND, 2939*
    POLYPS
        RADIATION INDUCED, DOG, RAT, 2600*

GENETICS
    "CANCER" GENES
        ORIGIN, CARCINOGENIC VIRUSES, 2684*
    CHILDHOOD CANCER
        REVIEW, 2477*
    CONTROL OF SUSCEPTIBILITY
        ROUS SARCOMA VIRUS, FOWL, 2681*
    IMMUNE RESPONSE
        ACUTE LYMPHOCYTIC CHORIOMENINGITIS VIRUS, MOUSE, 2712
    ONCONOGENESIS
        CHANGES IN BLOOD GROUP SUBSTANCES, 2747
    SPLENOMEGALY
        FRIEND LEUKEMIA VIRUS, MOUSE, 2651

GAMMAGLOBULIN
    SYNTHESIS
        MYELOMA TUMORS, MOUSE, 2734

GLUCOSE-6-PHOSPHATE DEHYDROGENASE
    CELL ORIGIN MARKER
        BURKITT'S LYMPHOMA, 2754

GLUCURONIC ACID
    LEVELS
        MAMMARY CARCINOMA, RAT, 2568*

BETA-GLUCURONIDASE

- ACTIVITY
  - REGRESSION, ESTRONE-INDUCED MAMMARY TUMOR, RAT, 2551*

GLUCURONYLTRANSFERASE
- ACTIVITY
  - DIETHYLSTILBESTROL, LIVER, HAMSTER, RAT, 2523

GLYCOLIPID
- CELL DENSITY
  - TRANSFORMED CELLS, HAMSTER, 2956

GONADATROPHIN
- OVARIAN TUMORIGENESIS
  - STERILE MOUSE, 2912*

GRAFT-VS-HOST REACTION
- IMMUNOSUPPRESSION
  - LYMPHOMA, MOUSE, 2726
- VIRUS, LEUKEMIA
  - INTERFERON, MOUSE, 2674*

GRANULOCYTOPENIA
- LEUKEMIA, ACUTE
  - HUMORAL FACTORS, HUMAN, 2908*

GRANULOMA FORMATION
- BCG-INDUCED DELAYED HYPERSENSITIVITY
  - EFFECT OF CORTISONE ACETATE, LUNG, RABBIT, 2876*

GROWTH
- BONE MARROW CELLS
  - RAUSCHER LEUKEMIA VIRUS-INFECTED MICE, 2688*
- BURKITT SOMATIC CELLS
  - HYBRID, SELECTION, HUMAN CELL LINE, 2643
- CELL
  - MEMBRANE SITE MOBILITY, 2758
- TUMOR CELL POPULATIONS
  - PHOTOMETRIC DNA DETERMINATION, MOUSE, 2996*

GROWTH, TUMOR
- BIOLOGICAL FEATURES
  - REVIEW, 2466*
- CELL KINETICS
  - REVIEW, 2464*
- EWING'S SARCOMA
  - POTENTIAL, CLINICAL STUDY, 2936*
- MYELOMA, MULTIPLE
  - IMMUNOGLOBULIN SYNTHESIS, REVIEW, 2478*
- PHYTOHEMAGGLUTININ
  - FIBROSARCOMA-BEARING MICE, 2792*
- PROGRESSION
  - HISTOCHEMISTRY, ELECTRON MICROSCOPY, 2995*
- RIBOFLAVIN
  - CANCER, REVIEW, 2413

GYNECOLOGICAL TUMOR
- INCIDENCE
  - CHILDHOOD, ADOLESCENCE, REVIEW, 2471*

GYNECOMASTASIA
- FIBROADENOMATOSIS
  - HISTOCHEMISTRY, HUMAN, 2900

HARD PALATE CARCINOMA
- NICOTINE STOMATITIS
  - REVERSE SMOKERS, 2571*

HEART
- RHABDOMYOSARCOMA
  - ORTHO-TOLUIDINE TREATMENT, RAT, 2563*

HELA CELLS
- DNA SYNTHESIS
  - ISOLATED NUCLEI, 2975
- PHOTOMETRIC DNA DETERMINATION
  - CELL GROWTH, MOUSE, 2996*
- TUMORIGENICITY
  - IN VITRO TEST, ANTITHYMOCYTE GLOBULIN, MONKEY, 2746

HEPATOCARCINOGENESIS
- ALPHA-FETOPROTEIN
  - MOUSE, 2518

HEPATOCARCINOMA
- CELL SURFACE CHANGE
  - ELECTROPHORESIS, RAT, 2972

HEPATOMA
- ANTIGEN
  - SERUM, RAT, 2756
- 5-BROMO-2-DEOXYURIDINE
  - FILAMENTOUS A PARTICLES, RAT, 2509
- CHROMATIN
  - RAT, 2964
- CIRRHOSIS, ALCOHOLISM
  - INCIDENCE, BOSTON CITY HOSPITAL, 2924
- DIETHYLNITROSAMINE-INDUCED
  - IMMUNOLOGIC REJECTION, GUINEA PIG, 2699
- DIMETHYL SULFOXIDE
  - FILAMENTOUS A PARTICLES, RAT, 2509
- ALPHA-FETOPROTEIN
  - RAT, 2706
- HEXOSAMINIDASE
  - RAT, 2954
- (2-HYDROXYPROPYL)PROPYLNITROSAMINE
  - HAMSTER, 2485
- INCIDENCE
  - PAPUA NEW GUINEA, REVIEW, 2429*
- 3-METHYL-4-DIMETHYLAMINOAZOBENZENE
  - CARCINOGEN-BINDING PROTEIN, RAT, 2564*
- MITOCHONDRIA
  - OXIDATIVE PHOSPHORYLATION, RAT, 2979
- MYCOTOXIN
  - REVIEW, 2410
- (2-OXOPROPYL)PROPYLNITROSAMINE
  - HAMSTER, 2485
- PYRUVATE CARBOXYLASE
  - ENZYMATIC, IMMUNOLOGIC STUDY, RAT, 2720

HEPATOMA, MORRIS
- DNA POLYMERASE
  - RAT, 2963

HEPATOMA, NOVIKOFF
    FOLATE DEFICIENCY
        METHYL METABOLISM, RAT, 2970

HEPATOTOXICITY
    CARBON TETRACHLORIDE
        EFFECT OF 3-METHYLCHOLANTHRENE, RAT, 2567*
        EFFECT OF PHENOBARBITOL, RAT, 2567*
    DIMETHYLNITROSAMINE
        DOG, 2539*

BETA-HEXACHLOROCYCLOHEXANE
    TOXICITY
        LONG TERM STUDIES, MOUSE, 2508

GAMMA-HEXACHLOROCYCLOHEXANE
    TOXICITY
        LONG TERM STUDIES, MOUSE, 2508

HEXOSAMINIDASE
    HEPATOMA
        RAT, 2954

HODGKIN'S DISEASE
    ETIOLOGY
        REVIEW, 2461*
    HORIZONTAL TRANSMISSION
        EPIDEMIOLOGY, 2931
    IMMUNOLOGY
        REVIEW, 2460*
    INFECTIVE AGENTS
        REVIEW, 2418
    INITIAL SITE
        REVIEW, 2456*
    LYMPHOCYTE SURFACE IMMUNOGLOBULINS, 2838*
    MITOGEN, POKEWEED
        LYMPHOCYTE RESPONSE, 2844*
    PATHOGENESIS
        LYMPHOCYTE ISOLATION, 2913*

HORMONE
    ACTIVITY
        PITUITARY NEOPLASTIC CELLS, MOUSE, RAT, 2853*
    CARCINOGENESIS
        REVIEW, 2470*
    MAMMARY TUMOR
        RAT, 2744

HYBRIDIZATION
    NUCLEIC ACID
        HERPESVIRUS 2, CERVICAL CARCINOMA, HUMAN, REVIEW, 2417

HYDROCARBON, POLYCYCLIC
    IODINE-INDUCED FREE RADICALS
        ELECTRON PARAMAGNETIC RESONANCE STUDIES, 2542*

HYDROCARBON, POLYCYCLIC AROMATIC
    TUMORIGENESIS
        RAT, 2557*

HYDROGENATED TERPHENYL
    NUCLEAR REACTOR COOLANT
        OCCUPATIONAL HAZARD, 2491

N-HYDROXY-2-ACETAMIDOPHENANTHRENE
    REACTIONS OF ESTERS WITH CELLULAR NUCLEOPHILES
        FORMATION OF FREE RADICALS, 2550*

N-HYDROXY-4-ACETYLAMINOBIPHENYL
    MAMMARY GLAND
        TRANSACETYLASE ACTIVITY, RAT, 2479

N-HYDROXY-2-ACETYLAMINOFLUORENE
    DNA DAMAGE
        HUMAN SKIN FIBROBLASTS, 2499

N-HYDROXY-N-2-FLUORENYLACETAMIDE
    CARCINOGENESIS
        ACETANILIDE, MOUSE, HAMSTER, RAT, 2504
        P-HYDROXYACETANILIDE, MOUSE, HAMSTER, RAT, 2504

P-HYDROXYACETANILIDE
    N-2-FLUORENYLACETAMIDE CARCINOGENESIS
        MOUSE, HAMSTER, RAT, 2504
    N-HYDROXY-N-2-FLUORENYLACETAMIDE CARCINOGENESIS
        MOUSE, HAMSTER, RAT, 2504

4-HYDROXYAMINOQUINOLINE-1-OXIDE
    XERODERMA PIGMENTOSUM
        SENSITIVITY, HUMAN CELLS, 2481

(2-HYDROXYPROPYL)PROPYLNITROSAMINE
    HEPATOMA
        HAMSTER, 2485
    RESPIRATORY SYSTEM TUMORS
        HAMSTER, 2485

HYPERPLASIA
    PROSTATE
        ENZYME ACTIVITY, HUMAN, 2891
    THYMUS
        MAGNESIUM DEFICIENCY, RAT, 2752

HYPERSENSITIVITY
    BACILLUS CALMETTE-GUERIN
        EFFECT OF CORTISONE ACETATE, LUNG, RABBIT, 2876*
        GRANULA FORMATION, LUNG, RABBIT, 2876*
    DELAYED CUTANEOUS
        SOLUBLE ANTIGEN, TUMOR, HUMAN, 2773*

BETA2-MICROGLOBULIN
    IMMUNOGLOBULIN IGG
        LYMPHOID CELL LINES, HUMAN, 2857*

IMMUNE COMPLEX
    BINDING
        LYMPHOCYTE, MOUSE, 2798*
    TUMOR-SPECIFIC
        SERUM, AMINOAZO DYE-INDUCED HEPATOMA, RAT, 2756

IMMUNE RESPONSE
    BACILLUS CALMETTE-GUERIN
        BURKITT'S LYMPHOMA, EPSTEIN-BARR VIRUS, 2727
        SARCOMA, MOUSE, 2522

- CELL-MEDIATED
  - AGE EFFECT, MOUSE, 2868*
- CELLULAR AND HUMORAL
  - ANTIGEN RECOGNITION SPECIFICITY, 2743
- DELAYED HYPERSENSITIVITY
  - BCG EFFECT, CANCER PATIENTS, 2849*
- DIETHYLNITROSAMINE-INDUCED HEPATOMA
  - MACROPHAGES, BASOPHILIC LEUKOCYTES, GUINEA PIG, 2699
- ESSENTIAL AMINO ACID DEFICIENCY
  - TUMOR, MOUSE, 2696
- HUMORAL
  - CARCINOMA CELL RESISTANCE, SURFACE ACTIVITY, HUMAN, 2709
- IMMUNE STIMULANT TREATMENT
  - CHEMOTHERAPEUTICALLY-REGRESSED BURKITT'S LYMPHOMA PATIENT, 2822*
- IMMUNOGLOBULIN IGE
  - PRIMED SPLEEN CELL-TREATED, IRRADIATED MICE, 2774*
- INFLUENZA VACCINE
  - METASTATIC CARCINOMA, LYMPHOGRANULOMATOSIS, MULTIPLE MYELOMA, 2874*
- LYMPHOCYTE
  - AUTOCHTHONOUS TUMOR CELLS, HUMAN, 2738
- MER POTENTIATION
  - GUINEA PIG, 2795*
- MIXED TUMOR CHALLENGE
  - ASCITES MAMMARY ADENOCARCINOMA, MOUSE, 2835*
- PASSIVE TRANSFER
  - HAMSTER ERYTHROCYTES, MOUSE, 2829*
- PLASMACYTOMA RNA
  - MOUSE, 2828*
- RNA REPLICASE
  - MOUSE, 2691
- SV40 SURFACE ANTIGEN
  - TRANSFORMED CELL LINE, HAMSTER, 2733
- TRANSPLANTATION
  - LYMPHOMA, MOUSE, 2726
- TRANSPLANTATION ANTIGENS
  - INTRAMUSCULAR TUMOR, RAT, 2705
  - SARCOMA, SYNGENEIC, MOUSE, 2724
- VIRUS, ACUTE LYMPHOCYTIC CHORIOMENINGITIS
  - GENETIC CONTROL, MOUSE, 2712
- VIRUS, MOLONEY SARCOMA
  - LYMPHOCYTE, MOUSE, 2723
- VIRUS, MURINE LEUKEMIA
  - LYMPHOCYTE TRANSFORMATION, MOUSE, 2656
- VIRUS, RAUSCHER LEUKEMIA
  - CELL-MEDIATED, MOUSE, 2717
- X-IRRADIATED TUMOR CELLS
  - CANCER PATIENTS, 2702
- XENOGENEIC TUMOR
  - MOUSE, 2811*

IMMUNE SURVEILLANCE
- CANCER
  - HUMAN, 2770*

IMMUNITY
- ANTITUMOR
  - EFFECT ON CONCEPTUS, IMMUNIZED PREGNANT MICE, 2812*
  - MACROPHAGE-TUMOR CELL INTERACTION, MOUSE, 2818*
- CELL-MEDIATED
  - ALLOGRAFT AND TUMOR TRANSPLANT ANTIGENS, MOUSE, 2741
  - ALLOGRAFTS, RAT, 2780*
  - BCG THERAPY-HUMAN TUMORS, 2817*
  - CANCER PATIENTS, 2809*
  - "ECLIPSE", TUMOR-BEARING MOUSE, 2713
  - LEUKOCYTE BLASTOGENIC TRANSFORMATION, CHICKEN, 2753
  - TRANSPLANTED SYNGENEIC SARCOMA, MOUSE, 2724
- CELL-MEDIATED TRANSFER
  - IMMUNIZED ALLOGENEIC DONORS, 2805*
  - NEURAMINIDASE-TREATED LEUKEMIA CELL-IMMUNIZED MICE, 2886*
- CONCOMITANT
  - METHYLCHOLANTHRENE-INDUCED FIBROSARCOMA, MOUSE, 2839*
- CORYNEBACTERIUM PARVUM
  - LUNG METASTASES INCIDENCE, RAT, 2736
- DELAYED HYPERSENSITIVITY
  - HUMORAL MEDIATORS, 2888*
- HUMORAL
  - ANTILYMPHOCYTE SERUM EFFECT, GUINEA PIG, 2796*
- IRRADIATED TUMOR CELL-MEDIATED
  - SERUM INHIBITORS, HUMAN, 2702
- LEUKEMIA, ACUTE
  - HUMAN, 2887*
- LYMPHOCYTE SEQUESTRATION
  - LIVER, MOUSE, 2732
- LYMPHOCYTE TRAPPING
  - TUMOR REGRESSION, MOUSE, 2723
- MELANOMA, MALIGNANT
  - HALO NEVI, OPERATED MELANOMA PATIENTS, 2813*
- PLASMA CELL TUMOR-SPECIFIC
  - IN VITRO INDUCTION, MOUSE, 2741
- RADIATION
  - CANCER, REVIEW, 2401
- RAUSCHER VIRUS INFECTION
  - ERYTHROCYTE CLEARANCE STUDY, MOUSE, 2628
- SERUM-MEDIATED
  - LYMPHOCYTE INHIBITION, TUMOR-ASSOCIATED SPECIFICITY, 2695
  - TRANSPLANTED SYNGENEIC SARCOMA, MOUSE, 2724
- SERUM TRANSFER
  - NEURAMINIDASE-TREATED LEUKEMIA CELL-IMMUNIZED MICE, 2886*
- TOBACCO SMOKING
  - REVIEW, 2454*
- TUMOR-SPECIFIC
  - REVIEW, 2837*
- VIRUS, GROSS LEUKEMIA
  - ROUTES OF TRANSMISSION, RAT, 2690*
- VIRUS, ROUS SARCOMA
  - AVIAN LEUKOSIS VIRUSES, CHICKEN, 2753

IMMUNITY, CELLULAR
- ANTIGEN, TUMOR-ASSOCIATED

- HUMAN, REVIEW, 2414
- ANTIGENS, TRANSPLANTATION
  - RABBIT, HUMAN, REVIEW, 2430*

IMMUNODEPRESSION
- RADIATION
  - CANCER, REVIEW, 2401

IMMUNOGLOBULIN
- CELL-SURFACE
  - LEUKEMIA, HUMAN, 2716
- CULTURED LYMPHOMA CELLS
  - MOUSE, 2820*
- FREE AND MEMBRANE-BOUND POLYSOMES
  - PLASMACYTOMA, MOUSE, 2721
- LYMPHOCYTE SURFACE
  - HODGKIN'S DISEASE, 2838*
- PLASMA CELL NEOPLASMS
  - STRUCTURE, REVIEW, 2421*
- RECEPTOR CLASS SHIFT
  - SPLEEN CELL, MOUSE, 2708
- RECEPTORS
  - B AND T LYMPHOCYTES, 2728
- SERUM LEVEL
  - LYMPHOID TUMOR-BEARING TOADS, 2834*
- SURFACE ANTIGEN
  - ANTIBODY-INDUCED CHANGE, MYELOMA, MOUSE, 2737
- SYNTHESIS
  - MULTIPLE MYELOMA, GROWTH KINETICS, REVIEW, 2478*
  - MYELOMA CELLS, MOUSE, 2766

IMMUNOGLOBULIN IG-1B
- ALLOTYPE SYNTHESIS
  - SUPPRESSION, T CELLS, MICE, 2693

IMMUNOGLOBULIN IGA
- ANTI-STREPTOCOCCAL HYALURONIDASE ACTIVITY
  - MYELOMA, HUMAN, 2871*
- BACILLUS CALMETTE-GUERIN
  - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
- BRONCHOPULMONARY CARCINOMA
  - SALIVA, HUMAN, 2782*
- HETEROGENEITY
  - MYELOMA, HUMAN, 2870*
- J PROTEIN
  - MYELOMA, HUMAN, 2870*
- OROPHARYNGEAL CARCINOMA
  - SALIVA, HUMAN, 2782*
- SHARED DETERMINANT, IMMUNOGLOBULIN IGM
  - SERUM, HUMAN, 2784*

IMMUNOGLOBULIN IGE
- IMMUNE RESPONSE
  - PRIMED SPLEEN CELL-TREATED, IRRADIATED MICE, 2774*

IMMUNOGLOBULIN IGG
- BACILLUS CALMETTE-GUERIN
  - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
- BETA2-MICROGLOBULIN
  - LYMPHOID CELL LINES, HUMAN, 2857*
- RECOMBINATION, H, L CHAINS
  - MYELOMA PROTEIN SUBCLASSES, 2845*

IMMUNOGLOBULIN IGM
- BACILLUS CALMETTE-GUERIN
  - PARABLASTIC LEUKEMIA, CHILDREN, 2880*
- CELL ORIGIN MARKER
  - BURKITT'S LYMPHOMA, 2754
- CHRONIC LYMPHOCYTIC LEUKEMIA
  - HUMAN, 2771*
- SHARED DETERMINANT, IMMUNOGLOBULIN IGA
  - SERUM, HUMAN, 2784*

IMMUNOLOGY
- CANCER
  - REVIEW, 2435*, 2878*
- HODGKIN'S DISEASE
  - REVIEW, 2460*
- TUMOR
  - CLINICAL ASPECTS, 2873*

IMMUNOPATHOLOGY
- CANCER
  - REVIEW, 2877*

IMMUNOSUPPRESSION
- ANTIBODY
  - REVIEW, 2760
- AZATHIOPRINE
  - NEPHROTIC SYNDROME, MALIGNANCY, CASE REPORT, 2830*
- DIETHYLNITROSAMINE
  - GUINEA PIG, 2761
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - MECHANISM, MOUSE, 2525
- 7-12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMOR
  - ANTI-LYMPHOCYTE SERUM, RAT, 2527
- 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMOR
  - CYCLOPHOSPHAMIDE, RAT, 2531
- LEUKEMIA, ACUTE LYMPHOBLASTIC
  - ANTIBIOTICS, 2546*
- LYMPHOMA
  - ULTRASTRUCTURE TISSUE CULTURE, MOUSE, 2701
- MALIGNANT LYMPHOMA INDUCTION
  - GRAFT-VS-HOST REACTION, 2726
  - LYMPHOID CELL TRANSPLANT, MOUSE, 2726
- METHYLCHOLANTHRENE
  - GUINEA PIG, 2761
- TRANSPLANTATION
  - INTRAVENOUSLY INJECTED ALLOGENEIC TUMOR CELLS, RAT, 2889*

IMMUNOSURVEILLANCE
- IMMUNOSUPPRESIVE PROTEIN
  - PLASMA, HUMAN, 2781*
- SPONTANEOUS LYMPHOMA
  - "NUDE" MOUSE, 2764

INFECTIOUS MONONUCLEOSIS
- LEUKOCYTE INTERFERON PRODUCTION
  - HUMAN, 2748
- PHA RESPONSE
  - HUMAN, 2748

INTERFERON
- CELL SENSITIVITY TO PRODUCTION
  - LEUKEMIA-INFECTED EMBRYO

    - FIBROBLASTS, MOUSE, 2661
  - PRODUCTION
    - LEUKOCYTES, MONONUCLEOSIS, HUMAN, 2748
  - VIRUS, LEUKEMIA
    - GRAFT VERSUS HOST REACTION, MOUSE, 2674*

- INTESTINE
  - LARGE BOWEL CANCER
    - PHYTOHEMAGGLUTININ, LYMPHOCTYE TRANSFORMATION, HUMAN, 2848*

- INTESTINE, LARGE
  - DIVERTICULOSIS
    - HAWAII JAPANESE, AUTOPSY STUDY, 2914*

- IODINE 131
  - THYROID CARCINOMA
    - CASE REPORTS, 2610*
  - THYROID CARCINOMA DEVELOPMENT
    - HYPERTHYROIDISM, HUMAN, 2588

- IRRITANTS
  - CARCINOGEN, CHEMICAL
    - REVIEW, 2447*

- L-ISOPROTERENOL
  - CYCLIC AMP ACCUMULATION
    - BENZO(A)PYRENE, SKIN, MOUSE, 2490

- KIDNEY
  - ADENOMA
    - 1-NITROSO-5,6-DIHYDROURACIL, RAT, 2516
  - CARCINOMA
    - ENZYME CYTOCHEMISTRY, ELECTRON MICROSCOPY, 2969
  - DIMETHYLNITROSAMINE
    - EMBRYONAL TISSUE, MOUSE, 2579*
  - VIRUS, POLYOMA
    - ALLOGRAFT RECIPIENTS, CLINICAL STUDY, 2638

- KIDNEY TUMOR
  - BASIC LEAD ACETATE
    - RAT, 2576*
  - N-BUTYL-N(4-HYDROXYBUTYL)NITROSAMINE
    - RAT, 2576*
  - 3-METHYLCHOLANTHRENE
    - RAT, 2576*
  - 4-NITROQUINOLINE-1-OXIDE
    - RAT, 2576*
  - N-NITROSODIBUTYLAMINE
    - RAT, 2576*
  - N-NITROSODIMETHYLAMINE
    - POLYNUCLEOTIDE LIGASE ACTIVITY, RAT, 2486
    - RAT, 2576*

- LACTATE DEHYDROGENASE
  - SPECTRUM SHIFTS
    - MALIGNANT TUMORS, HUMAN, 2994*

- LARYNX
  - PRECANCEROUS, CANCEROUS CONDITIONS
    - ELECTRON MICROSCOPY, HUMAN, 2904*

- LEUKEMIA
  - ANTIGEN
    - HUMAN, REVIEW, 2409
    - ONCORNAVIRUS-ASSOCIATED ANTIBODY, CAT, 2730
    - REVIEW, 2425*
  - -ASSOCIATED ANTIGEN
    - HUMAN, 2832*
  - CHILDREN
    - INTRA-UTERINE RADIATION, CLINICAL STUDY, 2604*
  - DISCRIMINATION OF LEUKEMIC FROM NORMAL CELLS
    - REVIEW, 2474*
  - DNA POLYMERASE
    - PLASMA, GUINEA PIG, 2982
  - EARLY AND ATYPICAL DIAGNOSIS
    - CLINICAL STUDY, 2991*
  - EARLY STAGES
    - DIAGNOSIS, HUMAN, 2911*
  - EPIDEMIOLOGY
    - NEBRASKA, 2941*
  - ETIOLOGY, VIRAL
    - MOLECULAR MECHANISMS, REVIEW, 2402
  - IMMUNOGLOBULIN, CELL-SURFACE
    - HUMAN, 2716
  - INTER-SPECIES TRANSFER
    - CAYMAN, 2739
  - NERVOUS SYSTEM, CENTRAL
    - PYRIMETHAMINE, REVIEW, 2455*
  - N-(4-(5-NITRO-2-FURYL)-2-THIAZOLYL) ACETAMIDE
    - EFFECT OF SPLENECTOMY, MOUSE, 2582*
    - EFFECT OF THYMECTOMY, MOUSE, 2582*
  - POLYRIBOSOME
    - PHYSICOCHEMISTRY, RAT, 2960
  - RADIATION
    - REVIEW, 2408
  - RNA
    - HUMAN, 2976
    - LABELLING PATTERNS, 2974
  - VIROLOGICAL RESEARCH
    - HUMAN, REVIEW, 2472*
  - VIRUS, C-TYPE RNA
    - CAT, 2662
  - VIRUS, RNA TUMOR
    - ETIOLOGY, HUMAN, 2612
    - HUMAN, 2632
    - MOLECULAR BIOLOGY, REVIEW, 2412

- LEUKEMIA, ACUTE
  - BLOOD PLATELETS
    - ULTRASTRUCTURE, CLINICAL STUDY, 2999*
  - GRANULOCYTOPENIA
    - HUMORAL FACTORS, HUMAN, 2908*
  - HEMOPOIESES
    - PROLIFERATION KINETICS, HUMAN, 2908*
  - IMMUNITY
    - HUMAN, 2887*
  - PRECONDITIONS
    - HEMATOLOGIC SYNDROME, CLINICAL STUDY, 2906*
    - HEMOPOIETIC STEM CELL, HUMAN, 2903*
  - UNIMODAL CLUSTERING
    - STATISTICAL TEST, ATLANTA, GEORGIA,

2946*
- VIRUS, C-TYPE
  - ENZYME, HUMAN, 2622

LEUKEMIA, ACUTE LYMPHOBLASTIC
- IMMUNOSUPPRESSION
  - ANTIBIOTICS, 2546*
- VIRAL ETIOLOGY
  - ARGININE DEPRIVATION, HUMAN, 2634

LEUKEMIA, ACUTE MYELOBLASTIC
- INTRAVASCULAR COAGULATION
  - LEUKOCYTES, HUMAN, 2988*

LEUKEMIA, ACUTE MYELOID
- BLASTIC CRISIS
  - CLINICAL STUDY, 2894

LEUKEMIA, CHLORO-
- KINETICS
  - DIFFUSION CHAMBERS, RAT, 2986

LEUKEMIA, CHRONIC GRANULOCYTIC
- KARYOTYPE EVOLUTION
  - CLINICAL STUDY, 2901

LEUKEMIA, CHRONIC LYMPHOBLASTIC
- VIRAL ETIOLOGY
  - ARGININE DEPRIVATION, HUMAN, 2634

LEUKEMIA, CHRONIC LYMPHOCYTIC
- IMMUNOGLOBULIN IGM
  - HUMAN, 2771*
- LYMPHOCYTES
  - RESPONSE TO PLANT MITOGENS, 2856*
- THYMUS-DERIVED PROLIFERATING LYMPHOCYTES, 2814*

LEUKEMIA, CHRONIC LYMPHOID
- RECTAL CARCINOMA, PLASMACYTOMA
  - FAMILIAL OCCURRENCE, CASE REPORT, 3000*
- TRANSMISSION BY EMBRYONAL CHICKEN
  - ELECTRON MICROSCOPE EXAMINATION, 2667*

LEUKEMIA, CHRONIC MYELOGENOUS
- CHROMOSOME ABERRATIONS
  - CLINICAL STUDY, 2902

LEUKEMIA, CHRONIC MYELOID
- BLASTIC CRISIS
  - ACUTE TRANSFORMATION, CLINICAL STUDY, 2894
- MALIGNANT RETICULOSIS
  - HUMAN, 2909*
- PHILADELPHIA CHROMOSOME-NEGATIVE, LYSOZYME-POSITIVE
  - CASE REPORTS, 2893
- TRANSMISSION BY EMBRYONAL CHICKEN
  - ELECTRON MICROSCOPE EXAMINATION, 2667*

LEUKEMIA, FELINE
- INFECTIOUS ANEMIA
  - ASSOCIATION, CAT, 2892
- PERITONITIS
  - MULTIPLE OCCURRENCE IN HOUSEHOLD, 2921

LEUKEMIA, PLASMA CELL
- DNA SYNTHESIS
  - CYTOPHOTOMETRY, AUTORADIOGRAPHY, 2948*

LEUKOCYTE
- ANOMALIES
  - BONE MARROW PROLIFERATIVE DISEASES, HUMAN, 2990*
- INTERFERON PRODUCTION
  - INFECTIOUS MONONUCLEOSIS, HUMAN, 2748
- PHA RESPONSE
  - INFECTIOUS MONONUCLEOSIS, HUMAN, 2748
- RNA SYNTHESIS
  - INFECTIOUS MONONUCLEOSIS, HUMAN, 2748

LEUKOSIS, LYMPHOID
- RADIATION, X-RAY
  - CHICK, 2589

LIGASE
- ACTIVITY
  - N-NITROSODIMETHYLAMINE-INDUCED KIDNEY TUMOR, RAT, 2486

LINOLEATE, OXIDIZED
- CARCINOGENIC RISK DETECTION
  - SENSITIVITY OF FEEDING TESTS, RAT, 2492

LIP
- ORAL CANCER
  - ETIOLOGY, PATHOGENESIS, REVIEW, 2436*

LIP, LOWER
- CARCINOMA
  - ETIOLOGY, HUMAN, REVIEW, 2416

LIVER
- ARYLAMINE ACETYL-TRANSFERASE
  - HAMSTER, GUINEA PIG, MOUSE, RAT, DOG, 2503
- CARCINOGENESIS
  - GLYCOLOSIS REGULATION, 2993*
- CHOLESTEROL SYNTHESIS
  - 2-ACEYLAMINOFLUORENE-FED RATS, 2501
- DIETHYLSTILBESTROL
  - GLUCURONYLTRANSFERASE ACTIVITY, HAMSTER, RAT, 2523
- DIMETHYLNITROSAMINE CARCINOGENESIS
  - FERRIC OXIDE SYNERGISM, HAMSTER, 2505
- DNA SYNTHESIS
  - INHIBITION BY URETHANE, RAT, 2495
- HEPATOMA
  - FOLATE DEFICIENCY, RAT, 2970
  - MYCOTOXINS, REVIEW, 2410

LIVER CANCER
- AFLATOXIN
  - RAINBOW TROUT, 2566*

LIVER CELL CARCINOMA
- 1-NITROSO-5,6-DIHYDROURACIL

    - RAT, 2516

LSD
- CHROMOSOME ABERRATIONS
  - RABBIT, 2520

LUNG
- ASBESTOS
  - ULTRASTRUCTURE, MESOTHELIOMA AND NORMAL TISSUE, 2923
- METASTASIS
  - HEPATOMA, LEG, RAT, 2736
  - REGRESSION, TESTICULAR TUMOR, HUMAN, 2824*
- PULMONARY ADENOCARCINOMA
  - DIBUTYLNITROSOAMINE, RAT, 2565*
- PULMONARY ADENOMA
  - DIETHYLNITROSAMINE, ULTRASTRUCTURE, HAMSTER, 2573*
- TUMOR IMMUNITY
  - IRRADIATED TUMOR CELLS, RAT, 2736

LUNG CANCER
- ASBESTOS
  - REVIEW, 2919
- EMPYEMA
  - REVIEW, 2449*
- EPIDEMIOLOGY
  - HUMAN, REVIEW, 2407
- MORTALITY
  - NATIONAL CIGARETTE CONSUMPTION, 2498
- TOBACCO SMOKING
  - AGE-SPECIFIC DEATH RATES, 2938*
  - CIGAR, PIPE, RETROSPECTIVE STUDY, 2932
  - HUMAN, 2561*

LUNG TUMOR
- RADIATION, X-RAY
  - CLONOGENIC GROWTH, RAT, 2598*
  - EXPOSURE RESPONSE, MOUSE, 2607*

LUTEOSKYRIN
- PRODUCTION
  - BIOLOGICAL FACTORS, REVIEW, 2468*

LYMPH NODE
- MORPHOLOGY
  - IMMUNOLOGICAL FUNCTION, 2804*

LYMPHOCYTE
- ALLOGENEIC LYMPHOCYTE-STIMULATED
  - THYMUS DEPENDENCE, 2831*
- ANTIGEN-STIMULATED
  - THYMUS DEPENDENCE, 2831*
- ANTISERUM
  - HELA TUMOR SURVIVAL, MOUSE, 2847*
- B AND T
  - ANTIGEN RECEPTORS, 2728
- B-CELL
  - ACTIVATION MECHANISM, 2801*
  - ANTITUMOR ACTIVITY, 2673*
  - IMMUNE COMPLEXES RELEASE, MOUSE, 2797*
- B CELL
  - PHYTOHEMAGGLUTININ TRANSFORMATION, 2840*
- B-CELL, ACTIVATION
  - COMPLEMENT C3 RECEPTOR, 2793*
- BLASTIC TRANSFORMATION INDUCTION
  - ANTILYMPHOCYTE GLOBULIN INJECTION SITE, CASE REPORT, 2742
- CYTOTOXICITY
  - ISOANTISERUM INHIBITION, MOUSE, 2715
  - SOLUBLE TUMOR MEMBRANE FRACTION INHIBITION, 2852*
- HISTOINCOMPATIBILITY
  - ALLOGENEIC EFFECT, IRRADIATED HOST, MOUSE, 2714
- MIGRATION
  - TUMOR IMMUNITY, MOUSE, 2732
- MITOGEN-STIMULATED
  - THYMUS DEPENDENCE, 2831*
- NUCLEAR POCKETS INCIDENCE
  - C-TYPE VIRUS PARTICLES, BULL, 2636
- PHYTOHEMAGGLUTININ-RESPONSIVE
  - CHRONIC LYMPHOCYTIC LEUKEMIA CELL CULTURES, HUMAN, 2863*
- PRECURSOR CELLS
  - NU/NU (THYMUSLESS) MOUSE, 2763
- QUANTITATION OF PROLIFERATIVE RESPONSES TO MITOGENS
  - T AND B CELLS, MOUSE, 2765
- REACTIVITY, TUMOR-ASSOCIATED
  - AUTOCHTHONOUS TUMOR CELLS, HUMAN, 2738
- RESPONSE TO PHYTOHEMAGGLUTININ
  - CANCER PATIENTS, 2762
- RESPONSE TO PLANT MITOGENS
  - CHRONIC LYMPHOCYTIC LEUKEMIA, 2856*
- ROSETTE-FORMING CELLS
  - LYMPHOID TISSUE-DERIVED, HUMAN, 2740
- SERUM INHIBITORY EFFECT
  - AUTOCHTHONOUS TUMOR CELLS, HUMAN, 2695
- STIMULATION
  - SOLUBLE ANTIGEN, TUMOR, HUMAN, 2773*
- SURFACE ANTIGENS AND RECEPTORS
  - MOUSE, 2698
- SURFACE IMMUNOGLOBULINS
  - HODGKIN'S DISEASE, 2838*
- T, B CELL INTERACTION
  - HISTOINCOMPATIBILITY EFFECT, 2842*
- T CELL AUTOSENSITIZATION
  - CULTURED CELLS, MOUSE, RAT, 2775*
- THYMUS AND BONE MARROW DERIVED
  - ANTIGEN RECOGNITION, 2743
- THYMUS-DERIVED PROLIFERATING
  - CHRONIC LYMPHOCYTIC LEUKEMIA, 2814*
- TRANSFORMATION
  - INDUCTION BY CONCANAVALIN A, ACETYL CONCANAVALIN A, HUMAN, 2860*
  - LEUKEMIA VIRUS, IMMUNE RESPONSE, MOUSE, 2656
  - MALIGNANT DISEASE, 2769*
  - NONSPECIFIC CYTOTOXICITY, 2794*
  - PHYTOHEMAGGLUTININ, HUMAN CHORIONIC GONADOTROPHIN EFFECT, 2866*
  - PHYTOHEMAGGLUTININ, HUMAN CHORIONIC SOMATOMAMMOTROPHIN

    - EFFECT, 2866*
  - PHYTOHEMAGGLUTININ, LARGE BOWEL CANCER, HUMAN, 2848*
  - RNA ACCUMULATION, 2825*

LYMPHOCYTE RESPONSE
- POKEWEED MITOGEN
  - HODGKIN'S DISEASE, 2844*

LYMPHOGRANULOMATOSIS
- IMMUNE RESPONSE
  - INFLUENZA VACCINE, 2874*

LYMPHOID TISSUE
- LARGE STELLATE DENDRITE CELL
  - MORPHOLOGY, DISTRIBUTION, MOUSE, 2711

LYMPHOLYSIS
- CELL-MEDIATED
  - H-2 REGION, LYMPHOCYTE, MOUSE, 2787*

LYMPHOMA
- ANTIGEN, THETA
  - CULTURED CELLS, MOUSE, 2820*
- ANTIGEN, VIRUS-RELATED
  - BACILLUS CALMETTE-GUERIN INOCULATIONS, 2727
- EPIDEMIOLOGY
  - NEBRASKA, 2941*
- IMMUNOGLOBULIN
  - CULTURED CELLS, MOUSE, 2820*
- INDUCED BY ANTIGENIC STIMULATION, IMMUNOSUPPRESSION
  - ULTRASTRUCTURE TISSUE CULTURE, MOUSE, 2701
- NUCLEAR RNA
  - LABELLING PATTERNS, 2974
- PROMOTION, IN VITRO
  - THIOLS, MOUSE, 2692
- SPONTANEOUS
  - IMMUNOSURVEILLANCE, "NUDE" MOUSE, 2764
- TUMOR-SPECIFIC TRANSPLANTATION ANTIGENS
  - FRIEND VIRUS COMPLEX, 2663*
- VIRUS, RNA TUMOR
  - HUMAN, 2632
- X-IRRADIATION
  - DNA SYNTHESIS, RAT, 2611*

LYMPHOSARCOMA
- VIRUS, C-TYPE
  - CATTLE, 2637

MACROPHAGE
- ANTI-TUMOR ANTIBODY
  - ACTIVATED FC, MOUSE, RAT, 2750
- CYTOTOXICITY
  - SYNGENEIC, ALLOGENEIC ERYTHROCYTES, MOUSE, 2785*
- TUMOR CELL INHIBITION
  - CYTOSTATIC MECHANISM, RAT, 2694

MAGNESIUM
- DEFICIENCY
  - THYMIC HYPERPLASIA, RAT, 2752

MALIGNANCY
- CHROMOSOME CONSTITUTION
  - L CELL-TUMOR CELL HYBRID, MOUSE, 2958

MAMMARY ADENOCARCINOMA
- LACTATE AND MALATE DEHYDROGENASE ISOZYMES
  - MOUSE, 2664*
- TRANSPLANTATION SITE PREIRRADIATION
  - VASCULAR SPACE, MICE, 2591*

MAMMARY CARCINOMA
- CARBON TETRACHLORIDE
  - GLUCURONIC ACID LEVELS, RAT, 2568*
- CARCINOEMBRYONIC ANTIGEN
  - HUMAN, 2700
- CHROMATIN, SEX
  - CLINICAL STUDY, 2998*
- PROTON SPIN-LATTICE RELAXATION
  - MOUSE, 2981
- RNA, VIRUS-SPECIFIC
  - MOLECULAR HYBRIDIZATION, MOUSE, 2657
- VIRUS, RNA TUMOR
  - A AND B PARTICLES, MOUSE, 2623

MAMMARY GLAND
- ADENOCARCINOMA
  - LACTATE AND MALATE DEHYDROGENASE ISOZYMES, MOUSE, 2664*
- GYNECOMASTASIA, FIBROADENOMATOSIS
  - HISTOCHEMISTRY, HUMAN, 2900
- N-HYDROXY-4-ACETYLAMINOBIPHENYL
  - TRANSACETYLASE ACTIVITY, RAT, 2479

MAMMARY TUMOR
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - EFFECT OF DIMETHYLBENZYLDESMETHYLRIFAMPICIN, RAT, 2537*
  - IMMUNOSUPPRESSION, ANTI-LYMPHOCYTE SERUM, RAT, 2527
  - IMMUNOSUPPRESSION, CYCLOPHOSPHAMIDE, RAT, 2531
  - MAST CELLS, RAT, 2517
- 7,12 DIMETHYLBENZ(A)ANTHRACENE
  - PROLACTIN LEVEL, PLASMA, RAT, 2512
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - RAT, 2744
  - SUPPERSSION BY PREGNANCY, RAT, 2519
  - SUPPRESSION BY CHLOROTRIANISENE, RAT, 2519
- ESTROGEN RECEPTORS
  - HUMAN, 2977
- ESTRONE-INDUCED
  - REGRESSION, ENZYME ACTIVITIES, RAT, 2551*
- ETIOLOGY, VIRAL
  - MOLECULAR MECHANISMS, REVIEW, 2402
- HORMONE
  - RAT, 2744
- IMMUNOSUPPRESSION EFFECTS
  - RAT, 2744
- PROGESTERONE IMPLANTATION
  - ISOGRAFT-BEARING ORCHIDECTOMIZED MICE, 2757
- PROLACTIN LEVELS

- ISOGRAFT-BEARING ORCHIDECTOMIZED MICE, 2757
- RADIATION, X-RAY
  - RAT, 2597*
- 7,8,12-TRIMETHYLBENZANTHRACENE
  - EFFECT OF DIMETHYLBENZYLDESMETHYLRIFAMPICIN, RAT, 2537*

MAXILLARY TUMOR
- OCCUPATIONAL HAZARD
  - WOOD WORKERS, 2575*

MELANOMA
- ENZYME CYTOCHEMISTRY
  - ELECTRON MICROSCOPY, 2969
- RNA POLYMERASE
  - PROPERTIES, HUMAN, 2980

MELANOMA, MALIGNANT
- CHROMOSOME ABERRATIONS
  - CELL CULTURE, HUMAN, 2967
- EPIDEMIOLOGY, SURVIVAL
  - CALIFORNIS, HUMAN, 2916
- IMMUNITY
  - HALO NEVI, OPERATED MELANOMA PATIENTS, 2813*
- IRIS
  - ULTRASTRUCTURE, HUMAN, 2961
- SKIN
  - INCIDENCE, ENGLAND, 2925
- VACCINATION SCAR
  - CASE REPORT, 2631

MEMBRANE
- ANTIGEN
  - SERUM, SV40 TUMOR-BEARING HAMSTER, 2854*
  - SV40-TRANSFORMED CELLS, HAMSTER, 2855*
  - THYMUS CELLS, RABBIT, 2808*
- ANTIGEN, TUMOR-SPECIFIC
  - SERUM, CHEMICALLY-INDUCED SARCOMA, RAT, 2890*
- FLUID NATURE
  - LYMPHOCYTE, MOUSE, 2698
- LIGAND-INDUCED MOLECULAR MOVEMENT
  - CELL METABOLISM B LYMPHOCYTES, MOUSE, 2786*
- SITE MOBILITY
  - CELL GROWTH, 2758

MENINGIOMA
- ACROCENTRIC CHROMOSOMES
  - CELL CULTURE, HUMAN, 2971

MESOTHELIOMA
- ASBESTOS
  - OCCURRENCE, NETHERLANDS, 2915
  - REVIEW, 2919
  - ULTRASTRUCTURE, 2923
- ASBESTOS EXPOSURE
  - MORTALITY STUDY, 2928
- EPIDEMIOLOGY
  - CANADA, 2934

MESOTHELIOMA, MALIGNANT
- UTERUS
  - DIETHYLSTILBESTROL, MONKEY, 2529

MESTRANOL
- CARCINOGENESIS
  - MOUSE, 2502

METABOLISM
- AFLATOXIN B1
  - BIOCHEMICAL EFFECTS, HYPOPHYSECTOMIZED RAT, 2532
- ARENE OXIDE
  - NIH SHIFT, REVIEW, 2405
- CARCINOGEN, CHEMICAL
  - NIH SHIFT, REVIEW, 2405
- 4-DIMETHYLAMINOBIBENZYL
  - RAT, 2493
- TRANS-4-DIMETHYLAMINOSTILBENE
  - RAT, 2493
- DIMETHYLNITROSAMINE
  - AMPHIBIANS, FISH, IN VITRO, 2543*
- 4,4-DINITROBIPHENYL
  - GUINEA PIG, RAT, 2526
- GLYCOLYSIS
  - LIVER CARCINOGENESIS, 2993*
- NITROSAMINES
  - RAT, 2511

METALS
- VIRAL INFECTIONS
  - MOUSE, 2536*

METASTASIS
- INTRACRANIAL TUMOR
  - INCIDENCE, IBADAN, NIGERIA, 2944*
- LUNG
  - HEPATOMA, LEG, RAT, 2736
  - REGRESSION, TESTICULAR TUMOR, HUMAN, 2824*
- RADIATION, X-RAY
  - LUNG TUMOR, RAT, 2598*
- RESPIRATORY SYSTEM TUMORS
  - INCIDENCE, MOUSE, 2895

METASTATIC CARCINOMA
- IMMUNE RESPONSE
  - INFLUENZA VACCINE, 2874*

D-METHAMPHETAMINE-HCL
- CHROMOSOME ABERRATIONS
  - RABBIT, 2520

3-METHYL-4-DIMETHYLAMINOAZOBENZENE
- ADENOCARCINOMA
  - CARCINOGEN-BINDING PROTEIN, RAT, 2564*
- HEPATOMA
  - CARCINOGEN-BINDING PROTEIN, RAT, 2564*

3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
- PROTEIN TARGET
  - LIVER, RAT, 2474
- THYROIDITIS, CHRONIC
  - RAT, 2556*
- TUMOR INDUCTION
  - LIVER, RAT, 2474

2-METHYL-N,N-DIMETHYLAMINOAZOBENZENE
- DNASE
  - LIVER, RAT, MOUSE, 2489

- 3'-METHYL-N,N-DIMETHYLAMINOAZOBENZENE
  - DNASE
    - LIVER, RAT, MOUSE, 2489

- N-METHYL-N-NITROSOUREA
  - DNA DAMAGE
    - LIVER, RAT, 2506

- N-METHYL-N-NITROSOURETHAN
  - DNA DAMAGE
    - LIVER, RAT, 2506

- 3-METHYL-4-NITROPYRIDINE-1-OXIDE
  - XERODERMA PIGMENTOSUM
    - SENSITIVITY, HUMAN CELLS, 2481

- 2-METHYL-4-NITROQUINOLINE-1-OXIDE
  - XERODERMA PIGMENTOSUM
    - SENSITIVITY, HUMAN CELLS, 2481

- METHYLAZOXYMETHANOL ACETATE
  - DNA DAMAGE
    - LIVER, RAT, 2506

- METHYLCHOLANTHRENE
  - FIBROSARCOMA
    - CONCOMITANT IMMUNITY, MOUSE, 2839*
  - IMMUNOSUPPRESSION
    - GUINEA PIG, 2761
  - TUMOR INDUCTION
    - GUINEA PIG, 2488

- 3-METHYLCHOLANTHRENE
  - CARBON TETRACHLORIDE HEPATOTOXICITY
    - RAT, 2567*
  - FIBROSARCOMA
    - ARYL HYDROCARBON HYDROXYLASE, MOUSE, 2483
  - KIDNEY TUMOR
    - RAT, 2576*
  - URINARY BLADDER TUMOR
    - RAT, 2576*
  - UTERUS
    - HISTOLOGICAL CHANGES, MOUSE, 2480

- 20-METHYLCHOLANTHRENE
  - CERVIX
    - 3H-THYMIDINE UPTAKE, MOUSE, 2530
  - SKIN
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
  - SPLEEN
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
  - UTERINE CERVIX
    - ULTRASTRUCTURE, CASTRATION, RAT, 2577*

- METHYLCHOLANTRENE
  - TUMOR INDUCTION
    - SARCOMA, MOUSE, 2522

- METHYLNITROSOUREA
  - URINARY BLADDER CARCINOMA
    - RAT, 2545*

- METHYLPROPYLNITROSAMINE
  - RESPIRATORY SYSTEM TUMORS
    - HAMSTER, 2485

- MILK
  - VIRUS, SIMIAN-TYPE PARTICLE
    - ELECTRON MICROSCOPIC DETECTION, HUMAN, 2689*

- MITOCHONDRIA
  - ETHIDIUM BROMIDE RESISTANCE
    - SV40-TRANSFORMED HAMSTER CELLS, 2687*
  - HEPATOMA
    - OXIDATIVE PHOSPHORYLATION, RAT, 2979

- MITOGEN
  - LYMPHOCYTE ACTIVATION, QUANTITATION
    - T AND B CELLS, MOUSE, 2765

- MITOGEN, POKEWEED
  - EFFECT ON PERIPHERAL LYMPHOCYTES
    - CHRONIC LYMPHOCYTIC LEUKEMIA, 2856*
  - HODGKIN'S DISEASE
    - LYMPHOCYTE RESPONSE, 2844*

- MORTALITY
  - CARDIOVASCULAR CANCER
    - WATER HARDNESS, ENGLAND, 2951*
  - LUNG CANCER
    - NATIONAL CIGARETTE CONSUMPTION, 2498
  - PANCREATIC CANCER
    - REVIEW, 2419*
  - STOMACH CANCER
    - ANTIOXIDANTS IN FOOD, REVIEW, 2431*
    - REVIEW, 2419*

- MUTAGENESIS
  - DIMETHYLNITROSAMINE
    - METABOLISM, RAT LIVER MICROSOMES, 2555*

- MYCOTOXIN
  - HEPATOMA
    - REVIEW, 2410
  - INTERNATIONAL AFLATOXIN CHECK SAMPLE PROGRAM
    - 1972 STUDY, 2569*
  - PRODUCTION
    - BIOLOGICAL FACTORS, REVIEW, 2468*

- MYELOMA
  - GAMMAGLOBULIN
    - MOUSE, 2697
  - GAMMAGLOBULIN SYNTHESIS
    - MOUSE, 2734
  - GLOBULINS
    - ANTIBODY ACTIVITY, HUMAN, REVIEW, 2420*
  - IMMUNOGLOBULIN SURFACE ANTIGEN
    - CELL MEMBRANE, MOUSE, 2737

- MYELOMA, MALIGNANT
  - LATITUDE
    - CLINICAL STUDY, 2592*

- MYELOMA, MULTIPLE
  - FAMILIAL OCCURRENCE
    - CASE REPORT, 2959

- IMMUNE RESPONSE
    - INFLUENZA VACCINE, 2874*
- IMMUNOGLOBULIN SYNTHESIS
    - GROWTH KINETICS, REVIEW, 2478*
- PRODUCTION OF BOTH IGG TYPE LAMBDA PROTEINS
    - SINGLE PLASMA CELL LINE, CASE REPORT, 2815*
- PURPURA, MONOCLONAL HYPERGLOBULINEMIC
    - IGG ANTI-IGG COMPLEXES, 2861*

MYXOSARCOMA
- ENDOPLASMIC RETICULUM, TUBULES
    - ULTRASTRUCTURE, RABBIT, 2966

NASOPHARYNGEAL CARCINOMA
- LYMPHOBLASTOID TRANSFORMATION
    - ULTRASTRUCTURE, 2897
- VIRUS, EPSTEIN-BARR
    - ANTIGEN, TUMOR BIOPSIES, 2710
    - CELL CULTURE TRANSFORMATION, 2897

NEOPLASIA
- ETIOLOGY, VIRAL
    - MOLECULAR MECHANISMS, REVIEW, 2402

NEOPLASMS
- PLASMA CELL
    - IMMUNOGLOBULIN STRUCTURE, REVIEW, 2421*

NEPHROSIS
- COLON CARCINOMA
    - CARCINOEMBRYONIC ANTIGEN-ANTIBODY COMPLEXES, CASE REPORT, 2707

NERVOUS SYSTEM, CENTRAL
- GLIAL TUMOR CELLS
    - X-IRRADIATION, RAT, 2606*
- LEUKEMIA
    - PYRIMETHAMINE, REVIEW, 2455*

NERVOUS SYSTEM TUMORS
- MALIGNANT
    - INCIDENCE, MIGRANT EAST AFRICANS, 2945*

NEURAMINIDASE
- TRANSPLANTATION LOSS
    - LEUKEMIA, MOUSE, 2886*

NEUROBLASTOMA
- BRAIN SPECIFIC PROTEIN
    - MOUSE, 2846*

NICOTINE STOMATITIS
- HARD PALATE CARCINOMA
    - REVERSE SMOKERS, 2571*

NITRO COMPOUNDS
- N-OXIDATION
    - URINARY BLADDER, DOG, MONKEY, 2510

N-(4-(5-NITRO-2-FURYL)-2-THIAZOLYL) ACETAMIDE
- LEUKEMIA
    - EFFECT OF SPLENECTOMY, MOUSE, 2582*
    - EFFECT OF THYMECTOMY, MOUSE, 2582*
- STOMACH CARCINOMA
    - EFFECT OF SPLENECTOMY, MOUSE, 2582*
    - EFFECT OF THYMECTOMY, MOUSE, 2582*

4-NITROQUINOLINE-1-OXIDE
- ENDOREDUPLICATION
    - CULTURED CELLS, HAMSTER, 2554*
- KIDNEY TUMOR
    - RAT, 2576*
- URINARY BLADDER TUMOR
    - RAT, 2576*
- XERODERMA PIGMENTOSUM
    - SENSITIVITY, HUMAN CELLS, 2481

NITROSAMINES
- ADRENAL CORTEX CELLS
    - STIMULATION, RAT, 2534
- FORMATION
    - SECONDARY AMINES, NITRITE, RAT, 2521
- FORMATION IN VITRO
    - BACTERIA, 2541*
- METABOLISM
    - RAT, 2511

N-NITROSAMINES
- OCCUPATIONAL HAZARD
    - HANDLING PRECAUTIONS, 2553*

1-NITROSO-5,6-DIHYDROURACIL
- CARCINOGENESIS
    - RAT, 2516
- LIVER CELL CARCINOMA
    - RAT, 2516

N-NITROSOBUTYLUREA
- CARCINOGENICITY
    - MOUSE, RAT, 2514

N-NITROSODIBUTYLAMINE
- KIDNEY TUMOR
    - RAT, 2576*
- URINARY BLADDER TUMOR
    - RAT, 2576*

N-NITROSODIMETHYLAMINE
- KIDNEY TUMOR
    - POLYNUCLEOTIDE LIGASE ACTIVITY, RAT, 2486
    - RAT, 2576*
- URINARY BLADDER TUMOR
    - RAT, 2576*

NORETHYNODREL
- CARCINOGENESIS
    - MOUSE, 2502

NUCLEIC ACID
- HYBRIDIZATION
    - HERPESVIRUS 2, CERVICAL CARCINOMA, HUMAN, REVIEW, 2417

OCCUPATIONAL HAZARD
- ASBESTOS
    - ASSESSMENT AND RECOMMENDATIONS, 2558*
    - LUNG CANCER, MESOTHELIOMA, 2919
    - MESOTHELIOMA, MORTALITY STUDY,

2928

- MESOTHELIOMA, NETHERLANDS, 2915
- REVIEW, 2427*

- ETHMOID TUMOR
  - WOOD WORKERS, 2575*
- HYDROGENATED TERPHENYL
  - NUCLEAR REACTOR COOLANT, 2491
- MAXILLARY TUMOR
  - WOOD WORKERS, 2575*
- NATIONAL STANDARDS
  - LABOR DEPARTMENT, UNITED STATES, 2949*
- N-NITROSAMINES
  - HANDLING PRECAUTIONS, 2553*
- PAINTS, SOLVENTS, CHEMICALS
  - BLADDER CANCER, CLINICAL STUDY, 2920
- RUBBER CHEMICALS
  - DUPONT, 2947*
- TALC
  - REVIEW, 2427*

- ORAL CANCER
  - ETIOLOGY
    - PATHOGENESIS, REVIEW, 2436*

- ORAL CAVITY
  - TUMORS
    - OCCURRENCE, KENYA, 2940*

- OROPHARYNGEAL CARCINOMA
  - IMMUNOGLOBULIN IGA
    - SALIVA, HUMAN, 2782*

- ORTHO-TOLUIDINE
  - RHABDOMYOSARCOMA
    - HEART, RAT, 2563*

- OSTEOSARCOMA
  - NATURALLY OCCURRING VIRUS
    - BIOLOGICAL CHARACTERISTICS, MOUSE, 2641
  - RADIATION, STRONTIUM 90
    - BEAGLE, 2585

- OVARIAN CARCINOMA
  - ANALYTICAL APPROACHES
    - REVIEW, 2459*
  - ANTIGEN
    - HUMAN, 2827*
  - CLINICAL SURVEY
    - REVIEW, 2424*
  - CYTOGENETIC STUDIES
    - REVIEW, 2453*
  - ESTRADIOL UPTAKE
    - BINDING-SITE STUDIES, REVIEW, 2433*
  - EXPERIMENTAL APPROACHES
    - REVIEW, 2432*
  - PLASMA GONADATROPHIN LEVELS
    - STERILE MOUSE, 2912*
  - PREGNANCY
    - CLINICAL STUDY, 2927
  - STATISTICAL ASPECTS OF A CO-OPERATIVE TRIAL
    - REVIEW, 2423*

- OVARIAN TUMOR
  - INDUCTION
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE, RAT, 2494

- OVARY
  - BRENNER TUMOR
    - ULTRASTRUCTURE, HUMAN, 2896

- (2-OXOPROPYL)PROPYLNITROSAMINE
  - HEPATOMA
    - HAMSTER, 2485
  - RESPIRATORY SYSTEM TUMORS
    - HAMSTER, 2485

- PANCREATIC CANCER
  - STOMACH CANCER
    - MORTALITY, REVIEW, 2419*

- PAPILLOMA
  - VIRUS, SHOPE
    - NUCLEIC ACID PRECURSOR UPTAKE, RABBIT, 2654

- PAPILLOMA, SQUAMOUS CELL
  - DIBUTYLNITROSOAMINE
    - ESOPHAGUS, RAT, 2565*

- PATULIN
  - PRODUCTION
    - BIOLOGICAL FACTORS, REVIEW, 2468*

- PENILE CANCER
  - EPIDEMIOLOGY
    - HUMAN, REVIEW, 2407

- PHENOBARBITAL
  - TOXICITY
    - LONG TERM STUDIES, MOUSE, 2508

- PHENOBARBITOL
  - CARBON TETRACHLORIDE HEPATOTOXICITY
    - RAT, 2567*

- PHORBOL MYRISTATE ACETATE
  - TUMOR-PROMOTING AGENT
    - SKIN, MOUSE, 2525

- PHYTOHEMAGGLUTININ
  - EFFECT ON PERIPHERAL LYMPHOCYTES
    - CHRONIC LYMPHOCYTIC LEUKEMIA, 2856*
  - GROWTH, TUMOR
    - FIBROSARCOMA-BEARING MICE, 2792*
  - LYMPHOCYTE RESPONSE
    - CANCER PATIENTS, 2762
  - LYMPHOCYTE TRANSFORMATION
    - HUMAN CHORIONIC GONADOTROPHIN EFFECT, 2866*
    - HUMAN CHORIONIC SOMATOMAMMOTROPHIN EFFECT, 2866*
    - LARGE BOWEL CANCER, HUMAN, 2848*
  - TRANSFORMATION
    - B CELL, 2840*

- PITUITARY
  - NEOPLASTIC CELLS
    - HORMONAL ACTIVITIES, HISTOCHEMICAL STAINING, MOUSE, RAT, 2853*

- PITUITARY GLAND

- RADIATION-INDUCED CARCINOGENESIS
    - DYNAMICS, RAT, 2602*

PLASMA
- ROUS SARCOMA VIRUS PROLIFERATION
    - FIBROBLASTS, CHICKEN, 2640

PLASMA CELL
- NEOPLASMS
    - IMMUNOGLOBULIN STRUCTURE, REVIEW, 2421*

PLASMA MEMBRANE
- CONCANAVALIN A BINDING SITES
    - NORMAL, TUMOR, TRANSFORMED CELLS, 2955

PLASMACYTOMA
- CHRONIC LYMPHOID LEUKEMIA,RECTAL CARCINOMA
    - FAMILIAL OCCURRENCE, CASE REPORT, 3000*
- DEVELOPMENTAL HISTORY
    - MOUSE, REVIEW, 2469*
- IMMUNOGLOBULIN SYNTHESIS
    - MEMBRANE-BOUND, FREE POLYSOMES, MOUSE, 2721

PLATINUM
- -INDUCED PIGMENTED TUMORS
    - EYE, RAT, 2572*

POLYINOSINIC:POLYCYTIDYLIC ACID
- SKIN CANCER
    - TWO-STAGE INHIBITION, MOUSE, 2735

POLYRIBOSOME
- LEUKEMIA
    - PHYSICOCHEMISTRY, RAT, 2960
- LIVER, SPLEEN MALIGNANT CELLS
    - PHYSICOCHEMISTRY, RAT, 2960

PREGNANCY
- SUPPRESSION OF DMBA-TUMOR INDUCTION
    - RAT, 2519

PRELEUKEMIA
- REVIEW, 2451*

PROGESTERONE
- IMPLANTATION
    - MAMMARY TUMOR, ISOGRAFT-BEARING ORCHIDECTOMIZED MICE, 2757

PROLACTIN
- LEVELS
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED MAMMARY TUMORS, RAT, 2512
    - MAMMARY TUMOR, ISOGRAFT-BEARING ORCHIDECTOMIZED MICE, 2757

PROSTATE
- BENIGN HYPERPLASIA, ADENOCARCINOMA
    - ULTRASTRUCTURE, HUMAN, 2962
- CARCINOMA
    - ENZYME CYTOCHEMISTRY, ELECTRON MICROSCOPY, 2969
    - GONORRHEA, EPIDEMIOLOGY, 2926
- HYPERPLASIA
    - ENZYME ACTIVITY, HUMAN, 2891
- TUMOR, MALIGNANT
    - ENZYME ACTIVITY, HUMAN, 2891
    - MORPHOLOGY, HUMAN, 2891

PROTEIN
- BRAIN-SPECIFIC
    - NEUROBLASTOMA CELLS, MOUSE, 2846*
- BRONCHIAL MUCOSA
    - AGAROSE-GEL ELECTROPHORESIS, 2540*
- BRONCHOGENIC CARCINOMA
    - AGAROSE-GEL ELECTROPHORESIS, 2540*
- CARCINOGEN-BINDING
    - NORMAL AND TUMOR CELLS, RAT, 2564*
- EOSINOPHIL GRANULES
    - ULTRASTRUCTURE,, GUINEA PIG, 2779*
- IMMUNOSUPPRESSIVE
    - PLASMA, HUMAN, 2781*
- NON-HISTONE
    - ANGIOGENESIS, WALKER CARCINOMA, RAT, 2973
- PHOTOCHEMICAL REACTIONS
    - REVIEW, 2452*

PROTEIN, CAPSID
- VIRUS, ADENO 1
    - IMMUNOFLUORESCENCE AND CYTOFLUOROMETRIC DATA, 2679*

PROTEIN, NUCLEAR
- SYNTHESIS
    - MYELOMA CELLS, MOUSE, 2766

PULMONARY ADENOCARCINOMA
- DIBUTYLNITROSOAMINE
    - RAT, 2565*

PULMONARY ADENOMA
- DIETHYLNITROSAMINE
    - ULTRASTRUCTURE, HAMSTER, 2573*

PURPURA, MONOCLONAL HYPERGLOBULINEMIC
- MYELOMA, MULTIPLE
    - IGG ANTI-IGG COMPLEXES, 2861*

PYRIMETHAMINE
- LEUKEMIA, CENTRAL NERVOUS SYSTEM
    - REVIEW, 2455*

PYRUVATE CARBOXYLASE
- HEPATOMA
    - ENZYMATIC, IMMUNOLOGIC STUDY, RAT, 2720

RADIATION
- CARCINOGENESIS
    - ATOMIC BOMB SURVIVORS, REVIEW, 2403
    - LATE EFFECTS, REVIEW, 2403
    - REVIEW, 2408
- IMMUNITY
    - CANCER, REVIEW, 2401
- IMMUNODEPRESSION
    - CANCER, REVIEW, 2401
- LEUKEMIA
    - REVIEW, 2408
- MUTAGENESIS
    - HUMAN LEUKOCYTES, MOUSE

    - FIBROBLASTS, TISSUE CULTURE, 2603*
  - POLYPS, INTESTINAL
    - DOG, RAT, 2600*

- RADIATION, GAMMA
  - CARCINOGENESIS
    - OVARIES, HYPOPHYSIS, ADRENALS, RAT, 2602*
  - DNA
    - INFRARED SPECTROSCOPY, CALF, 2584
  - RHABDOMYOSARCOMA
    - DNA CONTENT, CELL VOLUME, RAT, 2599*

- RADIATION, UV
  - CARCINOGENESIS
    - CHOLESTEROL OXIDE FORMATION, MOUSE, 2583
  - CHROMOSOME ABERRATIONS
    - REPAIR ACTIVITY, MAMMALS, 2596*
  - DNA PROTECTION
    - MELANIN PIGMENT, MOUSE, 2594*
  - GRAIN DISTRIBUTION
    - PIGMENTED, UNPIGMENTED SKIN, GUINEA PIGS, 2593*
  - MUTATION
    - ASPARGININE REQUIREMENT, JENSEN SARCOMA CELLS, 2595*
  - SQUAMOUS CELL CARCINOMA
    - CHOLESTEROL OXIDE FORMATION, MOUSE, 2583

- RADIATION, X-RAY
  - BONE MARROW
    - REPOPULATING ABILITY, MOUSE, 2605*
  - CELL DIVISION
    - HELA CELLS, HAMSTER, 2608*
  - CHROMOSOME ABERRATIONS
    - LEUKOCYTES, SPERMATOGONIAL CELLS, MOUSE, HAMSTER, 2609*
  - DNA SYNTHESIS
    - LYMPHOMA, RAT, 2611*
  - EHRLICH ASCITES TUMOR
    - LETHALITY ENHANCED BY AMINOHYDROXYTHYMINE HYDRAZINE, MOUSE, 2601*
  - GLIAL TUMOR CELLS
    - MOPHOLOGICAL MODIFICATION, RAT, 2606*
  - INTRA-UTERINE
    - LEUKEMIA, CHILDREN, 2604*
  - LEUKOSIS, LYMFHOID
    - CHICK, 2589
  - LUNG TUMOR
    - CLONOGENIC GROWTH, RAT, 2598*
    - EXPOSURE RESPONSE, MOUSE, 2607*
  - MAMMARY TUMOR
    - RAT, 2597*
  - RHABDOMYOSARCOMA
    - DNA CONTENT, CELL VOLUME, RAT, 2599*
  - THYROID CARCINOMA
    - CLINICAL STUDY, 2586
  - TUMOR CELL IMMUNIZATION
    - SERUM INHIBITORY EFFECT, CANCER PATIENTS, 2702

- RECTAL CARCINOMA
  - CHRONIC LYMPHOID LEUKEMIA, PLASMACYTOMA
    - FAMILIAL OCCURRENCE, CASE REPORT, 3000*
  - 1,2-DIMETHYLHYDRAZINE
    - EXPERIMENTAL MODEL, RAT, 2560*
  - EPIDEMIOLOGY
    - REVIEW, 2476*

- REGAN ISOENZYME
  - ASSAY
    - SERUM, NEOPLASTIC DISEASE, HUMAN, 2864*

- REGRESSION, TUMOR
  - ESTRONE-INDUCED MAMMARY TUMOR
    - ENZYME ACTIVITIES, RAT, 2551*
  - LUNG METASTASES, TESTICULAR TUMOR
    - HUMAN, 2824*
  - VIRUS, MOLONEY SARCOMA
    - LYMPHOCYTE, MOUSE, 2723

- RENAL CARCINOMA
  - TEMPERATURE SENSITIVE VIRAL INDUCTION
    - FROG, 2630

- RESISTANCE
  - ETHIDIUM BROMIDE
    - SV40-TRANSFORMED HAMSTER CELLS, 2687*
  - INDUCED ALLOGENEIC GRAFT REJECTION
    - AUTOCHTHONOUS GRAFT, SPONTANEOUS MAMMARY TUMOR, MOUSE, 2883*
  - LEUKEMIA TRANSPLANTATION
    - NEURAMINIDASE-TREATED LEUKEMIA CELL-IMMUNIZED MICE, 2886*
  - TRANSPLANTATION
    - RADIOSTRONTIUM-INDUCED OSTEOSARCOMA, MOUSE, 2755
  - TUMOR TRANSPLANTATION
    - EFFECT OF PRESENCE OF TUMOR ANTIGEN IN VIVO, 2882*

- RESPIRATORY SYSTEM TUMORS
  - B16 MELANOMA CELLS
    - MOUSE, 2895
  - DIPROPYLNITROSAMINE
    - HAMSTER, 2485
  - (2-HYDROXYPROPYL)PROPYLNITROSAMINE
    - HAMSTER, 2485
  - METASTASIS
    - INCIDENCE, MOUSE, 2895
  - METHYLPROPYLNITROSAMINE
    - HAMSTER, 2485
  - (2-OXOPROPYL)PROPYLNITROSAMINE
    - HAMSTER, 2485

- RETICULOENDOTHELIAL SYSTEM
  - LOADING WITH COLLOID
    - INHIBITION, ACCELERATION, RAUSCHER LEUKEMIA, MOUSE, 2633

- RETICULUM CELL SARCOMA
  - ANTILYMPHOCYTE GLOBULIN INJECTION SITE
    - CASE REPORT, 2742
  - THYMUS GRAFT
    - MOUSE, 2718
  - TUMOR-SPECIFIC TRANSPLANTATION ANTIGENS

- FRIEND VIRUS COMPLEX, 2663*
- VIRUS, MURINE LEUKEMIA
  - INDUCTION, MOUSE, 2620

REVERSE TRANSCRIPTASE
- VIRUS, RNA TUMOR
  - LEUKEMIA, MOLECULAR BIOLOGY, REVIEW, 2412

RHABDOMYOSARCOMA
- HEART
  - ORTHO-TOLUIDINE TREATMENT, RAT, 2563*
- IRRADIATED TUMOR GROWTH
  - MATHEMATICAL MODEL, RAT, 2590*
- PHOTOMETRIC DNA DETERMINATION
  - CELL GROWTH, MOUSE, 2996*
- RADIATION, GAMMA
  - DNA CONTENT, CELL VOLUME, RAT, 2599*
- RADIATION, X-RAY
  - DNA CONTENT, CELL VOLUME, RAT, 2599*

RIBOFLAVIN
- GROWTH, TUMOR
  - CANCER, REVIEW, 2413

RNA
- ACCUMULATION
  - LYMPHOCYTE TRANSFORMATION, 2825*
- LEUKEMIA
  - HUMAN, 2976
  - LABELLING PATTERNS, 2974
- PLASMACYTOMA
  - MOUSE, 2828*
- TRANSFORMATION, EPIGENETIC
  - HUMAN NEOPLASTIC CELLS, REVIEW, 2415
- TUMOR-SPECIFIC
  - MOLECULAR HYBRIDIZATION, MAMMARY TUMOR MOUSE, 2657
- VIRUS, AVIAN MYELOBLASTOSIS
  - SEDIMENTATION PROFILE, CHICK EMBRYO FIBROBLASTS, 2613
- VIRUS-SPECIFIC
  - SV40-INDUCED CHROMOSOME REPLICATION, MITOSIS, MOUSE CELL CULTUR, 2645

RNA, MESSENGER
- MUSCLE DIFFERENTIATION
  - CELL CULTURE, CHICKEN, 2644

RNA POLYMERASE
- MELANOMA
  - PROPERTIES, HUMAN, 2980
- RNA DEPENDENT
  - ROUS SARCOMA VIRUS VIRIONS, 2683*

RNA REPLICASE
- RNA DEPENDENT
  - IMMUNE RESPONSE, MOUSE, 2691

RNA SYNTHESIS
- DNA TEMPLATE
  - TEMPERATURE SENSITIVE VIRUS, FROG, 2630
- LEUKOCYTE
  - INFECTIOUS MONONUCLEOSIS, HUMAN, 2748

RNA TRANSCRIPTION
- DNA POLYMERASE
  - LEUKEMIC CELLS, HUMAN, 2622

RNA, TRANSFER
- NUCLEOSIDE COMPONENTS
  - URINARY EXCRETION, CANCER PATIENTS, 2968

SACCHARIN
- URINARY BLADDER CARCINOMA
  - RAT, 2545*

SALIVARY GLAND
- TUMORS
  - OCCURRENCE, KENYA, 2940*

SARCOMA
- INDUCTION, AVIAN VIRUS
  - SPECIES, AGE RESISTANCE, MOUSE, 2626
- VIROLOGICAL RESEARCH
  - HUMAN, REVIEW, 2472*
- VIRUS, RNA TUMOR
  - HUMAN, 2632

SARCOMA, JENSEN
- UV RADIATION
  - ASPARGININE REQUIREMENT, MUTATION, 2595*

SELENIUM
- STOMACH CANCER MORTALITY
  - REVIEW, 2431*

SERUM
- BLOCKING
  - ALLOGRAFTS, RAT, 2780*
- INHIBITORY EFFECT
  - IRRADIATED TUMOR CELL IMMUNIZATION, CANCER PATIENTS, 2702
- LYMPHOCYTE
  - HUMORAL IMMUNITY, GUINEA PIG, 2796*
- ROUS SARCOMA VIRUS PROLIFERATION
  - FIBROBLASTS, CHICKEN, 2640

SILICOTUNGSTATE
- VIRUS, RNA TUMOR
  - IN VITRO EFFECTS, 2678*

SKIN
- BENZENE
  - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
- HYDROGENATED TERPHENYL
  - NUCLEAR REACTOR COOLANT, OCCUPATIONAL HAZARD, 2491
- MALIGNANT MELANOMA
  - EPIDEMIOLOGY, CALIFORNIA, 2916
- MELANOMA, MALIGNANT
  - INCIDENCE, ENGLAND, 2925
- 20-METHYLCHOLANTHRENE
  - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
- TOBACCO SMOKE CONDENSATE

-
  -
    - COCARCINOGENICITY, MOUSE, 2535
  - TUMOR-PROMOTING AGENT
    - PHORBOL MYRISTATE ACETATE, MOUSE, 2525
  - UV RADIATION
    - PIGMENTED, UNPIGMENTED, GUINEA PIG, 2593*

- SKIN CANCER
  - EPIDEMIOLOGY
    - HUMAN, REVIEW, 2407
  - POLYINOSINIC:POLYCYTIDYLIC ACID
    - TWO-STAGE INHIBITION, MOUSE, 2735

- SPLEEN
  - BENZENE
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*
  - 20-METHYLCHOLANTHRENE
    - DNA SYNTHESIS, ERYTHROPOIETIC SERUM, MOUSE, 2538*, 2574*

- SQUAMOUS CELL CARCINOMA
  - ANTIGEN
    - UREA, MOUSE, 2703
  - RADIATION, UV
    - CHOLESTEROL OXIDE FORMATION, MOUSE, 2583

- SQUAMOUS CELL PAPILLOMA
  - ESOPHAGUS
    - DIBUTYLNITROSOAMINE, RAT, 2565*

- STERIGMATOCYSTIN
  - CONVERSION TO AFLATOXIN B1
    - ASPERGILLUS PARASITICUS, 2544*
  - PRODUCTION
    - BIOLOGICAL FACTORS, REVIEW, 2468*

- STEROID REDUCTASE
  - ADRENOCORTICAL CARCINOMA
    - RAT, 2965

- STOMACH
  - PRECANCEROUS STATE
    - DIAGNOSTIC PROBLEMS, HUMAN, 2907*

- STOMACH CANCER
  - MORTALITY
    - ANTIOXIDANTS IN FOOD, REVIEW, 2431*
  - PANCREATIC CANCER
    - MORTALITY, REVIEW, 2419*

- STOMACH CARCINOMA
  - N-(4-(5-NITRO-2-FURYL)-2-THIAZOLYL) ACETAMIDE
    - EFFECT OF SPLENECTOMY, MOUSE, 2582*
    - EFFECT OF THYMECTOMY, MOUSE, 2582*

- STRONTIUM 90
  - BONE TUMOR
    - BEAGLE, 2585
    - ESTROGEN EFFECT MOUSE, 2587

- TALC
  - ENVIRONMENTAL HAZARD
    - REVIEW, 2427*
  - OCCUPATIONAL HAZARD
    - REVIEW, 2427*

- THEOPHYLLINE
  - CELL-SURFACE ANTIGEN
    - CULTURED CELLS, MOUSE, 2800*

- THYMUS
  - DIETHYLNITROSAMINE CARCINOGENESIS INHIBITION
    - LIVER, RAT, 2496
  - GRAFTS
    - RETICULUM CELL SARCOMA INCIDENCE, MOUSE, 2718
  - MAGNESIUM DEFICIENCY
    - HYPERPLASIA, RAT, 2752
  - RECOVERY
    - TUMOR REMOVAL, TUMOR-BEARING RATS, 2788*

- THYROID CARCINOMA
  - IODINE 131
    - CASE REPORTS, 2610*
  - RADIATION, X-RAY
    - CLINICAL STUDY, 2586
  - SURGERY AND RADIOIODINE THERAPY
    - HYPERTHYROIDISM, HUMAN, 2588

- THYROIDITIS, CHRONIC
  - N-2-FLUORENYLDIACETAMIDE
    - RAT, 2556*
  - 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    - RAT, 2556*

- TOBACCO SMOKE CONDENSATE
  - COCARCINOGENICITY
    - SKIN, MOUSE, 2535

- TOBACCO SMOKING
  - IMMUNITY
    - REVIEW, 2454*
  - LUNG CANCER
    - AGE-SPECIFIC DEATH RATES, 2938*
    - CIGAR, PIPE, RETROSPECTIVE STUDY, 2932
    - HUMAN, 2561*

- TRANSACETYLATION
  - N-HYDROXY-4-ACETYLAMINOBIPHENYL
    - MAMMARY GLAND, RAT, 2479

- TRANSFORMATION
  - BLASTOGENIC
    - ASSAY, IMMUNOLOGIC REACTIVITY TO AVIAN ONCORNAVIRUSES, CHICKEN, 2753
  - FIBROBLAST
    - INHIBITION BY CHICK INTERFERON, 2618
  - LYMPHOCYTE
    - INDUCTION BY CONCANAVALIN A, ACETYL CONCANAVALIN A, HUMAN, 2860*
    - LEUKEMIA VIRUS, IMMUNE RESPONSE, MOUSE, 2656
    - MALIGNANT DISEASE, 2769*
    - NONSPECIFIC CYTOTOXICITY, 2794*
    - PHYTOHEMAGGLUTININ, HUMAN CHORIONIC GONADOTROPHIN EFFECT,

        2866*
      - PHYTOHEMAGGLUTININ, HUMAN CHORIONIC SOMATOMAMMOTROPHIN EFFECT, 2866*
      - PHYTOHEMAGGLUTININ, LARGE BOWEL CANCER, HUMAN, 2848*
      - RNA ACCUMULATION, 2825*
    - PHYTOHEMAGGLUTININ
      - B CELL, 2840*
    - VIRUS, AVIAN MYELOCYTOMATOSIS
      - TARGET CELLS, CHICK EMBRYO CELLS, 2646
    - VIRUS, EPSTEIN-BARR
      - CELL CULTURE, HUMAN, 2897
    - VIRUS, POLYOMA
      - CELL TISSUE CULTURE, REVIEW, 2463*
    - VIRUS, ROUS SARCOMA
      - INHIBITION, CHICK EMBRYO FIBROBLASTS, 2618
    - VIRUS, SV40
      - ETHIDIUM BROMIDE RESISTANCE, HAMSTER CELLS, 2687*

- TRANSFORMATION, EPIGENETIC
  - RNA
    - HUMAN NEOPLASTIC CELLS, REVIEW, 2415

- TRANSFORMATION, MALIGNANT
  - BLASTIC, LYMPHOCYTE
    - ANTILYMPHOCYTE GLOBULIN INJECTION SITE, CASE REPORT, 2742
  - HYPOXIA
    - CELL CULTURE, RAT, 2898
  - MORPHOLOGY, BIOCHEMISTRY
    - DISCUSSION, 2997*
  - VIRUS ACTIVATION
    - CELL CULTURE, HUMAN, 2612
  - VIRUS, CYTOMEGALO-
    - EMBRYO FIBROBLASTS, HAMSTER, 2658

- TRANSPLACENTAL CARCINOGENESIS
  - REVIEW, 2450*, 2581*

- TRANSPLANTATION
  - AGE
    - WALKER 256 CARCINOSARCOMA, RAT, 2821*
  - ANTIGEN
    - CELLULAR IMMUNITY, REVIEW, 2430*
    - SARCOMA, SYNGENEIC, MOUSE, 2724
  - DIETHYLNITROSAMINE-INDUCED TUMOR
    - GUINEA PIG, 2488
  - IMMUNE RESPONSE
    - LYMPHOMA, MOUSE, 2726
  - METHYLCHOLANTHRENE-INDUCED TUMOR
    - GUINEA PIG, 2488
  - NEURAMINIDASE-INDUCED LOSS
    - LEUKEMIA, MOUSE, 2886*
  - RESISTANCE
    - RADIOSTRONTIUM-INDUCED OSTEOSARCOMA, MOUSE, 2755
  - SARCOMA, SYNGENEIC
    - LYMPH NODE RESPONSE, MOUSE, 2724

- TRI-P-ANISYLCHLORETHYLENE
  - SUPPRESSION OF DMBA-TUMOR INDUCTION
    - RAT, 2519

- 7,8,12-TRIMETHYLBENZANTHRACENE
  - MAMMARY TUMOR
    - EFFECT OF
      - DIMETHYLBENZYLDESMETHYLRIFAMPICIN, RAT, 2537*

- TUMOR
  - CELL DIVISION
    - CIRCADIAN RHYTHM, 2950*
  - IMMUNOLOGY
    - CLINICAL ASPECTS, 2873*
  - PLANT
    - AGROBACTERIUM TUMEFACIENS, CROWN-GALL, 2668*
  - REMOVAL
    - THYMUS RECOVERY, TUMOR-BEARING RATS, 2788*

- URETHANE
  - DNA REPLICATION
    - ENZYME INDUCTION, RAT LIVER, 2495
    - PARTIAL HEPATECTOMY, RAT, 2495

- URIDINE KINASE
  - LEVELS
    - POLYOMA VIRUS-INFECTED MOUSE CELLS, 2677*
    - PSEUDORABIES VIRUS-INFECTED MOUSE CELLS, 2677*
    - VESICULAR STOMATITIS VIRUS-INFECTED MOUSE CELLS, 2677*
    - WESTERN EQUINE ENCEPHALOMYELITIS-INFECTED MOUSE CELLS, 2677*

- URINARY BLADDER
  - ACETAMIDES
    - N-OXIDATION, DOG, MONKEY, 2510
  - AMINES, AROMATIC
    - N-OXIDATION, DOG, MONKEY, 2510
  - NITRO COMPOUNDS
    - N-OXIDATION, DOG, MONKEY, 2510

- URINARY BLADDER CARCINOMA
  - METHYLNITROSOUREA
    - RAT, 2545*
  - PAINTS, SOLVENTS, CHEMICALS
    - OCCUPATIONAL HAZARD, CLINICAL STUDY, 2920
  - SACCHARIN
    - RAT, 2545*

- URINARY BLADDER TUMOR
  - BASIC LEAD ACETATE
    - RAT, 2576*
  - N-BUTYL-N(4-HYDROXYBUTYL)NITROSAMINE
    - RAT, 2576*
  - DIBUTYLNITROSOAMINE
    - RAT, 2565*
  - 3-METHYLCHOLANTHRENE
    - RAT, 2576*
  - 4-NITROQUINOLINE-1-OXIDE
    - RAT, 2576*
  - N-NITROSODIBUTYLAMINE
    - RAT, 2576*
  - N-NITROSODIMETHYLAMINE
    - RAT, 2576*

- UTERINE CERVIX
  - 20-METHYLCHOLANTHRENE

    - ULTRASTRUCTURE, CASTRATION, RAT, 2577*

UTERUS
- MESOTHELIOMA, MALIGNANT
    - DIETHYLSTILBESTROL, MONKEY, 2529
- 3-METHYLCHOLANTHRENE
    - HISTOLOGICAL CHANGES, MOUSE, 2480

VIRUS
- ADENO 2-SV40 HYBRID
    - SPECIFIC POLYPEPTIDE, 2686*
- CANCER
    - ETIOLOGY, REVIEW, 2422*
    - EXPERIMENTAL APPROACHES, REVIEW, 2442*
- CARCINOGENESIS MECHANISMS
    - GENERAL ASPECTS, 2685*
- HOST CELL-ANIMAL VIRUS INTERACTIONS
    - REVIEW, 2406
- -INDUCED CHROMOSOMAL CHANGES
    - REVIEW, 2428*
- INFECTIONS
    - EFFECT OF METALS, MOUSE, 2536*
- LEUKEMIA
    - ETIOLOGY, HUMAN, 2634

VIRUS, A-TYPE PARTICLE
- FILAMENTOUS FORMS
    - HEPATOMAS, RAT CELL CULTURE, 2509

VIRUS, ACUTE LYMPHOCYTIC CHORIOMENINGITIS
- IMMUNE RESPONSE
    - GENETIC CONTROL, MOUSE, 2712

VIRUS, ADENO 1
- PROTEIN, CAPSID
    - IMMUNOFLUORESCENCE AND CYTOFLUOROMETRIC DATA, 2679*

VIRUS, ADENO 2
- DNA REPLICATION
    - KB CELLS, 2650

VIRUS, ADENO 5
- ANTISERUM
    - CYTOTOXIC AGGREGATE INDUCTION, 2666*

VIRUS, ADENO 12
- STRUCTURAL POLYPEPTIDES
    - SYNTHESIS, HUMAN EMBRYONIC KIDNEY CELLS, 2648

VIRUS, AVIAN LEUKOSIS-SARCOMA
- ANTIGEN, CELL-SURFACE
    - CHICKEN, 2753

VIRUS, AVIAN MYELOBLASTOSIS
- RNA
    - SEDIMENTATION PROFILE, CHICK EMBRYO FIBROBLASTS, 2613

VIRUS, AVIAN MYELOCYTOMATOSIS
- TRANSFORMATION
    - TARGET CELLS, CHICK EMBRYO CELLS, 2646

VIRUS, AVIAN SARCOMA
- TUMOR INDUCTION
    - SPECIES, AGE RESISTANCE, MOUSE, 2626

VIRUS, B-TYPE
- DNA POLYMERASE
    - MAMMARY TUMOR, MOUSE, 2617

VIRUS, C-TYPE
- ANTIGEN
    - NORMAL CELLS AND TISSUES, MOUSE, 2859*
- LEUKEMIA, ACUTE
    - ENZYME, HUMAN, 2622
- LYMPHOSARCOMA
    - CATTLE, 2637

VIRUS, C-TYPE PARTICLE
- AVIAN BURSA
    - FABRICIUS CELLS, 2672*
- LYMPHOCYTE NUCLEAR POCKETS
    - HEMATOLOGY, ULTRASTRUCTURE, BULL, 2636

VIRUS, C-TYPE RNA
- COMPARATIVE SEROLOGICAL STUDIES
    - MOUSE, RAT, CAT, HAMSTER, MONKEY, 2767
- LEUKEMIA
    - CAT, 2662
    - HUMAN, REVIEW, 2472*
- PREVALANCE WITH NEOPLASIA
    - CAT, 2662
- SARCOMA
    - HUMAN, REVIEW, 2472*

VIRUS, CYTOMEGALO-
- TRANSFORMATION, MALIGNANT
    - EMBRYO FIBROBLASTS, HAMSTER, 2658

VIRUS, EPSTEIN-BARR
- ANTIBODY
    - BURKITT'S LYMPHOMA PATIENTS, 2727
- ANTIGEN
    - LYMPHOBLASTOID CELL LINES, HUMAN, 2759

VIRUS, EPSTEIN BARR
- BURKITT SOMATIC CELL HYBRID
    - PREFERENTIAL SELECTION, VIRUS GENOME, HUMAN CELL LINE, 2643

VIRUS, EPSTEIN-BARR
- DNA
    - LYMPHOBLASTOID CELL LINES, HUMAN, 2759
- INFECTION
    - CHROMOSOME ABERRATIONS, LYMPHOBLASTOID CELL LINE, 2619
- NASOPHARYNGEAL CARCINOMA
    - ANTIGEN, TUMOR BIOPSIES, 2710
    - CELL CULTURE TRANSFORMATION, 2897
- VIRAL GENOME ACTIVATION
    - LYMPHOBLASTOID CELLS, HUMAN, 2625

VIRUS, FELINE LEUKEMIA
- HORIZONTAL TRANSMISSION
    - CAT, 2921
- SYNCYTIAL GIANT CELL

    - FOCUS ASSAY, CANINE AND RAT CELL LINES, 2629

- VIRUS, FELINE SARCOMA
  - TRANSMISSION
    - CROSS SPECIES, 2615

- VIRUS, FIBROMA
  - ISOLATION FROM NATURALLY-OCCURRING SKIN TUMORS
    - VIROLOGIC AND ANTIGENIC PROPERTIES, RABBIT, 2671*

- VIRUS, FRIEND LEUKEMIA
  - INFECTION
    - GENETIC CONTROL, MOUSE, 2651
  - SPLEEN COLONY-FORMING UNITS
    - HEMOPOIESIS, MOUSE, 2627
  - SUSCEPTIBILITY
    - GENETIC CONTROL, MOUSE, 2651
  - TRANSMISSION
    - CROSS SPECIES, 2615
  - VIRAL CORE
    - ISOLATION AND CONSTITUENTS, 2665*

- VIRUS, FRIEND SPLEEN FOCUS-FORMING
  - TARGET CELLS
    - ASSAY, 2884*

- VIRUS, GROSS
  - SPECIFIC ANTIGEN
    - BIOCHEMICAL ANALYSIS, 2670*

- VIRUS, GROSS LEUKEMIA
  - IMMUNITY
    - ROUTES OF TRANSMISSION, RAT, 2690*

- VIRUS, HERPES
  - CERVICAL CARCINOMA
    - REVIEW, 2437*
  - GENERAL ASPECTS
    - REVIEW, 2440*
  - ONCOGENICITY
    - REVIEW, 2458*

- VIRUS, HERPES 1
  - ANTIBODY
    - HUMAN SERA, REVIEW, 2445*

- VIRUS, HERPES 2
  - ANTIBODY
    - HUMAN SERA, REVIEW, 2445*
  - CARCINOGENESIS
    - HUMAN, REVIEW, 2404
  - CERVICAL CARCINOMA
    - HUMAN, REVIEW, 2411
    - NUCLEIC ACID HYBRIDIZATION, HUMAN, REVIEW, 2417
    - SEROEPIDEMIOLOGIC STUDIES, 2917
  - REVIEW, 2441*, 2443*, 2446*

- VIRUS, HERPES SIMPLEX
  - ANTIGEN
    - ORAL AND GENITAL ORIGIN, REVIEW, 2444*
  - LATENT INFECTION
    - MOUSE, RABBIT, 2652
  - NOVIRION ANTIGEN
    - DETECTION, CELL CULTURE, 2751

- VIRUS, LEUKEMIA
  - INTERFERON
    - GRAFT VERSUS HOST REACTION, MOUSE, 2674*

- VIRUS, MAMMARY TUMOR
  - ANTIBODY
    - OCCURRENCE, NATURALLY INFECTED MICE, 2719
  - DNA POLYMERASE
    - MOUSE, 2617
  - RNA-DEPENDENT DNA POLYMERASE
    - MOUSE, 2660
  - RNA, VIRUS-SPECIFIC
    - MOLECULAR HYBRIDIZATION, MOUSE, 2657

- VIRUS, MAREK'S DISEASE
  - CELL-FREE PREPARATIONS
    - POULTRY DUST, 2682*

- VIRUS, MOLONEY SARCOMA
  - IMMUNE RESPONSE
    - LYMPHOCYTE, MOUSE, 2723
  - PROTECTION BY IMMUNE SPLEEN CELLS
    - EFFECT OF STAGE OF DONOR RESPONSE, 2676*
  - REGRESSION, TUMOR
    - LYMPHOCYTE, MOUSE, 2723
  - RNA SPECIES IN HELPER-INDEPENDENT STRAIN
    - RAT CELL LINE, 2649
  - TUMOR INDUCTION
    - LYMPHOCYTE, MOUSE, 2723

- VIRUS, MURINE LEUKEMIA
  - DIETHYLAMINOETHYL-DEXTRAN
    - POTENTIATION, MOUSE, 2635
  - GIANT CELL FORMATION
    - CULTURED CELLS, 2669*
  - IMMUNE RESPONSE
    - LYMPHOCYTE TRANSFORMATION, MOUSE, 2656
  - RETICULUM CELL SARCOMA
    - INDUCTION, MOUSE, 2620
  - TUMOR INDUCTION
    - MOUSE, 2655

- VIRUS, MURINE LEUKEMIA-SARCOMA
  - INTERFERON PRODUCTION
    - EMBRYO FIBROBLASTS, MOUSE, 2661

- VIRUS, MURINE SARCOMA
  - NATURAL OCCURRENCE
    - OSTEOSARCOMA, MOUSE, 2641

- VIRUS, POLYOMA
  - ANTIBODY
    - TUMOR REJECTION, MOUSE, 2725
  - ANTISERUM
    - ANTIGEN, MOUSE, 2725
  - INFECTION
    - URIDINE KINASE LEVEL, MOUSE EMBRYO CELLS, 2677*
  - KIDNEY
    - ALLOGRAFT RECIPIENTS, CLINICAL STUDY, 2638
  - NEOPLASM INDUCTION
    - MOUSE, 2725

- TRANSFORMATION
  - CELL TISSUE CULTURE, REVIEW, 2463*

VIRUS, PSEUDORABIES
- INFECTION
  - URIDINE KINASE LEVEL, MOUSE EMBRYO CELLS, 2677*

VIRUS, RAUSCHER LEUKEMIA
- AUTOIMMUNITY
  - ERYTHROCYTE CLEARANCE STUDY, MOUSE, 2628
- DIETHYLAMINOETHYL-DEXTRAN
  - POTENTIATION, MOUSE, 2635
- IMMUNE RESPONSE
  - CELL-MEDIATED, MOUSE, 2717
- INFECTION
  - GROWTH OF BONE MARROW CELLS, MOUSE, 2688*
- INHIBITION, ACCELERATION
  - COLLODIAL LOADING, RETICULOENDOTHELIAL SYSTEM, MOUSE, 2633

VIRUS, RETICULOENDOTHELIOSIS
- IMMUNOLOGY
  - TURKEY, DUCK, CHICKEN, 2642
- MORPHOLOGY
  - TURKEY, DUCK, CHICKEN, 2642
- TRANSMISSION
  - TURKEY, DUCK, CHICKEN, 2642

VIRUS, RNA-LIKE
- EPITHELIAL TUMOR
  - ULTRASTRUCTURE, HUMAN, 2621

VIRUS, RNA TUMOR
- CARCINOMAS
  - ETIOLOGICAL AGENT, HUMAN, 2632
- DNA POLYMERASE
  - TEMPLATE PRIMER, CHICKEN, 2639
- LEUKEMIA
  - ETIOLOGY, HUMAN, 2612
  - HUMAN, 2632
  - MOLECULAR BIOLOGY, REVIEW, 2412
- LYMPHOMA
  - HUMAN, 2632
- MAMMARY CARCINOMA
  - A AND B PARTICLES, MOUSE, 2623
- REPLICATION
  - ENHANCED BY CYCLIC AMP, OVARY CELLS, HAMSTER, 2616
- REVERSE TRANSCRIPTASE
  - LEUKEMIA, MOLECULAR BIOLOGY, REVIEW, 2412
- SARCOMA
  - HUMAN, 2632
- SILICOTUNGSTATE
  - IN VITRO EFFECTS, 2678*
- TUMORIGENESIS
  - REVIEW, 2434*

VIRUS, ROUS SARCOMA
- CONTROL OF SUSCEPTIBILITY
  - GENETIC FACTORS, FOWL, 2681*
- DEGENERATIVE RESPONSE
  - MUSCLE DIFFERENTIATION, CELL CULTURE, CHICKEN, 2644
- IMMUNITY
  - AVIAN LEUKOSIS VIRUSES, CHICKEN, 2753
- PROLIFERATION CONTROL
  - FIBROBLASTS, CHICKEN, 2640
- RAT-KANGAROO CELL LINES
  - KARYOLOGICAL ANALYSIS, 2680*
- RNA-DEPENDENT RNA POLYMERASE, 2683*
- TRANSFECTION
  - CHICKEN, 2614
- TRANSFORMATION
  - INHIBITION, CHICK EMBRYO FIBROBLASTS, 2618

VIRUS, SHOPE
- PAPILLOMA
  - NUCLEIC ACID PRECURSOR UPTAKE, RABBIT, 2654
- VX-2 CARCINOMA
  - NUCLEIC ACID PRECURSOR UPTAKE, RABBIT, 2654

VIRUS, SIMIAN HERPES
- CHARACTERIZATION
  - SQUIRREL, SPIDER MONKEY, 2653

VIRUS, SIMIAN SARCOMA
- SYNCYTIAL GIANT CELL
  - FOCUS ASSAY, CANINE AND RAT CELL LINES, 2629

VIRUS, SIMIAN-TYPE PARTICLE
- MILK
  - ELECTRON MICROSCOPIC DETECTION, HUMAN, 2689*

VIRUS, SV40
- DNA
  - HOST CELL-VIRUS INTEGRATION, 2624
- REVERTANT CELL LINES
  - MOUSE, 2659
- RNA, VIRUS-SPECIFIC
  - CHROMOSOME REPLICATION, MITOSIS, MOUSE KIDNEY CELLS, 2645
- TEMPERATURE-RESISTANT
  - SUPERINFECTING TEMPERATURE-SENSITIVE DNA, HUMAN LUNG, 2647
- TRANSFORMATION
  - ETHIDIUM BROMIDE RESISTANCE, HAMSTER CELLS, 2687*
- TRANSFORMED CELL LINES
  - TUMORIGENICITY, IN VITRO TEST, MONKEY, 2746
- TRANSFORMED CELLS
  - PHOSPHOLIPID MEMBRANE COMPONENT, RABBIT, HAMSTER, 2731
  - SURFACE ANTIGEN, HAMSTER, 2733
- TRANSFORMED FIBROBLASTS
  - CELLULAR ANTIGEN, SERUM, HAMSTER, 2768*

VIRUS, VESICULAR STOMATITIS
- INFECTION
  - URIDINE KINASE LEVEL, MOUSE EMBRYO CELLS, 2677*
- PRODUCTION
  - STIMULATED LYMPHOCYTES, LYMPHOBLASTOID LINES, 2843*

VIRUS, WESTERN EQUINE ENCEPHALOMYELITIS

INFECTION
URIDINE KINASE LEVEL, MOUSE EMBRYO CELLS, 2677*

WALKER CARCINOMA
ANGIOGENESIS
NON-HISTONE PROTEIN, RAT, 2973

WALKER 256 CARCINOSARCOMA
TRANSPLANTATION
AGE, RAT, 2821*

WALKER 256 SARCOMA
FOLATE DEFICIENCY
METHYL METABOLISM, RAT, 2970

XENOGENEIC TUMOR
IMMUNE RESPONSE
MOUSE, 2811*

XERODERMA PIGMENTOSUM
4-HYDROXYAMINOQUINOLINE-1-OXIDE
SENSITIVITY, HUMAN CELLS, 2481
3-METHYL-4-NITROPYRIDINE-1-OXIDE
SENSITIVITY, HUMAN CELLS, 2481
2-METHYL-4-NITROQUINOLINE-1-OXIDE
SENSITIVITY, HUMAN CELLS, 2481
4-NITROQUINOLINE-1-OXIDE
SENSITIVITY, HUMAN CELLS, 2481

YOSHIDA ASCITES SARCOMA
ALPHA-FETOPROTEIN
RAT, 2749

ZIMBAL'S GLAND
PRETUMOR CHANGES
RAT, 2905*

☆U.S. GOVERNMENT PRINTING OFFICE: 1974-544-914/5

LIBRARY U. OF I. URBANA [illegible]

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF H.E.W.
HEW 394

U.S.MAIL

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list

DHEW Publications No. (NIH) 72-301

JUNE 1973
Abstract Nos. 3001-3600

Vol. 11
No. 6

THE LIBRARY OF THE
MAR 11 1974
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

DD

[illegible]
LIBRARY

CARCINOGENESIS ABSTRACTS

National Cancer Institute

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service — National Institutes of Health

LIBRARY U. OF I. URBANA-[illegible]

Volume XI, No. 6
June

Abstract and Citation Nos. 3001 - 3600

CARCINOGENESIS ABSTRACTS

A monthly publication of the

National Cancer Institute

THE LIBRARY OF THE
MAR 11 1974 DD
UNIVERSITY OF ILLINOIS
AT URBANA-CHAMPAIGN

Editor
Robert Love, M.D.
Jefferson Medical College, Philadelphia

Associate Editor
George P. Studzinski, M.D.
Jefferson Medical College, Philadelphia

NCI Staff Consultants
Elizabeth Weisburger, Ph.D.
Sidney Siegel, Ph.D.

Literature Selected, Abstracted, and Indexed
by
The Franklin Institute Research Laboratories
Science Information Services
Biomedical Section

Bruce H. Kleinstein, Ph.D., Technical Editor

Contract Number N01 CP 33309

Public Health Service, USDHEW

LIBRARY U. OF I. URBANA

PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

Carcinogenesis Abstracts
Room C-325
Landow Building
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Use of Funds for Printing this publication
approved by the Director of the Bureau of
the Budget on July 25, 1967.

LIBRARY U. OF I. URBANA-CHAMPAIGN

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	RNA	ribonucleic acid
i.p.	intraperitoneal	s.c.	subcutaneous
IU	international unit(s)	sec	second(s)
i.v.	intravenous	U	unit(s)
kg	kilogram(s)	UV	ultraviolet
LD_{50}	median lethal dose(s)	WBC	white blood cells (leukocytes), white blood count
m	meter(s)		
M	molar	wk	week(s)
mEq	milliequivalent(s)	wt	weight(s)
mM	millimolar	yr	year(s)
μM	micromolar		
mC,μC	milli-,microcurie(s)		

LIBRARY U. OF I. URBANA-CHAMPAIGN

CONTENTS

	Cross Reference Abbreviations	Abstracts, Citations	Page
REVIEW	(Rev)	3001-3034	453
CHEMICAL CARCINOGENESIS	(Chem)	3035-3108	458
PHYSICAL CARCINOGENESIS	(Phys)	3109-3116	471
VIRAL CARCINOGENESIS	(Viral)	3117-3226	473
IMMUNOLOGY	(Immun)	3227-3316	490
PATHOGENESIS	(Path)	3317-3327	506
EPIDEMIOLOGY AND BIOMETRY	(Epid-Biom)	3328-3336	508
MISCELLANEOUS	(Misc)	3337-3600	510
AUTHOR INDEX			
SUBJECT INDEX			

LIBRARY U. OF I. URBANA [illegible]

3001 ETIOLOGICAL ASPECTS OF CANCER OF THE GASTROINTESTINAL TRACT.

(E.) Lowenfels, A. B. (New York Med. Coll., New York City). *Surg Gynecol Obstet* 137(2):291-299, 1973.

The likely causes of cancer of the gastrointestinal tract are reviewed. Certain carcinogenic agents have been found which are surprisingly site-specific. For example, polycyclic hydrocarbons, nitrosyl compounds and irradiation of the stomach lead to stomach cancer, and methylazoxymethanol induces cancer of the colon. Alcohol consumption and tobacco smoking are the chief factors related to cancer of the esophagus. Stomach cancer increases in frequency with distance from the equator and occurs in high rates in certain countries bordering the ocean. Furthermore, higher living standards and accompanying dietary factors were associated with lower mortality rates from cancer of the stomach. A negative association between stomach cancer and cancer of the colon has been found. Cancer of the large intestine is the most common gastrointestinal cancer and the survival rate is low, the 5 yr survival rate being less than 40%. High rates correspond to high levels of economic development. Migrant studies imply that environmental factors are more important than host factors. Dietary differences are the most likely explanation for variation in occurrence. It is likely that the agent responsible for cancer of the large intestine arises in the colon, and its activity is enhanced by prolonged contact of fecal material and the colon due to refined diet. Cancer of the small intestine is uniformly rare throughout the world, occurring with 2.5% the frequency of cancer of the stomach or cancer of the colon. Evidence indicates that an intestinal immune system offers protection and that an intestinal microsomal system might afford additional security against injested foreign substances. Familial occurrence and association of certain genetic diseases with gastrointestinal carcinomas implicate genetic factors in carcinogenesis, although evidence indicates that environmental causes outweigh hereditary causes. (121 references)

3002 SSTs, OZONE, AND SKIN CANCER.

(E.) Johnson, F. S. (U. Texas, Dallas). *Astronaut Aeronaut* 11(7):16-21, 1973.

A reduction of ozone in the atmosphere would increase the incidence of skin cancer. According to available evidence, SST fleet operation would substantially reduce ozone, mainly due to the introduction of nitric oxide from the engine exhaust into the atmosphere. Reduction in the atmospheric ozone would increase the ultraviolet radiation at the Earth's surface in the spectral band responsible for skin cancer (290-320 nm) in a way that is entirely predictable. There is a controversy between two views: that the erythemal (sunburn) sensitivity curve is the correct one for evaluation of production of skin cancer; and that skin cancer is caused by genetic damage in the DNA molecules produced by ultraviolet absorption. The erythemal curve is in common use today. When the erythemal sensitivity curve is used to evaluate the dependence of exposure to ultraviolet radiation on ozone, the percentage change in exposure is about 2.5 times the percentage change in ozone; i.e., a 1% decrease in ozone causes a 2.5% increase in exposure. For the DNA absorption curve, the factor is near unity. Skin type, degree of tanning and amount of time spent out-of-doors are obvious factors that need to be taken into account in a more accurate treatment of the subject. There may be a threshold intensity below which exposure is of no consequence, which would have the effect of amplifying the change in skin cancer to be associated with a given ozone decrease. (17 references)

3003 LEUKOCYTE KINETICS IN NORMAL AND LEUKEMIC SUBJECTS.

(E.) Walker, R. I. (U. North Carolina Sch. Med., Chapel Hill). *North Carolina Med J* 34(9):693-697, 1973.

Leukocyte kinetics may be studied by one of two radioisotope tracer techniques: 1) building a precursor bearing such a tracer into the DNA of cells when they are formed and 2) affixing such tracers to existing cells by means of reactive chemicals. Using such techniques, it has been found that in chronic granulocytic leukemia (CGL) there is an accumulation of grossly excessive numbers of neutrophils of all morphologic stages of development. The DNA labeling curves in patients with CGL show a steady rise in activity from time 0. Part of this is related to cells in blood that are actively synthesizing DNA, but a substantial contribution may be from cells released early into blood from extravascular sites such as marrow and spleen. Cell labeling also shows a great expansion of the intravascular pool of neutrophils. There is speculation that the immaturity of many of the CGL cells is the important determinant of circulating time. There is also evidence in CGL that neutrophils exchange extensively between blood, spleen, and marrow. Thus, recirculation of labeled cells may contribute to the prolonged circulating time. Although the nature of the kinetic alterations in CGL is not thoroughly worked out, it is obvious that overproduction at every stage must occur to produce the abnormalities found in this disease. In chronic lymphocytic leukemia DNA labeling curves resemble those of lymphocyte labeling in normal subjects. There are no data about granulocyte production in patients with acute leukemia but the presumption is that the profound granulocytopenia seen regularly in these patients represents a failure of production. Perhaps the single most important bit of knowledge gained from the study of cell kinetics in leukemia is that there is no population of cells multiplying more rapidly than do normal cells. (22 references)

3004 INFECTION WITH HERPES-SIMPLEX VIRUSES 1 AND 2 (FIRST OF THREE PARTS).

(E.) Nahmias, A. J. (Emory U., Sch. Med., Atlanta, Ga.). *N Engl J Med* 289(13):667-674, 1973.

Recent advances in basic research on herpes-simplex virus (HSV) are reviewed. HSV-1 and HSV-2 are composed of a DNA-protein core surrounded by protein layers collectively known as the capsid. The capsid is enclosed within a proteinaceous tegument and a membranous envelope. The envelope contains lipids, polyamines, and at least 12 glycoproteins. The complete enveloped virus particle is 150-200 nm in diameter and is most likely the epidemiologically important infectious unit. The DNA of HSV-2, unlike that of HSV-1, probably contains repetetive sequences. Both HSV-1 and 2 contain an antigen, a component of the nucleocapsid, in common with the herpes-viruses associated with Burkitt lymphoma, Marek's disease, and the Lucké adenocarcinoma. There is, however, no apparent DNA homology between HSV-1 and 2 and these viruses. Although differences in DNA base composition exist between HSV-1 and HSV-2, there is at least 47% homology. Infection by HSV can be either productive, with associated cell lysis and death, or nonproductive. Both types of infection are probably involved in human disease. HSV can be transmitted from cell to cell without actual release of demonstrable virus into the extracellular space. Fusion of infected cells with uninfected cells is not an absolute requirement for this to occur. Actual viral nucleic acid replication and transcription occurs within the cell nucleus. Productive infection results in early cessation of host RNA, DNA, and protein synthesis with aberrant rRNA precursor maturation and nucleolar disaggregation. (73 references)

3005 B.A.C.R. 14TH ANNUAL GENERAL MEETING. PART III. THE 5TH WALTER HUBERT LECTURE. THE MAMMARY TUMOUR VIRUS (MTV) - A REVIEW. *(E.)* Muhlbock, O. (Netherlands Cancer Inst., Amsterdam). *Br J Cancer* 28(1):96-101, 1973.

Demonstration of transmission of mammary carcinoma in certain strains of mice via a factor in the milk of mother mice raised the question of the occurrence of a milk factor in humans. Evidence against this hypothesis were the observations that highest rates of breast cancer are found in human populations of social groups where breast feeding is least practised and that mammary tumor incidence is the same among the paternal as among the maternal grandmothers. Possibly relevant is the discovery of a new Netherlands strain of mice, called GR, in which the tumorigenic factor may be transmitted not only by mother's milk but also via the ovum or the father's sperm. The milk factor has now been shown to be an RNA-virus of unique morphology, called a B-particle. However, based on foster-nursing experiments, mammary tumor viruses (MTV) from different strains of mice differ from one another in biological properties if not in morphology. Another variant of the mouse mammary tumor virus, the nodule inducing virus, is usually present in low concentration, is transmitted with the genome at conception and has a very low carcinogenic potential. MTV is demonstrable only in a minority of inbred strains of mice. When given to strains of mice which do not naturally harbor the virus, the mammary tumor incidence is usually elevated, but the MTV tends to get lost in the offspring, due to genetic constitution. The finding that BALB/c strain of mice is free of MTV in the first half of its lifespan but shows MTV in its mammary glands later on, and that MTV can be induced in some mouse strains considered MTV free by treatment with x-rays or urethane, suggests that every mouse may carry genetic information for the production of MTV. Five different MTV-associated antigens have been demonstrated. S_1, the principal antigen of the virion, is group-specific. Specific antigens are also found in mammary tumors, but they are very weak, inconstant and difficult to find. Neonatally infected mice are at least partially tolerant to the antigens of the virus and to the virus-associated antigens in infected cells. Attempts to immunize MTV infected mice against mammary tumors often result in acceleration of tumor growth rather than suppression. Evidence for the occurrence of B-particles in human milk is inconclusive when tested by electron microscopic examination. A so-called simultaneous detection test, applied to milk, yielded wide ranges of positive samples. This test involves detection in milk of 70S single-stranded RNA and of reverse transcriptase, both components of MTV. The positive results could be explained, however, by any of 4 viruses (MTV, type C particles, foamy virus, and Mason-Pfizer virus), all of which may be found in the human population. Other evidence for possible occurrence of B-particles in human milk has been provided by demonstration of poly (A) stretches in 70S from human milk particles, comparable to those found in mouse MTV. Indications of common sequences of nucleotides in RNA viruses of humans and mice was provided by hybridization experiments between mouse MTV/DNA and RNA from human breast tumor tissue. It is concluded, however, that all the experiments have given no definite proof that a complete mammary tumor virus is present in human beings. (20 references)

3006 CARCINOGENS AND CARCINOGENESIS IN THE COLON. *(E.)* Cole, J. W. (Yale U. Sch. Med., New Haven, Conn.). *Hosp Practice* 8(9):123-130, 1973.

Epidemiological studies indicate that environmental factors are involved in the genesis of cancer of the colon. For example, adenomatous polyps are rare among the Bantu of South Africa but common among Americans, and Japanese have a low incidence of cancer of the colon whereas Nisei have the same incidence as other Americans. It was formerly thought that the neoplastic process began in the depths of the crypts - the regenerative zone - with hyperplastic clusters that coalesced and finally protruded above the surface epithelium. It now appears that the opposite occurs: neoplasia begins on the surface in cells that, instead of sloughing off, resume cell division. This was shown by studies in which labeled precursors of DNA and RNA were administered to patients with colonic tumors. Uptake of the precursors was seen in surface cells of tumors and areas of hyperplasia but only in cells lining the depths of the crypts in normal colonic mucosa. Evidence was also obtained which indicated that direct surface contact of carcinogen or its metabolite in the fecal stream was necessary for carcinogenesis in experimental systems. This was provided by the results of experiments in which three groups of rats, (one intact; one in which a portion of the colon was completely isolated by end-to-end colocolostomy; one in which the entire bowel except a few cm of rectum was isolated by subtotal colectomy with ileoproctostomy) were given the carcinogen 3,2'-dimethyl-4-aminobiphenyl. Only the first two groups developed tumors in the intestine, colon or rectum, the second group showed no tumors in the defunctionalized portion of colon, and the third group showed no tumors in the intestinal tract. Evidence that the hypothetical carcinogen must stay in contact with the susceptible cell for some time in order to trigger proliferation was provided by Burkitt who suggested that the low incidence of tumors of the colon and rectum found in the South African Bantu was related to their high fiber-content diet which produced bulky stools having an average bowel transit time of 30 hr compared to an average transit time of 81 hr for Western countries. Burkitt also suggested that the refined carbohydrate diet of Western countries resulted in an alteration of bacterial flora and degradation of bile salts that could result in the production of carcinogens in the bowel. The suggested role of bacteria was supported by work of Lacqueur who found that p.o. administration to rats of an extract of the nut of the cycad palm induced tumors of the large bowel. I.p. injection, or p.o. administration to germ-free rats, was ineffective. Following i.p. or i.v. administration to normal rats, or feeding to germ-free rats, 97% of the active substance was recovered unchanged in the urine or feces; following p.o. administration to rats, only 22% was excreted unchanged and most rats developed tumors. This and additional evidence indicated bacteria acted on the administered extract and that the product constituted the active agent. It was concluded that colonic neoplasia begins on the surface of the bowel, when normally senescent cells of the epithelium resume mitosis in response to some exogenous stimulus (e.g., something ingested or its metabolic products). It was also noted that the morphology of the tumor is probably determined by the number of cells originally involved. (8 references)

3007 ETIOLOGY OF BREAST CANCER II. EPIDEMIOLOGIC ASPECTS.

(E.) Vakil, D. V. (Sch. Hygiene, U. Toronto, Ontario, Canada) and R. W. Morgan. *Can Med Assoc J* 109(3):201-206, 1973.

The epidemiology of breast cancer is reviewed with particular emphasis on its etiology. The incidence of breast cancer is low in Japan and other Asian and African countries, as well as among American Indians who share a partially common genetic backround with the Japanese. Death rates from cancer of the breast and ovary are higher in Japanese Americans than in native Japanese. Further, breast cancer is much rarer in African women than in American blacks. It is more common in urban centers, Jewish women, women with breast disease and the U.S. native born. Mortality rates from breast cancer are higher in upper socio-economic groups than in lower ones, and increased height and weight correspond with increased breast cancer risk. High correlation of colonic cancer to breast cancer and national differences in incidence lend support to dietic epidemiology. The age-incidence curve continues to rise with age throughout life in high-risk countries, but levels off in middle age and then declines in the older age-groups in Japan. Entering menopause seems to decrease the lethal nature of breast cancer. The underlying etiological factor for breast cancer may be excessive estrogenic activity. Prior to 40-45 yr of age ovarian estrogenic activity is of etiological importance, but after menopause estrogenic activity is of adrenal origin. Higher risks are observed among unmarried and nulliparous women and among women with early menarche, although women with earlier menopause have a lower risk. Hormones may cause breast cancer or serve as prerequisites for action of another carcinogenic agent. Cancer of the male breast has been observed in patients administered therapeutic estrogen and in males exhibiting the Klinefelter syndrome. It has been hypothesized that estrogen fractions produced between puberty and the 25th yr of age are crucial determinants of a woman's lifetime breast cancer risk. Once cancer is present a different set of hormonal factors may determine the course of the disease. Mouse mammary tumor-like virus (type B) was found in human milk, and cells from the cancerous breast contained RNA molecules identical to all or part of the RNA genome of milk virus. If it is clearly established that type B particles are responsible for reverse transcriptase activity found in human milk, one could use the enzyme assay as an indicator of the presence of B particles. (97 references)

3008 SOME THOUGHTS ON A POSSIBLE RELATIONSHIP BETWEEN KNOWN GENE DOSAGE EFFECTS AND NEOPLASTIC TRANSFORMATION.

(E.) Gaudin, D. (Lab. Molecular Biol., U. Alabama, Birmingham). *J Theor Biol* 41:191-200, 1973.

A model for neoplastic transformation proposes that an unbalanced gene load resulting from viral infection, oncogenic chemicals, or ionizing radiation can include excess regulatory genes which produce an increased concentration of repressors and thereby cause a decreased production of some regulated proteins. If these proteins are involved in the feedback loop for mitotic inhibition or in the maintainance of cell membrane properties needed to maintain the intracellular concentration of mitotic inhibitor, then their loss or decreased production as a result of the unbalanced gene load can lead to the loss of control over cell growth. The model presented here may explain not only the nature of the malignant transformation but also how different degrees of malignancy can result from that transformation. If the highly malignant cell produced much less chalone or other protein important to the function of the feedback loop, then the degree of malignancy might be explained in terms of the amount of excess repressor produced and the affinity for its active binding site. (35 references)

3009 IMMUNOLOGIC ASPECTS OF LEUKEMIA.

(E.) Johnson, A. M. (U. North Carolina Sch. Med., Chapel Hill). *NC Med J* 34(9):698-701, 1973.

Since leukemia involves the very cells of immunity, the association of altered immunologic competency and leukemia is obvious. The two most prevalent theories of immunologic etiology for cancer are immunologic surveillance and immunologic enhancement. Both of these theories presuppose that leukemic cells are antigenic. If specific "tumor" antigens exist, they would also suggest that immunotherapy might be possible. However, no derepression or chemical-carcinogenic antigens have been demonstrated as yet in either lymphomas or leukemias. Virus-induced antigens are also a source of research interest, especially their possible identification in man. Cellular differentiation antigens may be present on leukemic cells, although they have not been systematically searched for as yet in human disease The potential immunologic subclassification of acute leukemia is of interest since cell-origin differences might explain differences in clinical presentation and response to therapy. Immunologic classification may also enhance the understanding of the relationships between immunologic deficits and leukemia. (17 references)

3010 ULTRAVIOLET RADIATION AS A CAUSE OF SKIN TUMORS. *(E.)* Emmett, E. A. (Dept. Environmental Hlth., U. Cincinnati, Ohio). *CRC Crit Rev Toxicol* 2(2):211-255, 1973.

Three broad areas related to skin tumors caused by UV radiation are reviewed: 1) solar radiation and human skin tumors, 2) experimental UV carcinogenesis, and 3) environmental considerations. Sun exposure has been linked to squamous cell carcinoma, basal cell epithelioma, keratoacanthoma, and malignant melanoma. The Irish and Scots have a propensity towards melanoma. This ethnic predisposition may be a general feature of peoples from the northern fringes of Europe and may reflect differences in the protective effect of melanin pigmentation in various peoples. This argument is strengthened when the high incidence of skin cancer in the albino members of heavily pigmented races is considered. Studies of the physical factors affecting UV carcinogenesis have dealt with such phenomena as the action spectrum for carcinogenesis by UV, the interaction of different wavebands in producing carcinogenic effects, and the effect of heat on UV carcinogenesis. Experiments have also been carried out to quantify the process of UV carcinogenesis, to determine the acute and chronic effects of UV radiation on the skin, and to determine the acute and chronic effects of UV radiation on the skin, and to determine the influence of exogenous chemicals, especially promotors, carcinogens, and phototoxic chemicals, on UV carcinogenesis. The effect of atmospheric pollution on UV carcinogenesis is also discussed. (313 references)

3011 CARCINOGENICITY OF DDT. *(Dutch)* Peereboom, J. W. C. (No affiliation). *Chem Weekbl* 69(14):5, 7, 1973. (14 references)

3012 HORMONE PRODUCING TUMORS OF THE LUNG. *(E.)* Lawrence, A. M. (Dept. Med., U. Chicago, Illinois). *Ann Clin Lab Sci* 3(4):259-264, 1973. (16 references)

3013 VIRAL PATHOLOGY: PAST AND PRESENT CONCEPTS. *(Fr.)* Waldvogel, F. A. (Cantonal Hosp., Geneva, Switzerland). *Schweiz Med Wochenschr* 103 (35):1209-1219, 1973. (72 references)

3014 ONCOGENIC VIRUSES AND CONCEPTS OF MODERN BIOLOGY. *(Fr.)* Montagnier, L. (Pasteur Inst., Paris, France). *Bull Inst Pasteur (Paris)* 71(3):281-298, 1973. (75 references)

3015 REVERSE TRANSCRIPTASES AND DNA POLYMERASES PROGRAMMED BY RNA. *(Fr.)* Garapin, A. C. (Pasteur Inst., Paris, France). *Bull Inst Pasteur (Paris)* 71(3):185-232, 1973. (98 references)

3016 IMMUNOLOGICAL FINDINGS IN LEUKEMIA. *(Fr.)* Levy, J. P. (No affiliation). *Bull Mem Soc Med Paris* 176(6):33-40, 1973. (No references)

3017 LEUKEMIA AND VIRUSES. *(Fr.)* Boiron, M. (No affiliation). *Bull Mem Soc Med Paris* 176(6):11-23, 1973. (7 references)

3018 CARCINOGENIC VIRUSES IN MAN? *(Fr.)* Jasmin, C. (Paul Brousse Hosp., Villejuif, France). *Councours Med* 95(19):3445-3447,3454, 1973. (13 references)

3019 THOROTRAST TUMORS IN JAPAN. REPORT OF TWO CASES WITH AUTOPSY FINDINGS AND A BRIEF REVIEW OF LITERATURE. *(E.)* Fujikura, T. (Gunma U. Sch. Med., Maesbashi, Japan), and S. Kawai. *Acta Path Jap* 23(1):139-154, 1973. (9 references)

3020 OVERVIEW OF GESTATIONAL TROPHOBLASTIC NEOPLASIA. *(E.)* Quigley, M. M. (Natl. Naval Med. Ctr., Bethesda, Md.). *NY State J Med* 73(14):1869-1874, 1973. (27 references)

3021 SOME ASPECTS OF CHEMICAL MUTAGENESIS. *(E.)* Sobels, F. H. (U. Leiden, Netherlands). *Agents Actions* 3(2):68-72, 1973. (No references)

3022 MAREK'S DISEASE AND ITS IMPLICATION FOR THE PREVENTION OF LYMPHOMA AND LEUKEMIA. *(E.)* Pomeroy, B. S. (Coll. Veterinary Med., U. Minnesota, St. Paul) and P. Paul. *Medicine (Baltimore)* 52(4): 363-365, 1973. (20 references)

3023 IMMUNOLOGY AND IMMUNOTHERAPY OF BRAIN TUMORS. CURRENT STATUS. *(Fr.)* Trouillas, P. (Neurol. Hosp., Lyon, France). *Rev Neurol (Paris)* 128(1): 23-38, 1973. (27 references)

3024 INFLUENCE ON ENZYME INDUCTION AS AN EARLY EFFECT OF CARCINOGENIC SUBSTANCES. *(Ger.)* Fischer, W. (Inst. Pathol. Med. Acad. Erfurt, E. Germany). *Arch Geschwulstforsch* 41(1):52-59, 1973. (49 references)

3025 SUNLIGHT AND THE AETIOLOGY OF MALIGNANT MELANOMA: A SYNTHESIS. *(E.)* Lee, J. A. H. (No affiliation) and J. M. Merrill. *Surv Opthamol* 18(1):96-97, 1973. (20 references)

3026 MODERN ASPECTS OF CHEMICAL CARCINOGENESIS. *(Ger.)* Wunderlich, V. (Central Inst. Cancer Res., Berlin, E. Germany). *Arch Geschwulstforsch* 41(1):60-64, 1973. (No references)

3027 POSSIBILITIES FOR ESTABLISHING MAXIMALLY PERMISSIBLE DOSES AND MAXIMALLY PERMISSIBLE CONCENTRATIONS FOR CARCINOGENIC AGENTS. *(Ger.)* Shabad, L. M. (Acad. Med. Sci. USSR, Moscow). *Arch Geschwulstforsch* 41(1):217-220, 1973. (14 references)

3028 ELEVATION MECHANISM OF AGING AS AN IMPORTANT RISK FACTOR FOR TUMOR DEVELOPMENT. *(Rus.)* Dil'man, V. M. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Vopr Onkol* 19(5):101-111, 1973. (15 references)

3029 PARAPROTEINS AS THE PRODUCT OF CLONAL IMMUNE PROLIFERATION - A CONTRIBUTION TO THE IMMUNOLOGY AND ONCOLOGY OF MONOCLONAL PROTEINS. *(Ger.)* Wetter, O. (Essen Clinic, Fed. Repub. Germany). *Klin Wochenschr* 51(18):889-899, 1973. (39 references)

3030 EPIDEMIOLOGY OF LEUKEMIA-LYMPHOMA IN MID-NEBRASKA. PART III. *(E.)* Caldwell, G. G. (Public Hlth. Service, Kansas City, Kansas), R. C. Rosenlof, H. M. Lemon, R. E. Bailey and T. D. Y. Chin. *Nebr Med J* 58(9):338-340, 1973. (41 references)

3031 IMMUNOLOGY AND CANCER. *(Ger.)* Brunner, K. T. (Swiss Inst. Exp. Cancer Res., Lausanne). *Schweiz Med Wochenschr* 103(36):1249-1252, 1973. (14 references)

3032 CHEMICAL CARCINOGENESIS AND TUMOURS OF THE CENTRAL NERVOUS SYSTEM. *(Ger.)* Schreiber, D. (Inst. Path., Martin-Luther U., Halle-Wittenberg, German Democ. Repub.), W. Jänisch and H. Wessel. *Arch Geschwulstforsch* 41(3):254-262, 1973. (80 references)

3033 VERTEBRATE AND INVERTEBRATE TUMOURS VIEWED FROM THE ANGLE OF COMPARATIVE PATHOLOGY. *(Ger.)* Krieg, K. (Acad. Sci., Berlin, German Democ. Repub.). *Arch Geschwulstforsch* 41(3):263-270, 1973. (39 references)

3034 THE INFLUENCE OF AGE ON NEGATIVE CORRELATION BETWEEN CANCER, CIRRHOSIS OF THE LIVER AND ATHEROSCLEROSIS OF THE AORTA, THE CORONARY AND THE CEREBRAL ARTERIES. *(Ger.)* Platt, D. (Justus Leibig-U. Med. Clinic, Giessen, Fed. Repub. Germany), F. E. Kie and h. P. Luboeinski. *Klin Wochenschr* 51(4):176-182, 1973. (26 references)

3035 *IN VITRO* BINDING OF LABELED 20-METHYLCHOLANTHRENE TO A SPECIFIC CYTOSOL-PROTEIN.

(E.) Slaga, T. J. (Pacific Northwest Res. Fdn., Seattle, Wash.), J. D. Scribner and J. M. Rice. *Chem Biol Interact* 7(1):51-62, 1973.

The *in vitro* binding kinetics and specificity of interaction of the ^{3}H-labeled carcinogen, 20-methylcholanthrene (MC), with mouse epidermal cytosol in the presence or absence of a purified mouse microsomal metabolizing system were studied. Following incubation the samples were dialyzed, subjected to gel filtration and ultrafiltration, and analyzed by basic polyacrylamide gel electrophoresis. A major part of the binding appeared in a single peak on the gel which had the same mobility as bovine and mouse serum albumin. The *in vitro* binding of MC to the cytosol protein could be inhibited by other hydrocarbons or promoters to degrees which correlated with their carcinogenicity or promoting ability. Essentially all of the MC which bound to cytosol in the absence of a microsomal metabolizing system was non-covalently bound. However, binding of MC to cytosol in the presence of microsomes plus NADPH was partly covalent as previously reported by others. When bovine or mouse serum albumin was incubated with ^{3}H-MC in the presence of competing non-radioactive carcinogens or promoters, little or no inhibition of binding was observed. It was also found in a preliminary experiment that radioactive 12-*O*-tetradecanoylphorbol-13-acetate (TPA), a promoter, was also bound primarily to the cytosol receptor protein when applied to mice or incubated with epidermal cytosol. It is concluded that the specific binding of MC and TPA to the mouse epidermal cytosol receptor protein may represent a critical interaction for the promotion stage of chemical carcinogenesis in mouse skin.

3036 STUDY OF LONG-TERM PERCUTANEOUS TOXICITY AND CARCINOGENICITY OF HAIR DYES (OXIDIZINE DYES) IN RATS.

(E.) Kinkel, H. J. (Biosci. Dept., Battelle Inst., Frankfurt/Main, W. Germany) and S. Holzmann. *Food Cosmet Toxicol* 11(4):641-648, 1973.

The long-term percutaneous toxicity and carcinogenicity of *p*-toluenediamine (3 or 4%), resorcinol (0.75%), and *m*-diaminoanisol (0.75%), which are important constituents of hair dyes, were studied in Sprague-Dawley rats. *p*-Toluenediamine alone (4%) in an appropriate vehicle or a mixture of all three compounds was applied to the shaved dorsal skin of the rats twice weekly for 2 yr, beginning at age 3 months. One group of control animals remained untreated while a second was treated with the vehicle alone. After the application period of 2 yr, surviving animals were observed for another 6 months. There were no indications that the substances examined had any adverse effects. Behavior, feed intake, body-weight changes, blood counts, activity of serum glutamic-pyruvic transaminase, and results of the sulfobromophtalein test for liver function were comparable in the control and experimental groups. No pathological formation of Heinz bodies or methemoglobin was associated with application of the dye components. Similarly, experimental and control animals showed no difference in respect of their mean life span or the type and incidence of tumors. No tumors or other skin reactions occurred at the site of application of the substances and histopathologic studies of the liver, kidneys, and lungs provided no evidence of degenerative change or functional disturbance. Acute studies involving s.c. or i.p. injection of the substances confirmed the lack of effect on erythropoiesis, and methemoglobin formation.

3037 THE ULTRASTRUCTURE OF A TRANSPLANTABLE RAT HEPATOMA INDUCED BY 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE.

(E.) Albrecht, C. F. (Natl. Chem. Res. Lab, Council Sci. Industrial Res., Pretoria, S. Africa), N. v.d. W. Liebenberg and J. J. Theron. *S Afr J Med Sci* 37(384):91-94, 1972.

To obtain a well-established, generally useful, azo dye-induced hepatoma of albino Wistar rats, similar to the established fluorenyl-induced Morris hepatomas of Buffalo rats, male Wistar rats were fed for 5 months with laboratory chow which contained 0.6375 g 3'-methyl-4-dimethylaminoazobenzene and 24 ml olive oil per kg chow, then with stock diet for 6 wk. At this stage about 80% of the rats presented palpable hepatomas and were sacrificed. Of 15 primary hepatomas subsequently transplanted into recipient rats, one (DAB-1) developed into a fast-growing tumor which has now been serially transplanted 26 times with an average of 22 days between transplantations. The tumorous mass contains tumor cells, necrotic tissue and a hematoma filled with disintegrating erythrocytes and neutrophils, and the tumor cells show ultrastructural dedifferentiation. Some of the tumor cells contained enlarged, endoplasmic reticulum-enclosed cisternae, called ER-lakes, which have been seen previously in fast growing human hepatoma cells.

3038 ENZYMES OF CARBOHYDRATE METABOLISM IN RAT HEPATOMA INDUCED BY 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE.

(E.) Balinsky, D. (S. African Inst. Med. Res., Johannesburg), E. Cayanis, C. F. Albrecht and D. I. Bersohn. *S Afr J Med Sci* 37(384):95-99, 1972.

Previous work with Morris hepatomas of the rat showed that key enzymes of gluconeogenesis decrease in hepatoma while key glycolytic enzymes increase. The present work showed comparable results for a new hepatoma, induced by 3'-methyl-4-dimethylaminoazobenzene. Thus, fructose-1,6-diphosphatase, α-glycerophosphate dehydrogenase and phosphoglucomutase activities were reduced in the hepatomas to 4%, 2% and 22% of host liver activity, respectively, while hexokinase and pyruvate kinase in the hepatomas were raised to 570% and 180% of host liver activities, respectively. The ratio of activity of aldolase with fructose-1,6-diphosphate to that with fructose-1-phosphate was 19 in hepatoma tissue compared to 1 in host liver. The activities of all enzymes except hexokinase were lower in host livers than in livers from control normal animals, suggesting a drain by the tumor of precursors for enzyme synthesis.

3039 EFFECT OF INCREASING DOSES OF ESTROGEN AND PROGESTERONE ON MAMMARY CARCINOGENESIS IN THE RAT.

(E.) McCormick, G. M. (Dept. Physiol. Biophys., U. Tennessee Med Units, Memphis) and R. C. Moon. *Eur J Cancer* 9(7):483-486, 1973.

Since pregnancy had been shown to stimulate both appearance and growth of mammary tumors induced in rats by 7,12-dimethylbenz(α)anthracene (DMBA), and since it had been generally accepted that both estrogen and progesterone are produced in increasing quantities during pregnancy, the present study was carried out to examine the effect of administration of these hormones on mammary tumor appearance in DMBA-fed, virgin rats. Female Sprague-Dawley rats were fed a single dose of 20 mg DMBA at 50 days of age. Fifteen days later, groups of rats received injections in incremental or constant doses of estradiol benzoate or progesterone, alone or in combination, for the next 21 days. The administration of incremental doses of progesterone plus a constant daily dose of estradiol benzoate, or the administration of incremental doses of progesterone alone showed a slight but insignificant stimulation of tumor appearance. Incremental doses of estradiol benzoate, either alone or in conjunction with progesterone, depressed tumor appearance, indicating that the stimulatory effect of pregnancy on tumor appearance most likely does not result from increasing estrogen secretion. It was noted that the use of larger doses of progesterone might have demonstrated a stimulating effect.

3040 ACTIVATION BY CHEMICAL CARCINOGENS OF γ-GLUTAMYL TRANSPEPTIDASE IN RAT AND MOUSE LIVER.

(E.) Fiala, S. (Veterans Administration Ctr., Martinsburg, W. Va.) and E. S. Fiala. *J Natl Cancer Inst* 51(1):151-158, 1973.

Feeding a diet containing carcinogenic 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB 0.06%), 2-acetylaminofluorene (2-AAP, 0.04%), Thioacetamide (0.32%), DL-ethionine (0.25%) gradually increased γ-glutamyl transpeptidase (glutathionase) in liver of 2-3 month old male Sprague-Dawley rats. Administration of dimethylnitrosamine in drinking water (40 ppm) did not significantly alter the level of glutathionase in rat liver during 30-40 days, but after 3 months the gluthathionase activity rose to a level several hundred percent higher than that of controls and remained high for several months. A single dose of strong hepatocarcinogen such as 1-2 ml dimethylnitrosamine 0.6% in saline i.p., 17 mg 2-AAF, or 40 mg 3'-Me-DAB led to higher levels of glutathionase within 24-48 hr. Diet containing carcinogenic *o*-aminoazotoluene (*o*-AAT, 0.1%) also activated glutathionase gradually in male albino Swiss mouse liver. Continuous feeding of diet containing the noncarcinogenic 2-methyl-4-dimethylaminoazobenzene caused only a temporary increase of glutathionase. Hypophysectomy inhibited the activation of glutathionase in liver of rats fed 3'-Me-DAB or 2-AAF. For 70 and 110 days, resp., the increased activity of glutathionase depended on the continued administration of 3'-Me-DAB or dimethylnitrosamine. After these periods, high glutathionase activity in rat liver became a permanent feature independent of further carcinogen. The results suggest that the activation of glutathionase in rat liver is related, almost from the beginning, to the carcinogenic process induced by chemical compounds.

3041 A SENSITIVE YEAST ASSAY SYSTEM FOR CARCINOGENIC NITROQUINOLINE OXIDES.

(E.) Koske, R. E. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada) and H. F. Stich. *Mutat Res* 19(2):265-268, 1973.

Mutants of the yeast *Saccharomyces cerevisiae* lacking in DNA repair abilities were used to detect the effect on viability of very low concentrations of 4-nitroquinoline 1-oxide (4NQO) and some of its derivatives. Four haploid strains of yeast were used. The mutant strains were sensitive to UV or X-rays or both. The double mutant (UV^{s}_{1} X^{s}_{1}) was the most sensitive to killing by 4NQO, having a surviving fraction of 3.6% after 2 hr in a 1×10^{-8} M solution. This mutant is 200 - 500 times more sensitive to 4NQO than is the wild type. When the UV^{s}_{1} X^{s}_{1} strain was exposed for 2 hr to a series of oncogenic and nononcogenic derivatives of 4NQO, the response of the yeast closely paralleled the rated oncogenicity of the compounds to mammalian systems and unscheduled DNA repair in cultured hamster cells exposed to the same chemicals.

3042 STUDIES ON THE FORMATION OF HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS BY THE BINDING OF DERIVATIVES OF 7-METHYLBENZ[a]ANTHRACENE TO DNA IN AQUEOUS SOLUTION AND IN MOUSE EMBRYO CELLS IN CULTURE.

(E.) Baird, W. M. (Inst. Cancer Res., London, England), A. Dipple, P. L. Grover, P. Sims and P. Brookes. *Cancer Res* 33(10):2386-2392, 1973.

DNA was isolated from mouse embryo cell cultures that had been treated with 7-methylbenz[a]anthracene-^{3}H and subsequently degraded with enzymes to deoxyribonucleosides that were chromatographed on a Sephadex LH-20 column. The resultant hydrocarbon-deoxyribonucleoside products were not present in similar enzyme digests of DNA that had been reacted in aqueous solution with either 7-bromomethylbenz[a]anthracene or 7-methylbenz[a]anthracene 5,6-oxide (the K-region epoxide). Thus, neither of these reactive derivatives is an adequate model for the 7-methylbenz[a]anthracene metabolite(s) involved in the binding of this carcinogen to DNA in cellular systems. Similarly, these hydrocarbon-deoxyribonucleoside products were not present in enzyme digests of DNA from cells treated in culture with 7-hydroxymethylbenz[a]anthracene, 5-hydroxy-7-methylbenz[a]anthracene, *cis*-5,6-dihydro- 5,6-dihydroxy-7-methylbenz[a]anthracene, or 7-methylbenz[a]anthracene 5,6-oxide. These data do not support mechanisms of binding of 7-methylbenz[a]anthracene to DNA in cells that require metabolic activation of the methyl group or the formation of a K-region epoxide.

3043 TRANSPLACENTAL EFFECT OF CARCINOLIPIN IN MICE. *(E.)* Shabad, L. M. (USSR Acad. Med. Sci., Moscow), T. S. Koleanichenko and L. A. Savluchinskaya. *Neoplasma* 20(3):347-348, 1973.

The effect of carcinolipin administration to pregnant A mice was investigated in their offsprings. The action of this substance in pregnant and 2-3 month-old female mice treated in the postnatal period was also studied. Carcinolipin was injected i.p. in the last third of pregnancy in olive oil; the total dose was 5-18 mg. Only lung tumors which appeared to be tubular-papillary adenomas have been found in experimental animals. In offspring, single lung adenomas were found in 2 of 6 mice, dead before 1 yr, and in 7 of 13 animals killed at 14 to 18 months. The incidence of lung tumors in the experimental group was 47.3% and in controls it was 10.7%. Single lung tumors appeared in only 5 of 27 animals treated by carcinolipin postnatally and during pregnancy (18.5%). The data illustrate the extreme sensitivity of embryonic organisms to blastomogenic agents.

3044 ISOLATION OF THE HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS FROM THE DNA OF MOUSE EMBRYO CELLS TREATED IN CULTURE WITH 7-METHYLBENZ[a]-ANTHRACENE-^{3}H. *(E.)* Baird, W. M. (Inst. Cancer Res., London, England) and P. Brookes. *Cancer Res* 33(10):2378-2385, 1973.

DNA from mouse embryo cells that had been treated in culture with 7-methylbenz[a]anthracene-^{3}H was degraded with enzymes to deoxyribonucleosides, and deoxyribonucleoside-hydrocarbon products were separated from the normal deoxyribonucleosides by chromatography on Sephadex LH-20 columns eluted with water:methanol gradients. The 7-methylbenz[a]anthracene-^{3}H-deoxyribonucleoside products were eluted as two peaks, the second of which appears to consist of at least two products. Only one product peak was released by treatment of the DNA with dilute acid. The material in both peaks that was released by enzyme digestion exhibited the thin-layer chromatographic behavior anticipated for hydrocarbon-deoxyribonucleoside products. Similar column elution profiles were obtained with enzyme digests of DNA isolated from mouse skin mouse embryo cells in primary culture, and human embryo lung cells in primary culture following treatment with 7-methylbenz[a]anthracene-^{3}H. In addition to the tritium present in the hydrocarbon-deoxyribonucleoside products, a substantial portion of the tritium associated with the DNA from the above sources was found in the normal deoxyribonucleosides. The chromatographic system described separates the normal deoxyribonucleosides from the hydrocarbon-deoxyribonucleoside products and allows the actual amount of hydrocarbon bound to DNA to be determined.

3045 ASBESTOS BODIES AND PLEURAL PLAQUES IN HUMAN LUNGS AT NECROPSY. *(E.)* Hägerstrand, I. (Genl. Hosp., Malmö, Sweden) and B. Seifert. *Acta Pathol Microbiol Scand* [A] 81(4):457-460, 1973.

Ninety-seven, largely consecutive subjects (59 men and 38 women) in Malmö, a coast town of 250,000 inhabitants in south Sweden, were examined post mortem for pleural plaques and asbestos bodies of the lungs. Two sections and one smear from the lower lobe of each lung were examined. Asbestos bodies were found in 47 subjects (32 men and 15 women) and pleural plaques in 29 subjects (26 men and 3 women). Sixteen out of the 32 men with asbestos bodies had also pleural plaques. In 7 subjects, all men, more than 20 asbestos bodies were found; four of these had pleural plaques. Carcinogenic urban air pollution of asbestos is discussed in light of these results along with results reported by Pooley's study of mesotheliomas in Malmö and Meurman's description of pleural plaques in Finland.

3046 STUDIES OF NITROSOAMINE ACCUMULATION IN WHEAT AFTER FERTILIZATION WITH NITRATE AND SECONDARY AMINES. *(Ger.)* Sander, J. (Hyg. Inst., U. Tubingen, Germany), B. Aeikens, F. Schweinsberg and G. Eisenbrand. *Z Krebsforsch* 80(1):11-15, 1973.

Winter wheat (Hano variety), planted in November, was fertilized in the spring with two applications of potassium ammonium nitrate (total of 110 kg/hectare) and peat moss mixed with dimethylamine (75 g/m^2) as the citrate or methylbenzylamine (30 and 60 g/m^2). The grain was analyzed for nitrosamines by gas chromatography and mass spectrometry or by acid-catalyzed denitrosation and determination of the 1-dimethylaminonaphthalene-5-sulfonyl derivatives formed with UV spectrophotometry. No traces of dimethyl-, methylbenzyl-, methylphenyl-, or any other nitrosamine were found (sensitivity of the method 0.3-2 parts per billion). Addition of 50% wheat to the diet of 24 female SIV$_{50}$ rats failed to produce tumors in any of the animals in a two-yr observation period. No evidence was found that the wheat contained any toxic or carcinogenic substances.

3047 THE CARCINOGENIC ACTIVITY OF ETHOXYDIAZENIUM SALTS (O-ETHYLDIALKYLNITROSIMMONIUM SALTS) IN RATS. *(Ger.)* Druckrey, H. (Preventive Med. Res. Group, Freiburg, Germany), J. Stekar and S. Hünig. *Z Krebsforsch* 80(1):17-26, 1973.

The tumorigenicity of O-ethyl fluoroborates of dimethylnitrosamine (I), methyl tert.-butylnitrosamine (II), nitrosopiperidine (III), and nitrosomorpholine (IV) was tested in acute and chronic experiments on 100-day-old rats of various BD strains. Single s.c. injections of I, II, III, and IV in pH 7 phosphate buffer were administered. Injection of 130 mg/kg s.c. of I induced a sarcoma at the site of injection in 1 of 2 rats, while no tumor developed in any of 5 rats given 100 mg/kg s.c. The highly toxic compound II (6 mg/kg s.c.) was nontumorigenic in 7 rats. A single injection of III (170 mg/kg s.c.) produced a local sarcoma in 1 rat; smaller doses were not tumorigenic. Of 10 rats given 320-430 mg/kg s.c. of IV, 5 died after 337-471 days with large sarcomas at the site of injection; one rat developed a sarcoma after receiving only 100 mg/kg. During the two-yr observation period, a single injection of sodium tetrafluoroborate (500 mg/kg s.c.) failed to induce tumors in any of the 15 animals treated. In chronic experiments

II and IV were administered in aqueous solutions. None of the 15 rats given 2 mg/kg/week of II for 27 weeks (total dose of 205 mg/kg) developed tumors. Of the 15 rats treated with IV (40 mg/kg/week s.c.), 7 died with large sarcomas at the site of injection after 250 to 354 days. After a total dose of 2 g/kg had been administered, treatment was discontinued. All of the remaining 8 rats developed similar tumors. Multiple metastases were found in the lungs in 4 cases and in the kidneys in 1. Histological examination showed that 8 tumors were spindle-cell sarcomas, 6 were polymorphocellular sarcomas, and 1 was a fibrosarcoma. No liver cancer or nasal tumors, which are characteristic for nitrosomorpholine, were found. From these experiments it is concluded that the tumorigenic activity of IV is due to its direct alkylating action.

3048 REACTION OF *N*-METHYL-*N*-NITROSOUREA (MNUA) WITH ^{32}P-LABELLED DNA: EVIDENCE FOR FORMATION OF PHOSPHOTRIESTERS. *(E.)* Lawley, P. D. (Inst. Cancer Res., Pollards Woods Res. Station, Chalfont St. Giles, England). *Chem Biol Interact* 7(2):127-130, 1973.

A group of new phosphorous-containing products has been isolated by Dowex 50 chromatography of enzymatic digests of *N*-methyl-*N*-nitrosourea (MNUA)-treated ^{14}C-thymine- or ^{32}P-labeled *E. Coli* DNA. About 12 products could be resolved. Seven contained thymidine and another was apparently chromatographically identical to a methylation product of thymidyl-(3'-5')thymidine. The amounts of the different products were roughly equal, as would be expected from random attack of MNUA on the known phosphodiester-linked base sequences of *E. coli* DNA. The alkylation products, which were alkali stable, contained two nucleoside moieties/P atom. The pattern of elution of the products from the Dowex columns indicated that they did not contain phosphodiester linkages.

3049 EFFECT OF THE ALKYLATING AGENT TRIETHYLENEIMINOBENZOQUINONE ON THE SYNTHESIS OF DNA AND ON THE INCORPORATION OF [^{3}H]LYSINE INTO NUCLEAR PROTEINS OF EHRLICH ASCITES TUMOR CELLS. *(E.)* Wolf, H. (Biochem. Inst., U. Freiburg, Germany), G. Raydt, B. Puschendorf and H. Grunicke. *FEBS Lett* 35(2):336-340, 1973.

Concentrations of the alkylating agent triethyleneiminobenzoquinone (Trienimon) which do not affect the biosynthesis of DNA in Ehrlich ascites cells are demonstrated to inhibit strongly the incorporation of [^{3}H]lysine into histones whereas [^{3}H]lysine incorporation into the chromosomal nonhistone proteins and the total cellular proteins is hardly affected. The time course of the effect of a single dose of the alkylating agent (6 x 10^{-7} moles/kg) on the incorporation rate of lysine into the acid soluble nuclear proteins reaches a minimum 4 hr after administration of the drug and can still be observed 24 hr after exposure. The amount of DNA which is synthesized during the 24-hr interval following administration of 6 x 10^{-7} moles/kg Trenimon is about the same in treated as in untreated populations of Ehrlich ascites cells. The data demonstrate that the alkylating agent is capable of dissociating the synthesis of DNA and the increase in the amount of the histones in the cell nucleus, two processes which are normally tightly coordinated. The data do not permit a decision on whether the suppression of the histone labeling is caused by an inhibition of histone synthesis or by a block in the transport of the preformed histones from the cytoplasm into the nucleus.

3050 CARCINOGEN-INDUCED SKIN TUMORIGENESIS IN MICE: ENHANCEMENT AND INHIBITION BY ULTRAVIOLET LIGHT. *(E.)* Stenbäck, F. (U. Nebraska Med. Ctr., Omaha) and P. Shubik. *Z Krebsforsch* 79(4):234-240, 1973.

The effect of a single application of UV light as well as repeated applications of UV light on skin tumorigenesis in mice induced by 7,12-dimethylbenz(a)anthracene (DMBA) was studied. A single subcarcinogenic application of UV light before repeated carcinogen treatment almost doubled the number of benign and malignant tumors, pointing to an enhancing effect of UV light. The morphological characteristics of tumors (squamous papillomas, keratoacanthomas and squamous cell carcinomas) induced by repeated chemical carcinogen applications were not significantly altered by a preceding application of UV light; only a slightly stronger stromal fibrosis and vascular hyalinization ensued. Repeated UV light applications alone produced tumors of both epidermal and dermal origin: acanthopapillomas, fibropapillomas, squamous cell carcinomas and fibrosarcomas. When both UV light and DMBA were given repeatedly (UV light was given after DMBA) the tumor response was morphologically similar to that produced by UV light alone; however, the number of tumors was slightly less and the latent period was longer than when DMBA was given alone, which points to an inhibitory effect of UV light. The apparent tumor inhibiting effect of repeated UV irradiation on repeated carcinogens application might be due to oxidation of DMBA by the light exposure. Another explanation may be that UV light induces a dermatosis which prevents neoplastic progression.

3051 MAMMARY CARCINOGENESIS IN THE RAT BY TOPICAL APPLICATION OF FLUORENYLHYDROXAMIC ACIDS. *(E.)* Malejka-Giganti, D. (VA Hosp., Minneapolis, Minnesota), H. R. Gutmann and R. E. Rydell. *Cancer Res* 33(10):2489-2497, 1973.

One topical application of 0.02 mmole of N-hydroxy-2-fluorenylacetamide to the left thoracic mammary glands of adult female Sprague-Dawley rats gave a 70% (5 of 7) tumor incidence at the site. The tumors appeared 3 to 6 months after the application and were mammary adenocarcinomas. In contrast, the tumor incidence after application of N-2-fluorenylacetamide was 30% (1 of 6), the latent period was 8 to 11 months, and the tumors were not confined to the site of application. N-Hydroxy-3-fluorenylacetamide, a strong mammary carcinogen by the i.p. route, and N-3-fluorenylacetamide, a marginally active mammary carcinogen, when applied locally showed a similar pattern of carcinogenicity as the 2-isomers. These data suggest that the fluorenylhydroxamic acids, rather than the arylamides, induce mammary tumors. Experiments comparing the microsomal N-hydroxylation of N-2- and N-3-fluorenylacetamide by mammary gland and liver showed that mammary gland microsomes, in contrast to those of liver, did not N-hydroxylate the arylamides. Experiments concerned with the sulfation of N-hydroxy-2- and -3-fluorenylacetamide by mammary gland gave no evidence that mammary gland formed the N-sulfates of N-hydroxy-2-fluorenylacetamide or of N-hydroxy-3-fluorenylacetamide. The data suggest that N-hydroxylation may be implicated in mammary carcinogenesis. However, the two-step activation mechanism believed to initiate hepatocarcinogenesis by N-2-fluorenylacetamide does not appear to be operative in mammary gland.

3052 ALKYLATION OF TRANSFER RNA BY N-METHYL-N-NITROSOUREA AND N-ETHYL-N-NITROSOUREA. *(E.)* Pegg, A. E. (Middlesex Hosp. Med. Sch., London, England). *Chem Biol Interact* 6(6):393-406, 1973.

The alkylation of several purified yeast and *E. coli* tRNA's by incubation at 37 C in the presence of the ^{14}C-labeled carcinogens, N-methyl-N-nitrosourea (MNU) and N-ethyl-N-nitrosourea (ENU), was studied *in vitro*. The alkylated t-RNA's were hydrolyzed, and alkylated nucleotides were identified by chromatography. The initial rate of reaction was greatest at pH values between 6 and 8, and the degree of alkylation was found to be directly proportional to the MNU or ENU concentration. MNU produced a 13-fold greater extent of alkylation than did ENU. Five specific t-RNA's studied were all alkylated to about the same extent. The major reaction product of MNU was 7-methyl-guanosine which accounted for 82% of the radioactivity present in the isolated t-RNA. 7-Ethylguanosine accounted for 79% of the radioactivity recovered from ENU-treated materials. Alkylation was not restricted to a single site within the t-RNA molecule and the results were consistent with a random distribution of MNU and ENU within all the guanine residues.

3053 CANCER OF RESPIRATORY ORGANS AMONG WORKERS AT A NICKEL REFINERY IN NORWAY. *(E.)* Pedersen, E. (Cancer Registry of Norway, Oslo), A. Hogetveit and A. Andersen. *Int J Cancer* 12(1):32-41, 1973.

A follow-up study was conducted to determine the incidence of respiratory tract cancers among 1,916 men employed at a nickel refining plant in Norway between 1953-71. During this period there were 48 cases of lung cancer, 14 of cancer of the nasal sinuses and 5 of laryngeal cancer. The overall incidence of respiratory tract cancers for all employees was 5.6 times that of the general population. The highest relative incidences were among men involved in electrolysis and those involved in roasting and smelting (7.4 and 7.0 times expected, resp.). The relative risk for cancer of the nasal cavities among these two groups was 40 times the expected incidence. The highest risk for respiratory tract cancer was among men who had begun work prior to 1930, with the observed to expected ratios becoming progressively smaller for those who has started in successive periods thereafter.

3054 THE EFFECT OF ZINC ON 9,10-DIMETHYL-1,2-BENZANTHRACENE (DMBA) INDUCED SALIVARY GLAND TUMOURS IN THE ALBINO RAT - A PRELIMINARY STUDY. *(E.)* Ciapparelli, L. (Dental Res. Unit, U. Witwatersrand, Johannesburg, S. Africa), D. H. Retief and L. P. Fatti. *S Afr J Med Sci* 37(384): 85-90, 1972.

Reports of the effects of dietary zinc on carcinogenesis have been controversial. In the present study, 20 Wistar albino rats 4-5 months old were divided into four groups, one given deionized drinking water, the other three given drinking water containing 50, 100 and 250 ppm zinc, respectively. Five-mg pellets of 9,10-dimethyl-1,2-benzanthracene (DMBA) were implanted in the right submandibular glands after the start of the experiment and, three months later, the glands were dissected, weighed and examined histologically. The results showed that tumor growth was significantly retarded when the zinc concentration in the water reached 250 ppm. Further, the pathology of the tumors changed with increased zinc in the drinking water. While in the group of rats on deionized water well differentiated squamous carcinomas developed, in the groups on increasing concentrations of zinc the amount of squamous epithelium decreased progressively and an inflammatory response became more marked. It was suggested that the increase amount of lymphatic tissue within the tumors of rats on high zinc levels could indicate an immune response to the developing carcinoma. The inhibitory effect and the change in pathogenesis produced by the zinc in the drinking water could not be explained.

3055 GLUCAGON AND PROSTAGLANDIN E$_1$ STIMULATION OF CYCLIC ADENOSINE 3',5'-MONOPHOSPHATE LEVELS AND ADENYLATE CYCLASE ACTIVITY IN BENIGN HYPERPLASTIC NODULES AND MALIGNANT HEPATOMAS OF ETHIONINE-TREATED RATS. *(E.)* Chayoth, R. (U. Pittsburgh Sch. Med., Pittsburgh, Pa.), S. M. Epstein and J. B. Field. *Cancer Res* 33(8):1970-1974, 1973.

Basal adenylate cyclase activity and cyclic adenosine 3',5'-monophosphate (cyclic AMP) concentrations and their responses to glucagon and prostaglandin E$_1$ (PGE$_1$) have been measured in liver from control rats and in uninvolved liver, benign hyperplastic nodules, and malignant hepatomas from rats treated with ethionine. Cyclic AMP concentrations were significantly higher and more responsive to glucagon and PGE$_1$ in uninvolved liver from ethionine-treated rats than that from controls. Adenylate cyclase activity was significantly greater in uninvolved liver compared to controls but the increase induced by glucagon and PGE$_1$ was similar in the two tissues. Although glucagon stimulation of adenylate cyclase activity exceeded that due to PGE$_1$, they were equivalent measuring cyclic AMP. Phosphodiesterase activity was similar in control liver and uninvolved liver from ethionine-treated rats, suggesting that the higher cyclic AMP

ncentrations in the uninvolved liver of ethionine-
eated rats compared to the controls probably re-
ects increased synthesis rather than decreased de-
adation of the nucleotide. Cyclic AMP concentra-
ons were similar in uninvolved liver and benign hy-
rplastic nodules but were significantly greater in
lignant hepatomas. PGE_1 caused equivalent augmenta-
on of cyclic AMP levels in uninvolved liver and be-
gn hyperplastic nodules but greater response in
lignant hepatomas. Glucagon increased cyclic AMP
vels in a similar fashion in all three tissues.
enylate cyclase activity was significantly higher
malignant hepatomas compared to benign hyperplastic
dules and uninvolved liver. PGE_1 stimulation of
zyme activity was similar in all three tissues but
s significantly less than that obtained with glu-
gon. Although glucagon increased cyclic AMP in
lignant hepatomas more than in the other two types
tissue, its stimulation of adenylate cyclase in
lignant hepatomas tended to be less than the other
o tissues. Phosphodiesterase activity was signi-
cantly decreased in malignant hepatomas.

56 THE EARLY STIMULATION OF PHOSPHOLIPID METABOLISM BY 12-*O*-TETRADECANOYL-PHORBOL-13-ACE- TE AND ITS SPECIFICITY FOR TUMOR PROMOTION. *(E.)*

hrschneider, L. R. (U. Wisconsin Med. Ctr., Madison)
d R. K. Boutwell. *Cancer Res* 33(8):1945-1952, 1973.

e relationship to tumor formation of the stimulated
ospholipid metabolism produced in mouse skin (female
arles River CD-1 mice, 7 to 8 wk old) by a single
plication of the tumor-promoting phorbol ester, 12-
tetradecanoyl-phorbol-13-acetate (TPA) was examined.
e TPA-induced stimulation of ^{32}P incorporation into
use skin phospholipids was first characterized and
en compared with the altered incorporation of ^{32}P
oduced by the application of either acetic acid, β-
opiolactone or 7,12-dimethylbenz[a]anthracene at
dose levels. The results indicated that only the
nor promoter, TPA, was able to stimulate the early
nthesis of phosphatidylethanolamine and phosphatidyl-
oline. This specificity was further emphasized by
e demonstration that only TPA stimulated the incor-
ration of choline-^{14}C into phosphatidylcholine and
anolamine-^{3}H into phosphatidylethanolamine. The
ding that mouse skin papillomas (initiated with
2-dimethylbenz[a]anthracene and promoted with cro-
oil) contained more phosphatidylcholine than the
sue of origin suggested that membrane alterations
the stimulated synthesis of phosphatidylcholine are
ortant factors in tumor formation. The specificity,
sitivity, and rapid response suggest that the stimu-
ed synthesis of phosphatidylcholine would be a
table and convenient assay for the detection of
ential environmental tumor-promoting agents.

57 LACK OF A BLADDER CARCINOGENIC EFFECT OF A BANANA DIET IN RATS AND GUINEA PIGS. *(Ger.)*

wald, H. (German Cancer Res. Ctr., Heidelberg).
ebensm Unters Forsch 152(5):287-290, 1973.

contrast to Sorrentino, who found that an ex-
sive amount of bananas in the diet induced pap-
omas and premalignant changes in the bladders of guinea pigs, only benign changes were found in the bladders of 2 and kidneys of 1 of 25 male guinea pigs fed peeled bananas. No correlation was found between the latent times for these benign changes and the type of diet or the time it had been fed. No tumors developed in any of 25 male Sprague-Dawley rats fed peeled bananas, while 3 of the 25 fed unpeeled bananas developed malignant tumors in other sites after long latent periods (722 to 818 days); no premalignant changes developed in either the bladder or kidneys of rats fed either peeled or unpeeled bananas.

3058 KINETICS OF PHOTO-INDUCED COOXIDATION OF BENZO(a)PYRENE AND PHENOLS WITH VARIOUS STRUCTURES. *(Rus.)*

Karu, T. (Inst. Chem., Acad. Sci. Estonian SSR, USSR), U. Kirso and M. Gubergrits. *Izv Akad Nauk Eston SSR* 22(3):217-223, 1973.

The kinetics of UV-induced oxidation of benzo(a)-pyrene (BP) was studied in 96% ethanol and n-octane solutions in the presence and absence of various phenols (o-, m- and p-cresols, phenol, 2,6-ditert-butyl-p-cresol and 4-nitro- and 2,4-dinitrophenol). Both phenol and BP oxidation followed zero-order kinetics in both solvents. The relation between structure and reactivity of the phenols, either alone or in the presence of BP, can be described accurately with the Hammett-Taft equation. BP had no effect on the kinetics or mechanism of phenol photooxidation, but it did inhibit phenol degradation. This inhibition was most evident with the more reactive phenols. The most reactive phenol, o-cresol, caused the greatest increase in the rat of BP photooxidation; phenol and its homologs also increased BP degradation. The least active phenol, 4-nitrophenol, inhibited BP degradation as did 2,4-dinitrophenol and 5-methylresorcinol. These results obtained *in vitro* are analogous to those obtained for BP photooxidation *in vivo* on intact and UV-irradiated animal skin.

3059 A HIGH SUSCEPTIBILITY OF FANCONI'S ANEMIA TO CHROMOSOME BREAKAGE BY DNA CROSS-LINKING AGENTS. *(E.)*

Sasaki, M. S. (Tokyo Med. Dental U., Japan) and A. Tonomura. *Cancer Res* 33(8):1829-1836, 1973.

Previous reports of high sensitivity of chromosomes of Fanconi's anemia (FA) patients to an alkylating agent, tetramethanesulfonil-d-mannit and to ionizing radiation suggested possible biological mechanisms underlying the high frequency of spontaneous chromosome aberrations and the increased risk to the development of malignant neoplasms in this hereditary disorder. In the present work, peripheral blood lymphocytes of patients with FA were tested for their susceptibility to chromosome breakage by caffeine, chloramphenicol, actinomycin D, methylmethanesulfonate, nitrogen mustard, mitomycin C, decarbamoyl mitomycin C, N-methyl-N'-nitro-N-nitrosoguanidine, 4-nitroquinoline 1-oxide, 8-methoxypsoralen, and ^{60}Co γ-ray and UV irradiations. A definitely abnormal response of the chromosomes of the FA cells was found when they were treated with nitrogen mustard and mitomycin C and after irradiation with long-wavelength UV light in the presence of 8-methoxypsoralen. The specifically increased susceptibility to these compounds, which can introduce interstrand cross-links into DNA, is interpreted as an indication that the FA cells are defective in the repair mechanism to tolerate the cross-links produced in their DNA. This impairment of the capacity to tolerate the lesions produced in DNA may be implicated in the increased risk to develop malignant neoplasms.

3060 METHYLATION OF DNA BY ^{3}H-^{14}C-METHYL-LABELLED *N*-METHYL-*N*-NITROSOUREA - EVIDENCE FOR TRANSFER OF THE INTACT METHYL GROUP.

(E.) Lawley, P. D. (Royal Cancer Hosp., London, England) and S. A. Shah. *Chem Biol Interact* 7(2):115-120, 1973.

The methylation of salmon sperm DNA by ^{3}H-^{14}C-labeled *N*-methyl-*N*-nitrosourea (MNUA) was studied *in vitro* at pH 8. The following products were isolated by Dowex 50 column chromatography of the DNA hydrolysate: 1-, 3-, and 7-methyladenines, 3-methyldeoxycytidine, 3-, 7-, and O^{6}-methylguanines, and 3- and O^{4}-methylthymidines. Comparison of $C^{3}H_{3}$ and $^{14}CH_{3}$ labeling showed that the $^{3}H/^{14}C$ ratio of the products was the same and equal to that of the original reagents. This finding is consistent with the concept that the methyl group is transferred intact from MNUA to DNA residues and does not support the existence of a diazomethane intermediate.

3061 TUMOR DEVELOPMENT IN MICE BEARING A PLASTIC CYLINDER AND INOCULATED WITH HUMAN NEOPLASTIC CELLS.

(E.) Pasqualini, C. D. (Fac. Med., U. Buenos Aires, Argentina), L. Sen, F. Saal, L. Schwartz and L. Z. Tkaczevski. *J Natl Cancer Inst* 51(1):275-282, 1973.

Human neoplastic cells from two stable lymphoma cell lines, inoculated near a plastic cylinder implanted s.c. in 97 adult BALB/c mice, led to tumor development in 47% of the animals; allogeneic AKR lymphoma cells gave 73% tumor incidence. Thus both values were significantly different from the 23% in control mice bearing only the plastic cylinder. Tumor latency averaged 11 months. Transplants were easily obtained in syngeneic BALB/c mice but not in AKR; the karyotypes were murine. Histologically all tumors were similar and could be classified as anaplastic sarcomas. Intracisternal A-type virus particles were observed in tumor cells. It is postulated that both human and AKR lymphoma cells contain an agent capable of potentiating the tumorigenic effect of a plastic implant.

3062 POTENTIAL PRECURSORS OF *N*-NITROSOPYRROLIDINE IN BACON AND OTHER FRIED FOODS.

(E.) Bills, D. D. (Dept. Food Sci. Technology, Oregon State U., Corvallis), K. I. Hildrum, R. A. Scanlan and L. M. Libbey. *J Agric Food Chem* 21(5):876-877, 1973.

The possibility of producing *N*-nitrosopyrrolidine from several compounds in a system designed to simulate the frying of fatty foods containing small amounts of water was investigated. With the exception of tests on *N*-nitrosoproline, equimolar amounts of the compound to be tested and sodium nitrite were added to the system prior to heating. With a heat-up time of 10 min plus 10 min at 170°, *N*-nitrosopyrrolidine was produced from *N*-nitrosoproline, pyrrolidine, spermidine, proline, and putrescine in yields of 2.6, 1.0, 1.0, 0.4, and 0.04% theoretical, resp. *N*-Nitrosopyrrolidine was not produced from glutamine, glutamic acid, and hydroxyproline. The occurrence of proline, spermidine, and putrescine in foodstuffs was discussed.

3063 INDUCTION OF ARYL HYDROCARBON HYDROXYLASE IN MOUSE 3T3 CELLS: RELATIONSHIP TO THE STATE OF CELL PROLIFERATION AND GUANOSINE CYCLIC 3', 5'-MONOPHOSPHATE.

(E.) Ruddon, R. W. (U. Michigan Med. Sch., Ann Arbor), D. E. Lundeen and L. E. Rikans. *Mol Pharmacol* 9(5):686-691, 1973.

The induction of aryl hydrocarbon hydroxylase was studied in a line of mouse 3T3 fibroblasts (Swiss) grown in continuous cell culture. The level of hydroxylase activity after induction by benz[*a*]anthracene appeared to be several times higher in this 3T3 line than in other continuously cultured cell lines previously studied. The induction of aryl hydrocarbon hydroxylase was linear for 24 hr after the addition of 13 μM benz[*a*]anthracene to the culture medium. Induction of the enzyme by benz[*a*]anthracene was markedly greater in 3T3 cells grown in the presence than in the absence of serum and was higher in nonconfluent than in confluent cells. Moreover, the addition of 0.01 μM dibutyryl cyclic guanosine 3',5'-monophosphate (a putative signal for increased cell proliferation) to serumless medium containing 6 μM benz[*a*]anthracene markedly potentiated induction of aryl hydrocarbon hydroxylase by the hydrocarbon. These results suggest that the inducibility of the hydroxylase system by benz[*a*]anthracene is greater in proliferating than in nonproliferating cells.

3064 FORMATION OF NITROSAMINES IN A MEAT CURING MIXTURE.

(E.) Sen, N. P. (Food Res. Lab., Hlth Protection Branch, Ottawa, Canada), W. F. Miles, B. Donaldson, T. Panalaks and J. R. Iyengar. *Nature* 245(5420):104-105, 1973.

Trace amounts of certain N-nitrosamines, well known carcinogens, in foods, especially nitrite-treated meat and fish products, have caused a great deal of concern. These compounds are usually formed from the interactions of nitrite and secondary or tertiary amines. Recent studies, however, have shown that certain naturally occurring quaternary ammonium compounds can also react with nitrite to form nitrosamines. Of the known nitrosamines, dimethylnitrosamine (DMN) and nitrosopyrrolidine (NPy) are the two compounds which have been most frequently found to be present in cured meat products. NPy is formed during frying of bacon. Its presence in sausages made by certain Canadian firms is reported in the present work. Sausages prepared with and without nitrite and also with and without curing mixture were studied. Only those prepared with the curing mixture contained NPy. Subsequently it was found that the curing mixture was heavily contaminated with both NPy and another nitrosamine, nitropiperidine (NPi). Incubation of black pepper, one of the constituents of the curing mixture, with nitrite yielded mainly NPi, while paprika, another constituent, produced mainly NPy under the same conditions. Both NPy and NPi were previously shown to be carcinogenic in laboratory animals.

3065 A QUANTITATIVE SYSTEM FOR ASSAY OF MALIGNANT TRANSFORMATION BY CHEMICAL CARCINOGENS USING A CLONE DERIVED FROM BALB/3T3. Kakunaga, T. (Res. Inst. Microbial Dis., Osaka U., Japan). *Int J Cancer* 12(2):463-473, 1973.

A subclone isolated from clone A31 of BALB/3T3 mice showed a high degree of contact inhibition and an extremely low incidence of spontaneous transformation. Treatment of this subclone (A31-714) with 4-nitroquinoline-1-oxide (4NQO), 3-methylcholanthrene, benzo(a)pyrene (BP) or N-methyl-N'-nitro-N-nitrosoguanidine produced foci of multilayered growth in the background of contact-inhibited monolayer within 2-4 wk. In this system the transformation frequency can be determined quantitatively on an inoculated cell basis or a surviving cell basis. The transformation frequency increased with the concentration of carcinogens within a certain range, and was affected by the duration of treatment and the cell density at the time of treatment. Concentrations above 0.1 μg/ml 4NQO, 4.0 μg/ml MCA, or 2.0 μg/ml BP resulted in no further increase in transformation frequency. Treatment with dimethyl sulfoxide alone or with the noncarcinogen substances 4-nitroquinoline, 4-aminoquinoline-1-oxide, phenanthrene, and pyrene did not cause any transformation. The cytotoxic effects of carcinogens were not directly correlated with the transformation frequencies. All transformed cell lines derived from each focus showed characteristic indices of malignant transformation, such as a criss-cross pattern or piling up of cells, a high saturation density, and the ability to grow progressively in soft agar. When the transformed cells were injected into the skin of the back of newborn or adult mice at a dose of 10^6 cells, fibrosarcomas were produced at the site of injection after 3-6 weeks. Neither untreated A31-714 cells nor morphologically untransformed cells in cultures with foci produced tumors on injection at a dose of 10^7 cells. In general, transformed cells had a reduced cloning efficiency, and almost the same susceptibility to the cytotoxic effects of carcinogens as untransformed cells, though these properties varied in different lines.

3066 CARCINOGENICITY OF 5-NITROFURANS, 5-NITROIMIDAZOLES, 4-NITROBENZENES, AND RELATED COMPOUNDS. *(E.)* Cohen, S. M. (U. Wisconsin Med. Sch., Madison), E. Ertürk, A. M. Von Esch, A. J. Crovetti and G. T. Bryan. *J Natl Cancer Inst* 51(2):403-417, 1973.

Some 5-nitrofurans, such as those used widely as antimicrobial agents in human and veterinary medicine, have shown evidence of carcinogenicity. To investigate this question further, 18 selected compounds were administered to weanling female Sprague-Dawley rats in their diets. Test with 1-[(5-nitrofurfurylidene)amino]hydantoin, a commonly used urinary-tract antibacterial agent, gave negative results in chronic carcinogenicity tests in rats. However, carcinogenic responses for structurally related compounds were moderate for 4-methyl-1-[(5-nitrofurfurylidene)amino]-2-imidazolidinone and 1-(2-hydroxyethyl)-3-[(5-nitrofurfurylidene)amino]-2-imidazolidinone, and strong for *l*-5-morpholinomethyl-3-[(5-nitrofurfurylidene)amino]-2-oxazolidinone hydrochloride and 1-[(5-nitrofurfurylidene)amino] -2-imidazolidinone. Several other nitrofurans, nitrobenzenes, aminobenzenes, nitroimidazoles, and related compounds were also tested for carcinogenicity with varying results. 5-Nitro-2-furamidoxime, 2-hydrazino-4-phenylthiazole, D-(−)-*threo*-1-(p-nitrophenyl)-2-dichloroacetamido-1,3-propanediol, 1-(2-hydroxyethyl)-2-methyl-5-nitroimidazole, and 2-amino-5-phenyl-2-oxazolin-4-one with $Mg(OH)_2$ gave negative results; hexamethylmelamine and 1,2-dimethyl-5-nitroimidazole were weakly tumorigenic; 4,6-diamino-2-(5-nitro-2-furyl)-s-triazine and its bisacetamide, formic acid 2-[4-(5-nitro-2-furyl)-2-thiazolyl]-hydrazide, 2-(2,2-dimethylhydrazino)-4-(5-nitro-2-furyl)thiazole, 2-hydrazino-4-(p-nitrophenyl)thiazole, and 2-hydrazino-4-(p-aminophenyl)thiazole were strongly carcinogenic. The significance of the structural variations is discussed with respect to nitrofuran carcinogenesis and the possible carcinogenicity of other nitroaromatic compounds.

3067 A NEW TOXIC FLUORESCENT METABOLITE DERIVED FROM THE INTERACTION OF RAT LIVER AND AFLATOXIN B_1. *(E.)* Friedman, L. (Div. Toxicol., Food and Drug Admin., Washington, D.C.), L. Yin and M. J. Verrett. *Toxicol Appl Pharmacol* 23:385-390, 1972.

A new toxic metabolite derived from incubation of aflatoxin B_1 with non-microsomal rat liver preparations has been identified by thin layer chromatography (TLC) and fluorescent densitometry. The substance was soluble and migrated on TLC at a rate different from that of all the known aflatoxins and aflatoxin metabolites. The non-microsomal fraction, as the supernatant after obtained centrifugation of rat liver extract for 60 min at 105,000 g, contained 83% of the metabolizing activity of the extract and required the presence of NADP. The new metabolite, which was partially purified by chloroform and hexane extraction, showed the same degree of toxicity toward chick embryos as did the parent compound, aflatoxin B_1.

3068 ACUTE CHANGES IN THE SURFACE MORPHOLOGY OF HAMSTER TRACHEOBRONCHIAL EPITHELIUM FOLLOWING BENZO(a)PYRENE AND FERRIC OXIDE ADMINISTRATION. *(E.)* Port, C. D. (TIT Res. Inst., Chicago, Illinois), M. C. Henry, D. G. Kaufman, C. C. Harris and K. V. Ketels. *Cancer Res* 33(10):2498-2506, 1973.

Acute changes in the surface morphology of hamster tracheobronchial epithelium were examined by scanning electron microscopy following intratracheal administration of ferric oxide or benzo(a)pyrene absorbed to ferric oxide. Multiple instillation of ferric oxide brought about a loss of ciliated cells and broad areas of abnormal, enlarged nonciliated cells with roughened or wrinkled surfaces. These were interpreted as areas of epithelial hyperplasia. Multiple intratracheal administrations of benzo(a)pyrene and ferric oxide caused similar changes and, in addition, produced squamous metaplasia and small foci of markedly abnormal protuberant cells. These findings are correlated with a previously reported observation obtained by transmission electron microscopy. The results suggest that scanning electron microscopy is useful in studies of the histogenesis of lung cancer.

3069 K-REGION AND NON-K-REGION METABOLISM OF BENZO(*a*)PYRENE BY RAT LIVER MICROSOMES. *(E.)*

Kinoshita, N. (Natl. Cancer Inst., Bethesda, Md.), B. Shears and H. V. Gelboin. *Cancer Res* 33(8):1937-1944, 1973.

The metabolism of benzo(*a*)pyrene (BP) by liver microsomes from normal and 3-methylcholanthrene (MC)-treated rats was quantitatively analyzed by a double label method using BP-^{3}H and BP-^{14}C. Qualitatively, the metabolism of BP by both microsomal preparations was similar. The identified metabolites were 7,8-dihydro-7,8-dihydroxy-BP, 4,5-dihydro-4,5-dihydroxy-BP, 9,10-dihydro-9,10-dihydroxy-BP, 3-hydroxy-BP, 9-hydroxy-BP, and BP-1,6-quinone and BP-3,6-quinone. The quantitative analysis by the double labeling method showed the profile of BP metabolites produced by rat liver microsomes from normal and MC-induced rats. The ratio of metabolites from induced microsomes to that from normal microsomes was greatest for the 7,8-dihydro-7,8-dihydroxy and 9,10-dihydro-9,10-dihydroxy metabolites, less for the phenols and K-region metabolites, and least for the quinones. Similar ratios were obtained when microsomes from MC-treated rats were diluted except that dilution reduced the relative amount of phenol formation. These results suggest that higher enzyme activity favors the formation of the non-K-region diols and phenols relative to the K-region diol. The detection of the K-region metabolite of benzpyrene may be of great importance in view of the K-region theory of polycyclic hydrocarbon carcinogenesis.

3070 AN IMPROVED EXPRESSION OF *IN VITRO* TRANSFORMATION RATE BASED ON CYTOTOXICITY PRODUCED BY CHEMICAL CARCINOGENS. *(E.)*

Umeda, M. (Christie Hosp., Manchester, England) and P. T. Iype. *Br J Cancer* 28(1):71-74, 1973.

Most chemical carcinogens tested for ability to transform mammalian cells in culture also have cytotoxic effects *in vitro*. When transformation rate and survival curves are plotted independently against concentration of carcinogen, it is difficult to compare transformation rates at *equitoxic* levels of different carcinogens. This difficulty was overcome by plotting the transformation rate against the relative plating efficiency of carcinogen-treated cells. To determine whether the observed transformation in a given system can be explained by selection of pre-existing transformed cells or by chemical induction, the observed curves are compared with a theoretical line based on the assumed selection of pre-existing transformed cells which are completely resistant to the cytotoxic action of the carcinogen. The theoretical line may be constructed with the aid of the observed value for the transformation rate at the point of 100% plating efficiency.

3071 MUTAGENICITY OF THE CARCINOGEN 7-BROMOMETHYLBENZ[a]ANTHRACENE: A QUANTITATIVE STUDY IN REPAIR-DEFICIENT STRAINS OF *ESCHERICHIA COLI*. *(E.)*

Tarmy, E. M. (Royal Cancer Hosp., London, England), S. Venitt and P. Brookes. *Mutat Res* 10(2):153-166, 1973.

The mutagenicity of 7-bromomethylbenz[a]anthracene, a reactive arylalkylating carcinogen, was investigated in several strains of *E. coli* WP2, using reversion from tryptophan auxotrophy (*ochre trpE* locus) as a measure of induced mutation. WP2, the wild-type with respect to DNA repair, was more resistant to the cytotoxic effects of 7-bromomethylbenz[a]anthracene than WP2*uvrA*, WP2*exrA*, or WP2*uvrAexrA*, the D_{37} doses of carcinogen being 22, 5, 8, and 1 µg/ml, resp. Mutagenesis in WP2 was observed only at doses in excess of the D_Q, whereas in WP2*uvrA* mutation was linearly related to dose throughout the range studied. No mutation was detectable in WP2*exrA* or WP2*uvrAexrA* even at doses which resulted in 95% and 99.9% lethality, resp. It was concluded that an intact Exr function was an absolute requirement for the induction of mutation by 7-bromomethylbenz[a]anthracene and that excision-repair was very efficient in removing premutational lesions. The use of [^{3}H]7-bromomethylbenz[a]anthracene at high specific radioactivity enabled the quantitation of mutation as a function of the extent of reaction of the mutagen with cellular macromolecules. Extent of reaction with DNA, RNA and protein was linearly related to dose, binding to DNA being 3 times that to RNA and 20 times that to protein. There was a linear relationship between binding and mutation in WP2*uvrA* and the effective target size for Exr-mediated mutation in this system was of the order of 0.04 nucleotides. Having established that the number of 7-bromomethylbenz[a]anthracene-induced mutants increased linearly with successive cell generations and, by use of T4 *ochre*427, that about 30% of the mutants scored were true revertants, it was estimated that the Exr pathway incorporates productive errors into the bacterial genome with a frequency of the order of 2×10^{-3}.

3072 VIRUS-CHEMICAL CARCINOGENESIS: A POSSIBLE VIRAL IMMUNOLOGIC INFLUENCE ON 3-METHYLCHOLANTHRENE SARCOMA INDUCTION. *(E.)*

Whitmire, C. E. (Microbiol. Associates, Inc., Bethesda, Md.). *J Natl Cancer Inst* 51(2):473-478, 1973.

In some cases, oncogenic and nononcogenic viruses combined with chemical carcinogens have been found to increase the incidence of or accelerate neoplastic expressions. In other cases, viral and chemical carcinogens have not acted in concert but have inhibited the effects of each other. The present study investigated the effects of neonatal infection with radiation leukemia virus (RadLV) on s.c. sarcoma induced by 3-methylcholanthrene (MCA) in C57BL/6 mice. The infection was produced with extracts of lymphoma transplants containing the virus and also with partially purified preparations of the virus. The MCA was injected s.c., and trioctanoin was injected s.c. for control mice. The mice were observed for 8 months after MCA administration. Progress of the infection was followed by tests for group-specific (gs) antigen of type-C RNA viruses, carried out on mouse tissue extracts with the use of sera for gs-1 murine virus antigen. The results indicated that neonatal RadLV infection reduced the incidence of MCA-induced sarcomas from 80% to 22% in one experiment, from 63% to 29% in another. This effect was similar to that previously demonstrated with formalin-inactivated RadLV vaccine. The incidence of RadLV-induced leukemias was low and unaffected by MCA treatment. The possible delay in leukemia development may indicate viral immunologic responses common to viral-induced leukemia and chemically-induced sarcoma.

3073 EVIDENCE FOR THE BINDING OF THE CARCINOGEN 3-METHYLCHOLANTHRENE TO BOTH THE PURINE AND THE PYRIMIDINE BASES OF HAMSTER FIBROBLAST DEOXYRIBONUCLEIC ACID. *(E.)* Jones, P. A. (Unit Molecular Cellular Cardiology, U. Stellenbosch, Tiervlei, South Africa), W. Gevers and A. O. Hawtrey. *Biochem J* 135(2):375-378, 1973.

The binding of $[^3H]$3-methylcholanthrene to the DNA of hamster fibroblasts was studied by using chemical methods for DNA degradation. The investigation of the nature of carcinogen binding to DNA has been complicated by the fact that the carcinogen renders the DNA resistant to complete enzymic hydrolysis and that $HClO_4$ hydrolysis causes degradation of the carcinogen. The relatively mild chemical methods used in this study may therefore be useful in the identification of the carcinogen moiety and the type of linkage involved. DNA depurinated by mild acid hydrolysis released approximately half of the radioactivity at the same rate as the purine bases, but the resulting apurinic acid still contained radioactive carcinogen, representing that carcinogen bound to the amino function of cytosine.

3074 OXIDATIVE ACTIVATION OF BENZ[a]ANTHRACENE AND METHYLATED DERIVATIVES IN MUTAGENESIS AND CARCINOGENESIS. *(E.)* Fahmy, O. G. (Royal Cancer Hosp., London, England) and M. J. Fahmy. *Cancer Res* 33(10):2354-2361, 1973.

Comparative genetic studies in *Drosophila* with benz[a]anthracene, its 7-methyl and 7,12-dimethyl substituents, 3-methylcholanthrene and their epoxides revealed that K-region oxidation dramatically altered the mutational spectrum of the hydrocarbon. The parent compounds were virtually inactive on the euchromatic parts of the genome but were effective on certain heterochromatic sites, especially the rRNA genes that yield *bobbed* mutations. This specific mutability was substantially higher for the methylated derivatives but did not appear to be dose dependent. In contrast, the K-region epoxides were decisively mutagenic on the euchromatic genes and were less effective on the rRNA loci than the corresponding parent hydrocarbons, and their mutagenicity was dose dependent. Mutagenic selectivity for the rRNA genes was quantitatively measured in terms of an index which was defined as the percentage proportion of *bobbed* mutants relative to the total X chromosome mutations recovered from the same sample of treated gametes. Statistical studies revealed that the genetic selectivity indices for a series of structurally related benz[a]anthracenes showed a highly significant correlation to their relative carcinogenic potencies in rodent skin, both at the rank level and when expressed as Iball's indices. The genetic and oncogenic indices for the K-region epoxides were invariably lower than those for the corresponding parent hydrocarbons, which argued against the suggestion that such oxidative products could be regarded as the ultimate carcinogens.

3075 CHANGES IN RIBONUCLEIC ACID METABOLISM ASSOCIATED WITH 3-METHYLCHOLANTHRENE-INDUCED ADULT RAT LIVER GROWTH. *(E.)* Hopkinson, J. (Dept. Biol., Syracuse U., N.Y.) and T. S. Argyris. *Lab Invest* 29(3):342-349, 1973.

The relative wt, protein and RNA content of livers of adult male rats are increased by 20-30% between 48 and 72 hr after a 20-mg/kg body wt injection of 3-methylcholanthrene (3MC). An analysis of hepatic DNA and nuclear count indicates that this liver growth is caused mainly by cellular hypertrophy, with probable increases in cell ploidy. At 12 hr after 3MC injection, the rate of incorporation of ^{3}H-orotic acid acid into total free uridine nucleotides remains unchanged, while that into all species of nuclear RNA migrating slower than 4S in acrylamide-agarose gels is increased, suggesting an enhanced rate of RNA synthesis. Between 24 and 72 hr after injection, 3MC treatment generally suppresses the rate of orotic acid incorporation into free uridine nucleotides and into total cytoplasmic and microsomal RNA's. At 72 hr after 3MC administration, the specific activities of 28-, 18-, 5-, and 4S cytoplasmic and microsomal RNA's are either unchanged or depressed. When the changes in RNA precursor pool sizes are considered, there is only a modest increase in the rate of RNA synthesis in the livers of 3MC-treated rats, a rate of synthesis not sufficient to account for the RNA accumulation associated with the induced liver growth. Decreased rates of RNA degradation may also be associated with 3MC-induced adult rat liver growth.

3076 ULTRASTRUCTURAL AND BIOCHEMICAL CHANGES INDUCED IN MOUSE EPIDERMIS BY A HYPERPLASTIC AGENT, ETHYLPHENYLPROPIOLATE. *(E.)* Raick, A. N. (Dept. Path., U. Toronto, Ontario, Canada) and K. Burdzy. *Cancer Res* 33(10):2221-2230, 1973.

A single application of ethylphenylpropiolate (EPP) to mouse skin induces an ordered sequence of morphological and metabolic changes which lead to cell proliferation and epidermal hyperplasia. EPP induces a marked stimulation of the incorporation of labeled precursors into the DNA and RNA of the skin and, to a lesser extent, into protein. The mitotic index, the number of nucleated cell layers, and the thickness of the interfollicular epidermis are increased. The most outstanding ultrastructural changes observed were the dispersion of the chromatin, enlargement of the nucleoli, expansion of the cytoplasm, and a marked increase in the number of polysomes. These ultrastructural changes reach a maximum 24 hr after treatment and then revert rapidly toward normal. They are considerably less 48 hr after treatment, and 96 to 120 hr after treatment the fine structural characteristics of the epidermis are again normal. Focal areas of necrosis are observed in the epidermis treated with EPP. "Dark cells" seen in these areas morphologically resemble the "dark cells" observed in epidermal wounds induced by other means and the "dark cells" induced in intact mouse epidermis by 12-*O*-tetradecanoylphorbol-13-acetate (TPA). EPP causes marked hyperplasia in mouse skin but has little promoting power. Since wounding has some promoting action in mouse skin, it is possible that the weak promoting action of EPP is due to the focal necroses it induces. The ultrastructural alterations induced in mouse epidermis by EPP differ markedly from those induced by the powerful tumor promoter TPA. EPP fails to induce the opening of the intercellular spaces between the epidermal cells and the reversal of epidermal cell differentiation which are among the most striking effects induced by TPA. It produces only slight alterations in the rough endoplasmic reticulum, Golgi complex, and mitochondria instead of the prominent changes induced by TPA, and fine structural changes induced by EPP are shorter lasting than those induced by TPA. It remains to be determined whether the differences between the reactions induced by EPP and TPA would be found if other hyperplastic and promoting agents were compared, and, if so, what their relevance is to promotion.

3077 SYNERGISTIC EFFECTS OF DIETHYLNITROSAMINE AND DIFFERENT DUSTS ON RESPIRATORY CARCINOGENESIS IN HAMSTERS. *(E.)* Stenbäck, F. G. (U. Nebraska Med. Ctr., Omaha), A. Ferrero and P. Shubik. *Cancer Res* 33(10):2209-2214, 1973.

A possible synergistic action between the carcinogen diethylnitrosamine injected s.c. and MgO, Al_2O_3, or carbon instilled intratracheally in the respiratory tract of Syrian golden hamsters was studied. Diethylnitrosamine alone induced tumors of the nasal cavity in 50% of the animals. Tumors were also produced in the larynx and trachea but not in the lungs. Groups receiving weekly intratracheal instillations of 0.9% NaCl solution suspensions of carbon or Al_2O_3 had no tumors of the respiratory system. MgO alone induced a significantly large number of lymphomas of the histiocytic type that were not induced by diethylnitrosamine and MgO. In the groups receiving diethylnitrosamine followed by intratracheal instillations of dust-0.9% NaCl solution suspensions, the tumor incidence in the upper respiratory tract was similar to that seen with diethynitrosamine alone, but lung tumors were also observed. Intratracheal instillations of 0.9% NaCl solution after diethylnitrosamine also caused a number of tumors to occur in the lower respiratory tract. It can be concluded that carbon and Al_2O_3 are not carcinogens for the respiratory system of hamsters. The increase in incidence of lower respiratory tract tumors previously seen with diethylnitrosamine followed by Fe_2O_3 and 0.9% NaCl solution was here obtained with diethylnitrosamine followed by 0.9% NaCl solution alone.

3078 THE SYNCARCINOGENIC EFFECT OF METHYLCHOLANTHRENE AND DIMETHYLNITROSAMINE IN SWISS MICE. *(E.)* Cardesa, A. (Eppley Inst. Res. Cancer, Omaha, Neb.), P. Pour, M. Rustia, J. Althoff and U. Mohr. *Z Krebsforsch* 79(2):98-107, 1973.

The combined administration of 3-methylcholanthrene (MCA), 0.3% in olive oil, intragastrically, 1.5 mg/kg 3 times daily, and dimethylnitrosamine (DMN) 0.24% in 0.9% saline i.p. 6 mg/kg weekly to Swiss mice resulted in an increased incidence of tumors and in a decreased tumor latency, compared with animals treated with each compound alone. In the group given both MCA and DMN the rate of lung carcinomas and neoplasms of the kidney was found to be significantly enhanced, while the DMN group showed an increase in vascular tumors, and the MCA group showed an increase in malignant lymphomas. The result of combined application of MCA and DMN was interpreted as a syncarcinogenic effect, resulting in an enhanced carcinoma development in the lungs and an increased tumor incidence in the kidney, with the DMN appearing to inhibit development of malignant melanomas and the MCA appearing to inhibit development of vascular tumors.

3079 LEUKEMIA FOLLOWING HANDLING OF HYDANTOIN. *(Ger.)* Wildhack, R. (Gen. Hosp., Hamburg, Fed. Repub. Germany). *Munch Med Wochenschr* 111(28): 1275-1279, 1973.

3080 INCIDENCE OF NEPHROBLASTOMAS IN IMMUNODEPRESSED SPRAGUE-DAWLEY RATS GIVEN DIMETHYLBENZANTHRACENE. *(Fr.)* Bourgoin, J. J. (Leon Berard Ctr., Lyon, France), J. Cueff, C. Bailly and M. Dargent. *Bull Cancer (Paris)* 59(4):429-434, 1972.

3081 INDUCTION OF MAMMARY TUMORS IN HAMSTERS. *(Rus.)* Georgadze, G. E. (Inst. Exp. Clin. Surg., Min. Hlth. Georgian SSR, USSR). *Soobshch Akad Nauk Gruz SSR* 70(1):225-227, 1973.

3082 SILICONE-INDUCED PENILE SCLEROSING LIPOGRANULOMA. *(E.)* Arthaud, J. B. (U. Texas Med. Sch., Houston). *J Urol* 110(2):210, 1973.

3083 THE EFFICACY OF CYCLOPHOSPHAMIDE IN THE TREATMENT OF METHYLCHOLANTHRENE-INDUCED SARCOMAS AS AFFECTED BY THE CYRCADIAN RHYTHM OF CELL DIVISION. *(Fr.)* Focan, C. (Liège U. Pathol. Anat. Lab., Belgium), H. Barbason and E. H. Betz. *C R Acad Sci* [*D*] *(Paris)* 276(13):2105-2108, 1973.

3084 CARCINOGENIC EFFECTS OF METHYL- AND ETHYL-NITROSOUREA (MNU AND ENU) AT SINGLE INTRA-CEREBRAL AND INTRACAROTIDAL INJECTION IN NEWBORN AND YOUNG BD-RATS. *(Ger.)* Druckrey, H. (Preventative Med. Res. Group, Freiburg, Fed. Repub. Germany), S. Ivankovic and J. Gimmy. *Z Krebsforsch* 79(4):282-297, 1973.

3085 DEOXYRIBONUCLEASES AND PHOSPHATASES OF RAT LIVER CARCINOMAS INDUCED BY DIETHYLNITROSAMINE. *(Ger.)* Tempel, K. (Vet. Fac., U. Munich, Fed. Repub. Germany) and R. Hollatz. *Z Krebsforsch* 79(4): 267-271, 1973.

3086 PREVENTION OF NITROSAMIDE-INDUCED HYDROCEPHALI BY ASCORBIC ACID AFTER PRENATAL ADMINISTRATION OF ETHYLUREA AND NITRITE TO RATS. *(Ger.)* Ivankovic, S. (German Cancer Res. Ctr., Heidelberg), R. Preussmann, D. Schmähl and J. Zeller. *Z Krebsforsch* 79(3):145-147, 1973.

3087 RELATIONS BETWEEN NUCLEAR DNA SYNTHESIS AND CELL PROLIFERATION DURING INITIATION OF SKIN CANCER WITH 3,4-BENZPYRENE. CYTOPHOTOMETRIC STUDY. *(Fr.)* Bernheim, J. L. (Acad. Hosp., Ghent, Belgium) and H. Roels. *C R Soc Biol (Paris)* 167(2): 380-387, 1973.

3088 EFFECTS OF REPEATED ADMINISTRATION OF ANTILYMPHOCYTE SERUM ON TUMORS INDUCED BY INJECTING NEWBORN MICE WITH 7,12-DIMETHYLBENZ(a) ANTHRACENE. *(It.)* Baroni, C. D. (Inst. Anat. Pathol. Histol II, U. Rome, Italy), R. Scelsi, L. Peronace, S. Uccini and A. Cavallero. *Tumori* 59(1):1-10, 1973.

3089 INTERPRETATION OF CARCINOGENESIS FROM UV-SPECTRA OF 3,4-BENZPYRENE AND 1,2-BENZPYRENE. *(Ger.)* Popp, F. A. (Radiology Clinic, U. Marburg, Fed. Repub. Germany). *Z Naturforsch [B]* 28(3/4):165-168, 1973.

3090 MALIGNANT TUMOURS OF LIVER AND LUNG IN RATS FED AMINOPYRINE OR HEPTAMETHYLENEIMINE TOGETHER WITH NITRITE. *(E.)* Lijinsky, W. (Oak Ridge Natl Lab., Tenn.), H. W. Taylor, C. Snyder and P. Nettesheim. *Nature* 244(5412):176-178, 1973.

3091 THE EFFECT OF HIGH AND LOW-SALT INTAKE ON 7,12-DIMETHYLBENZ(α)ANTHRACENE-INDUCED HYPERTENSION IN RATS. *(E.)* Kellen, J. A. (Banting Inst., U. Toronto, Ontario, Canada) and A. Mirakian. *Z Krebsforsch* 80(1):5-10, 1973.

3092 CRITICAL NOTES ON INTRA-ANIMAL CULTURE OF MICROORGANISMS, A METHOD FOR DETECTING CHEMICALLY-INDUCED MUTATION. *(Ger.)* Propping, P. (Inst. Anthropol. Human Genetics, U. Heidelberg, Germany), W. Buselmaier and G. Rohrbörn. *Arzneim Forsch* 23(6):746-749, 1973.

3093 COMPARATIVE EVALUATION ON THE ONCOGENIC ACTIVITY OF A BINARY MIXTURE OF ALKYL CARBAMATES AND ITS COMPONENTS. *(Rus.)* Iagubov, A. S. (Inst. Ind. Hyg. Occupational Dis., Moscow, USSR) and T. I. Suvalova. *Gig Tr Prof Zabol* (8):19-22, 1973.

3094 EXPERIMENTAL STUDY OF THE EFFECT OF CHLOROPRENE ON EMBRYOGENESIS. *(Rus.)* Sal'nikova, L. S. (Inst. Ind. Hyg. Occupational Dis., Moscow, USSR) and V. N. Fomenko. *Gig Tr Prof Zabol* (8):23-26, 1973.

3095 STUDY OF THE MUTAGENIC AND CYTOTOXIC PROPERTIES OF IRRADIATED FOOD PRODUCTS. *(Rus.)* Bronnikova, I. A. (F. F. Erisman Res. Inst. Hyg., Moscow, USSR) and L. A. Okuneva. *Vopr Pitan* (4): 46-49, 1973.

3096 THE TOPOCHEMISTRY OF TUBULAR ENZYME PATTERNS IN EXPERIMENTAL CARCINOGENESIS. STUDIES OF ENZYME HISTOCHEMISTRY IN LEAD-INDUCED CHRONIC KIDNEY DISEASE OF THE RAT. *(Ger.)* Stiller, D. (F.-Schiller U. Inst. Pathol. Jena, E. Germany). *Exp Pathol* 8(2):137-154, 1973.

3097 EVIDENCE FOR A CYRCADIAN RHYTHM OF CELL DIVISION IN METHYLCHOLANTHRENE-INDUCED SARCOMAS. *(Fr.)* Focan, C. (Liège U. Pathol. Anat. Lab., Belgium), H. Barbason and E. H. Betz. *C R Acad Sci [D] (Paris)* 276(14):2229-2232, 1973.

3098 ELICITATION OF LENS FORMATION FROM THE 'VENTRAL IRIS' EPITHELIUM OF THE NEWT BY A CARCINOGEN, N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. *(E.)* Eguchi, G. (Inst. Biophysics, U. Kyoto, Japan) and K. Watanabe. *J Embryol Exp Morphol* 30(1):63-71, 1973.

3099 THE PROLONGED BINDING OF ETHYL CARBAMATE-(^{14}C) TO DNA IN REGENERATING AND INTACT MOUSE LIVER. *(E.)* Lawson, T. A. (Dept. Path., U. Queensland, Australia) and A. W. Pound. *Eur J Cancer* 9(7):491-496, 1973.

3100 THE DEVELOPMENT OF CELL CLASSES IN THE LIVER OF RATS AND CHINESE HAMSTERS. *(Ger.)* Wiest, L. (Inst. Cancer Res., U. Vienna, Austria). *Exp Pathol* 8(2):109-114, 1973.

3101 CARCINOGENIC ACTION OF 1-(p-CARBOXYETHYLPHENYL)-3,3-DIMETHYLTRIAZENE AFTER S.C. AND I.V. ADMINISTRATION TO RATS. *(Ger.)* Schmähl, D. (German Cancer Res. Ctr., Heidelberg) and D. Steinhoff. *Arzneim Forsch* 23(6):800-802, 1973.

3102 TOXICITY STUDIES WITH METIAPINE. *(E.)* Gibson, J. P. (Richardson-Merrell, Inc., Cincinnati, Ohio), M. W. Rohovsky, J. W. Newberne and E. J. Larson. *Toxicol Appl Pharmacol* 25(2):220-229, 1973.

3103 THE THYROID FUNCTION IN REGRESSION OF EXPERIMENTAL TUMORS UNDER CONDITIONS OF THE HYPOTHALAMUS STIMULATION. *(Rus.)* Bordjushov, Ju. N. (Res. Inst. Oncology Rostov/on Don, USSR) and L. I. Gashnikova. *Vopr Onkol* 19(6):65-68, 1973.

3104 PATHOLOGICAL REACTIONS IN RAT LUNGS FOLLOWING INTRATRACHEAL INJECTION OF NICKEL SUBSULFIDE AND 3,4-BENZPYRENE. *(E.)* Kasprzak, K. S. (Med. Acad. Poznan, Poland), L. Marchow, and J. Breborowicz. *Res Commun Chem Pathol Pharmacol* 6(1):237-245, 1973.

3105 FORMATION OF GLUTATHIONE CONJUGATES AS METABOLITES OF 7,12-DIMETHYLBENZ[a]ANTHRACENE BY RAT-LIVER HOMOGENATES. *(E.)* Booth, J. (Chester Beatty Res. Inst., London, England), G. R. Keysell and P. Sims. *Biochem Pharmacol* 22(14):1781-1791, 1973.

3106 EFFECT OF PHENOL AND 5-METHYLRESORCINOL ON THE CARCINOGENIC ACTIVITY OF BENZO(a)PYRENE. *(Rus.)* Linnik, A. (Inst. Exp. Clin. Oncol., Moscow), U. Kirso, L. Savluchinskaia, M. Gubergrits and L. Shabad. *Izv Akad Nauk Eston SSR* 22(3):224-229, 1973.

3107 INDUCTION OF ARYL HYDROCARBON HYDROXYLASE IN HUMAN PULMONARY ALVEOLAR MACROPHAGES BY CIGARETTE SMOKING. *(E.)* Cantrell, E. T. (M. D. Anderson Hosp. Tumor Inst., Houston, Texas), G. A. Warr, D. L. Busbee and R. R. Martin. *J Clin Invest* 52(8):1881-1884, 1973.

3108 USE OF CHLORELLA IN RESEARCH OF SUBSTANCES WITH CARCINOGENIC ACTIVITY. *(Ger.)* Teichmann, B. (Central Inst. Cancer Res. Acad. Sci., Berlin, E. Germany), R. Rathsack and D. Ziebarth. *Arch Geschwulstforsch* 41(1):18-22, 1973.

See also:

* (Rev): 3011, 3021, 3024, 3026, 3027, 3032
* (Phys): 3110
* (Immun): 3270, 3272, 3303, 3305
* (Epid-Biom): 3328

3109 SEQUENTIAL PATHOPHYSIOLOGIC EFFECTS OF IONIZING RADIATION ON THE BEAGLE THYROID GLAND. *(E.)* Lu, S.-T. (Sch. Med. Dentistry, U. Rochester, N.Y.), S. M. Michaelson and W. J. Quinlan. *J Natl Cancer Inst* 51(2):419-441, 1973.

Although radiation-induced thyroiditis and tumor formation have been studied extensively in animals and man, the pathogenesis and mechanisms of the reaction involved have not been elucidated. Dogs were used in the present work because of their longer lifespan and lower incidence of spontaneous thyroid neoplasia compared to rats, and because their metabolic rate is closer to that of man. Irradiation was carried out externally with X-rays, internally with ^{131}I, or with both. Thyroid function tests were based on radioiodine uptake and on protein-bound iodine. Response of the thyroid gland to ionizing radiation exposure manifested itself functionally by transient increased activity and progressive hypothyroidism and pathologically by degeneration, hyperplasia, and carcinogenesis. Increase in circulating thyroid stimulating hormone seemed secondary to the glandular damage. However, thyroid-stimulating hormone seemed to promote the progressive change, its self-perpetuation, and the sequence of events leading to permanent hyperplasia or carcinogenesis. Of 56 dogs studied, 32 survived 3 or more years after treatment and 11 of the 32 developed neoplastic thyroids. The postirradiation latency for induction of thyroid neoplasia in dog (4-10 yr), man (9-14 yr), and rat (1-2 yr) showed a direct relationship with basal metabolic rate, suggesting a fundamental connection with tissue metabolism and thyroid cell turnover rate.

3110 RADIATION-INDUCED LEUKEMIA IN RATS: SYNERGISTIC EFFECT OF URETHAN. *(E.)* Myers, D. K. (Biol. Hlth. Div., Chalk River Nuclear Lab., Ontario, Canada). *Experientia* 29(7):859-861, 1973.

In black hooded 'Collip' rats divided into groups of 23-32 animals, the leukemogenic effect of urethan (ethyl carbamate) and X-radiation together were greater than the sum of their separate effects. Leukemia developed in 64% of rats given five weekly exposures of 165 r X-rays plus five weekly injections of 0.9 g/kg urethan compared with an incidence of 8% in rats receiving urethan only and of 25% in rats treated with X-rays alone. However, X-radiation alone induced a higher incidence (58%) of mammary and skin tumors than the combined treatment (12%).

3111 RADIOSTRONTIUM INDUCED EARLY CHANGES IN THE HAEMATOPOIETIC TISSUES WITH SPECIAL REFERENCE TO THE THYMUS IN MICE. *(E.)* Järplid, B. (Div. Radiobiol., Res. Inst. Natl. Defence, Sundbyberg, Sweden). *Acta Radiol (Ther) (Stockh)* 12(2):145-154, 1973.

The effects of radiostrontium on the hematopoietic tissues have been examined 1 to 50 days after treatment. Two hundred twenty female CBA mice, aged 30 ± 3 days were divided into two groups and injected with 0.4 and 0.8 μCi ^{90}Sr/g body weight. The changes in mean total weight of the thymus were dose-dependent and revealed a diphasic process. Two days after Sr administration the volume distribution of the thymic cells was still normal although both the number of the smaller lymphocytes (100 to 140 μ^3) and the total number of lymphocytes had decreased to about half of the normal. This indicates that the reduction of the cell count applied to all sizes of cells. On day 4 it was chiefly cells less than 150 μ^3 that had been reduced in number, while the proportion of larger cells had increased, probably due to an incipient regeneration. The second depletion phase occurred after day 12 and continued through until day 30. The delayed regeneration of the thymus might be caused by strontium-induced damage to the bone marrow. It is also possible that certain parts of the thymus may be affected by radiation from strontium accumulated in surrounding bone. Splenic hyperplasia was dose-dependent and was a result of compensatory extramedullary hematopoiesis, which probably reflects the degree of bone marrow destruction. The lymphocyte depletion of the spleen follicles was probably an indirect effect of the Sr via the bone marrow or the thymus.

3112 CYTOGENETIC EFFECT OF SINGLE AND REPEATED EXPOSURE TO X-RAYS ON MOUSE BONE MARROW CELLS. *(Rus.)* Gogebashvili, N. K. (Inst. Gen. Genetics, Acad. Sci. USSR) and V. A. Tarasov. *Soobshch Akad Nauk Gruz SSR* 71(1):189-192, 1973.

White inbred male mice were exposed to single or divided doses of 25-100 rads of x-rays at a rate of 10.9 r/min, and chromosome damage was studied in their bone marrow 7 hr to 30 days after exposure. The percentage of cells with chromosome damage was highest 7 hr after exposure and decreased progressively with time; it did not differ significantly from that of controls 20 and 30 days after divided doses and 30 days after a single dose of 25 or 50 rads. The spectrum of chromosome aberrations (single and paired fragments, dicentric and ring chromosomes) was the same with a single dose and with divided doses, but the frequency of all types of abnormal chromosomes decreased when radiation was administered in divided doses. At the beginning, the effect of divided doses of x-rays on chromosomes was not related to the total dose, but the amount of damage at later periods increased with increases in total dose. At a dose of 100 rads, mice exposed to x-rays in divided doses had significantly fewer ring and dicentric chromosomes than those given single doses, but this was true only for the first cell cycle after exposure.

3113 SKELETAL LOCATION OF RADIATION-INDUCED AND NATURALLY OCCURRING OSTEOSARCOMAS IN MAN AND DOG. *(E.)* Thurman, G. B. (Anatomy Dept., U. Utah, Salt Lake City), C. W. Mays, G. N. Taylor, A. T. Keane and H. A. Sissons. *Cancer Res* 33(7):1604-1607, 1973.

Data were compiled from six major studies of naturally occurring and radiation-induced bone tumors in man and dogs, and their type and distribution were compared. The most frequent type of bone sarcoma was osteosarcoma in both humans and dogs. The distribution pattern of the radiation-induced osteosarcomas differed from the natural pattern in both animals and humans. Whereas 50% of naturally occurring human osteosarcomas were in or near the knee, only 10-30% of radium-induced tumors arose in this region. Bone-deposited radionuclides increased the frequency of pelvic osteosarcomas from 10% to 20-30% and decreased by one-half (from 44% to 22%) the incidence of osteosarcoma of the femur. Compared to man, dogs had a very high frequency of naturally occurring osteosarcomas of the radius and ulna and a low frequency of occurrence of pelvic and femoral tumors. Radiated dogs showed increased tumor frequency in vertebrae and the pelvis with decreased incidence in the radius. Compared to radiated humans, radiated dogs had an increased frequency of vertebral tumors and a lowered frequency of femoral and pelvic tumors. These results suggested that most of the naturally occurring osteosarcomas result from causes other than natural levels of the bone-seeking radionucnuclides (radium, pulonium, thorium and strontium).

3114 EFFECT OF INCORPORATED ^{226}Ra ON COLONY FORMING UNITS OF BONE MARROW IN SPLENECTOMIZED MICE. *(E.)* Klener, V. (Inst. Hygiene Epidemiology, Prague, Czechoslovakia) and V. Svoboda. *Acta Radiol* 12(2):155-164, 1973.

3115 CANCER AS A CAUSE OF ABORTIONS AND STILLBIRTHS: THE EFFECT OF THESE EARLY DEATHS ON THE RECOGNITION OF RADIOGENIC LEUKAEMIAS. *(E.)* Stewart, A. M. (Dept. Social Med., U. Oxford, England). *Br J Cancer* 27(6):465-472, 1973.

3116 ON TREATMENT OF EXPERIMENTAL TUMORS WITH LASER RADIATION ASSOCIATED WITH HIGH-SPEED ELECTRONS. *(Rus)* Kozlov, A. P. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR), A. A. Akimov and K. G. Moskalik. *Vopr Onkol* 19(6):93-97, 1973.

See also:

* (Rev): 3010, 3019
* (Chem): 3050
* (Viral): 3210

3117 STUDIES ON THE SUSCEPTIBILITY OF C57BL/6 MICE TO RAUSCHER VIRUS. III. KINETICS OF HUMORAL ANTIBODY RESPONSE TO CELL-SURFACE ANTIGENS IN NEONATALLY INOCULATED MICE.

(E.) Ishimoto, A. (Aichi Cancer Ctr. Res. Inst., Nagoya, Japan) and Y. Ito. *J Natl Cancer Inst* 51(2):553-564, 1973.

Adult C57BL/6 mice are resistant to tumor induction by both Gross and Rauscher leukemia viruses. Neonatal mice also are resistant to Gross virus, but susceptible to Rauscher virus. The present study was undertaken to determine to what extent the immune response of C57BL/6 mice may be related to their resistance and susceptibility to Rauscher virus. The procedure was to measure at monthly intervals the cell surface-reactive antibody of sera of the mice before and after inoculation with the virus. Purified virus was used for inoculating neonatal mice and homogenates of spleens of virus-infected BALB/c mice, for weanling or mature mice. The antibody was measured by indirect fluorescent staining, using serially diluted serum. AKR-C1 and RD-12 lines of cells were used as targets for Gross and Friend-Moloney-Rauscher antigens, respectively, both of which were present on the Rauscher virus-induced C57BL/6 lymphoma cells. The level of antibody titers against cell-surface antigens in the sera collected from C57BL/6 mice infected as weanlings or adults was higher than that of the natural antibody titers in normal mice. However, most titers in the sera of mice neonatally infected with the virus were lower than those of the natural antibody. Fluctuation patterns of the antibody titers in the mice neonatally infected with Rauscher virus were classified into 4 types: 1) runt and leukemia, 2) leukemia, 3) normal, and 4) immunized. Leukemias were most frequent in the mice having antibody titer patterns of the runt and leukemia type or leukemia type, both of which showed titers lower than those of natural antibody in normal mice. No mouse neonatally infected with Rauscher virus had specific immunologic tolerance to Gross and Friend-Moloney-Rauscher antigens. Some possible functions of humoral antibody to cell-surface antigens were discussed to explain the mechanisms of immune suppression by murine leukemia virus infection.

3118 ENHANCED GROWTH OF A POLYOMA TRANSFORMED TUMOR CELL IN *SPIROMETRA MANSONOIDES*-INFECTED HAMSTERS.

(E.) Tachovsky, T. G. (U. Rochester Sch. Med., N.Y.), J. D. Hare, A. L. Ritterson and J. F. Mueller. *Proc Soc Exp Biol Med* 143(3):780-782, 1973.

The growth of a polyoma virus-transformed tumor cell implanted in golden hamsters infected with *Spirometra mansonoides* spargana larvae was studied to determine the effect of the sparganum growth factor (SGF) on tumor growth. The rate of appearance of palpable tumors and relative tumor size were increased in infected animals while the 50% tumor dose was decreased fivefold. The enhanced tumor growth may be due to a direct response of the tumor cell to the growth factor or arise from an induced immunosuppressive state. Enhanced growth may also be an indirect consequence of overall host stimulation by the parasite or a combination of these effects.

3119 EFFECT OF HYDROCORTISONE ON CELL VIABILITY, EPSTEIN-BARR VIRUS GENOME EXPRESSION, AND INTERFERON SYNTHESIS IN HUMAN LYMPHOBLASTOID CELL LINES.

(E.) Joncas, J. (Inst. Microbiol. Hyg. Montreal, U. Montreal, Canada), J. Boucher, A. Boudreault and M. Granger-Julien. *Cancer Res* 33(9): 2142-2148, 1973.

The effect of hydrocortisone on six human lymphoblastoid cell lines was measured in relation to cell growth, number of Epstein-Barr (EB) virus antigen-positive cells, number of EB virus particles, and interferon synthesis. In two of the six cell lines (HRIK, MCI), hydrocortisone, in proportion to the dose, adversely affected cell viability concomitantly enhancing the expression of the EB virus genome. Recovery of normal viability in these two cell lines or lack of effect in the other three cell lines (EB3, Roswell Park Memorial Institute 1788, and AGI) appeared to be related to interferon synthesis. The observed effects occurred only in cell lines (HRIK, MCI) showing no detectable interferon prior to hydrocortisone, and recovery in these cell lines coincided with the appearance of detectable interferon. Conversely, in the three cell lines where interferon was either already present (AGI) or appeared within 3 days (EB3, Roswell Park Memorial Institute 1788), no adverse effect on cell viability and no definite activation of the EB virus genome could be demonstrated. In Raji, a few antigen-positive cells (4 to 23 per 10^5 cells) appeared but no viral particles. Cell viability was virtually unaltered and interferon was not detected. In Raji following bromodeoxyuridine (20 mg/ml) up to 1200 antigen-postive cells per 10^5 cells appeared with an associated interferon response. Priming of the HRIK cells with preformed human interferon, 500 to 1000 units/ml of cell suspension for 72 hr or more prevented the adverse effect of hydrocortisone (20 to 100 μg/ml) on cell viability. These findings may help to reestablish a relationship between interferon synthesis and viral replication in EB virus-carrying lymphoblastoid cell lines.

3120 COMPARATIVE STUDIES ON THE PATHOGENICITY FOR MICE OF TUMOUR HOMOGENATES AND OF NUCLEIC ACID EXTRACTED FROM CHICKEN SARCOMAS INDUCED BY THE BRYAN STRAIN OF ROUS SARCOMA VIRUS.

(E.) Nastac, E. (Stefan S. Nicolau Inst. Virology, Bucharest, Romania), M. Lungu and P. Athanasiu. *Rev Roum Virol* 10(2):139-146, 1973.

The oncogenic activity in random bred and CBA mice of homogenates from chicken sarcomas induced by Rous sarcoma virus (Bryan strain) (RSV-B) and of nucleic acid extracted by phenol from these cellular homogenates was examined. The induction period varied between 15 days and 20 months. In two groups of animals, random bred mice from females inoculated i.p. during pregnancy and newborn mice inoculated with RSV-B nucleic acid in the first 48 hr of life, macroscopic solid tumors were obtained. In the other groups of animals, the neoplastic disease was detected only microscopically. No significant differences were found between RSV-B and RSV-B nucleic acid pathogenicity for the same mouse line. Neither were there any differences depending on the inoculation route.

Immunofluorescence reactions showed group-specific Rous antigens in organ sections from mice with the experimental disease in 12% of the cases as well as anti RSV-B antibodies in the serum in 23% of cases examined. The oncogenic activity of the nucleic acid extracts might be ascribed to the RNA of Rous sarcoma virus or to its DNA provirus, possibly extracted by phenol from the chicken sarcomas. RNAase did not always inhibit the oncogenic activity of nucleic acid extracts.

3121 PROPAGATION OF RAUSCHER LEUKEMIA VIRUS TO HUMAN LYMPHOBLASTOID CELLS BY *IN VIVO* DIFFUSION CHAMBER CULTIVATION. *(E.)* Miyoshi, I. (Okayama U. Med. Sch., Japan), S. Yoshimoto, T. Tsubota, S. Fujiwara, H. Dabasaki, S. Abe and S. Irino. *Proc Soc Exp Biol Med* 143(3):850-853, 1973.

Diffusion chambers containing Epstein-Barr virus (EBV)-bearing human lymphoblastoid cells were placed temporarily within the peritoneal cavity of BALB/c mice with Rauscher leukemia. Reestablished human leukocyte cultures became dually infected with C type and EBV particles and have continuously produced abundant C type particles. These virus particles were nonleukemogenic when inoculated into BALB/c mice but afforded some protection against challenge infection with spleen-derived leukemogenic Rauscher leukemia virus, indicating that there are antigenic similarities between these two viruses. The advantage of the technique used in these experiments appears to be that cells are exposed to virus continually under more physiologic circumstances.

3122 THE EVOLUTION OF NEW SPECIES OF VIRAL DNA DURING SERIAL PASSAGE OF SIMIAN VIRUS 40 AT HIGH MULTIPLICITY. *(E.)* Brockman, W. W. (Johns Hopkins U. Sch. Med., Baltimore, Md.), T. N. H. Lee and D. Nathans. *Virology* 54(2):384-397, 1973.

Viral DNA isolated during serial undiluted passage of SV40 was analysed by electrophoresis of fragments produced by *H. influenzae* restriction endonuclease and by DNA-DNA hybridization. Compared to parental SV40 DNA, early passage DNA showed alterations at many sites in the population of DNA molecules, although certain sites were more frequently altered than others as evidenced by the reduced yield of some original DNA fragments and the appearance of new fragments. With continued passage, new dominant species of viral DNA evolved which yielded simple restriction endonuclease digest patterns. DNA molecules from all passages retained SV40 DNA sequences; however, the percentage of total DNA sequences which corresponded to SV40 sequences diminished to about 30% or less in late passage DNA. Repetitive host cell DNA sequences were detected in a high percentage of late passage DNA molecules, but accounted for 20% or less of total sequences present. Therefore the new species of viral DNA which evolved probably contain predominantly nonrepetitive host DNA sequences. The primary events in the mechanism producing this new species probably consist in the recombination between or within SV40 DNA molecules or between SV40 DNA and host DNA, the latter representing integration and mis-excision of viral DNA. In addition, errors of DNA replication may give rise to duplication or deletion of segments. Once a primary change has occurred, selection of the most rapidly replicating molecules might then ensue.

3123 A SIMPLE VIABILITY TEST OF CELLS PRODUCING VIRAL ANTIGEN. *(E.)* Mizuno, F. (Hokkaido U. Sch. Med., Sapporo, Japan) and T. Osato. *Arch Gesamte Virusforsch* 41:386-389, 1973.

A method has been devised for the simultaneous observation of viral synthesis and cell viability in Epstein-Barr virus (EBV)-carrier cultures. The method involves dye exclusion and immunofluorescence. A suspension of EBV-producing Burkitt cells was briefly mixed with an equal volume of 0.2% trypan blue at room temperature. The cells were washed, spread on glass microscope slides and dried for 90 minutes. After fixation with acetone, immunofluorescence staining was carried out by an indirect method. Standard serum from a patient with nasopharyngeal carcinoma containing antibodies to EBV was placed on the smears and allowed to stand for 60 minutes at 36 C. The slides were rinsed and fluorescein isothiocyanate-conjugated anti-human γ-globulin rabbit serum was applied. The immunofluorescence and trypan blue dye exclusion were simultaneously observed in the same microscope field changing UV to visible light. The pretreatment of cells with trypan blue yielded no detectable autofluorescence and was not unfavorable for the immunofluorescence reaction.

3124 VIRUS-FREE ADENOCARCINOMA OF THE FROG (SUMMER PHASE TUMOR) TRANSCRIBES LUCKE TUMOR HERPESVIRUS-SPECIFIC RNA. *(E.)* Collard, W. (St. Louis U. Sch. Med., Mo.), H. Thorton, M. Mizell and M. Green. *Science* 181(4098):448-449, 1973.

Winter phase renal carcinomas of the leopard frog have been shown to contain herpesvirus particles whereas summer phase tumors do not, and prolonged exposure to lowered temperature results in the change from the "virus-free" summer state to the overtly virus-containing winter state. The present work shows that ^{3}H-RNA isolated from summer phase renal adenocarcinomas of frogs labeled with ^{3}H-uridine hybridizes with DNA of herpesvirus particles isolated from the winter phase tumor. RNA from normal frog kidney did not hybridize significantly with the viral DNA. The observed transcription of the herpesvirus genome in virus-free tumors thus provides additional evidence for the viral etiology of the tumor.

3125 NEOPLASM INDUCTION BY MURINE TYPE-C VIRUSES PASSAGED DIRECTLY FROM SPONTANEOUS NON-LYMPHORETICULAR TUMOURS. *(E.)* Peters, R. L. (Microbiol. Assoc., Walkersville, Md.), G. J. Spahn, L. S. Rabstein, G. J. Kelloff and R. J. Huebner. *Nature* [*New Biol*] 244(134):103-105, 1973.

The incidence of lymphoreticular (LR) neoplasms was studied in Balb/cCr mice inoculated i.p. or s.c.

with murine leukemia virus (MuLV)-containing spleen or LR tumor preparations from tumor-bearing adult Balb/cCr donors. Inoculation of mice within 72 hr after birth with extracts containing MuLV detectable by either infectivity or complement fixation tests caused a significantly higher incidence of LR tumors within 2 yr than did inoculation with extracts without detectable MuLV ($P < 0.001$). The incidence of LR neoplasms in mice inoculated with extracts in which MuLV was undetectable was similar to that of the uninoculated controls (10%). In almost all incidences, the mean latent period for induction of LR tumors by virus-positive inocula was significantly less (by 48 to 127 days) than that for virus-negative extracts. A clear association was found between the presence of B-tropic virus (virus variant growing preferentially on Balb/c fibroblasts rather than NIH Swiss mouse fibroblasts) in the tumor and spleen inocula and the ability to induce neoplasms.

3126 POLY A AND SYMMETRICAL TRANSCRIPTION OF SV40 DNA. *(E.)* Aloni, Y. (Weizmann Inst. Sci., Rehovot, Israel). *Nature [New Biol]* 243(122):2-6, 1973.

Poly A has been shown to be attached to self-complementary RNA produced during a symmetrical transcription of SV40 virus DNA late in the infection of monkey BS-C-1 cells. Poly A-containing molecules were isolated from rapidly labeled RNA by binding to and elution from poly U-GF/C filters. The poly A was self-annealed, digested with RNase, and subjected to Sephadex G-100 chromatography. A peak of poly A containing material, representing 10-15% of the total labeled RNA extracted from BS-C-1 cells could be isolated in this manner. This material showed a specific and increased efficiency of hybridization of SV40 DNA and to cellular complementary RNA. The characteristic behavior of the self-annealed poly A-RNA to RNase, DNase, temperature changes and buoyant density centrifugation established that it consisted of double-stranded RNA molecules, and it was thus suggested that poly A was attached to self-complementary RNA produced during symmetrical transcription of SV40 DNA late in infection. Based on these data and on previous findings, it was proposed that percursor viral RNA molecules are transcribed symmetrically after which poly A is added to both self-complementary transcripts. Certain sequences are then degraded to yield poly A-containing non-complementary mature message.

3127 RESCUE OF CHEMICALLY INACTIVATED ROUS SARCOMA VIRUS TRANSFORMING ACTIVITY BY AVIAN LEUKOSIS VIRUS. *(E.)* Hung, P. P. (Abbott Lab., Chicago, Ill.). *Virology* 53(2):463-467, 1973.

The Bryan high-titer strain of Rous sarcoma virus, RSV(RAV-2), lost its ability to transform cultured chick embryo fibroblasts following treatment for 30 min with 10^{-2} or 10^{-3} M N-ethylmaleimide (NEM). The loss of transforming activity was accompanied by loss of reverse transcriptase activity as determined by *in vitro* radioassay using exogenous dTrA template. However, NEM-inactivated RSV(RAV-2) recovered its transforming activity when cells were coinfected with a non-transforming avian leukosis virus (RAV-1) which possessed active reverse transcriptase. Immunological analysis of progeny virus recovered after several passages indicated that RAV-1 rescued both inactivated RSV(RAV-2) and inactivated RAV-2, a sarcoma-leukemia complex also present in the Bryan high-titer stock. These results were consistent with a possible role of reverse transcriptase in the transformation process.

3128 SUPERINFECTION WITH EPSTEIN-BARR VIRUS OF HUMAN LYMPHOID CELL LINES OF DIFFERENT ORIGIN. *(E.)* Adams, A. (Karolinska Inst., Stockholm, Sweden) and G. Klein. *Nature [New Biol]* 242(121): 234-236, 1973.

The responses of eleven established virus receptor positive lymphoid cell lines to superinfection with Epstein-Barr (EB) virus have been studied. All lines showed a good capacity for adsorbing ^{3}H-labeled EB virus, with values ranging from 0.5 to 1.2 times the adsorbing capacity of the Raji line. Three lines (Raji, Daudi and NC-37) showed a significant increase in early antigen (EA) on superinfection. The other eight lines showed little or no EA response at moderate EB virus multiplicities. The line derived from a normal individual (NC-37) had the greatest susceptibility of all lines to superinfection when virus titration studies were performed, although the maximum virus adsorption capacity was the same as that for the two easily infectable Burkitt's lymphoma derived lines (Raji and Daudi). Among the eight poorly infectable lines, four could be stimulated to produce EA by high EB virus multiplicities. The other four could not, indicating the existence of at least two types of cellular interference that could block EB virus antigen expression. No correlation was found between the type of superinfection response and the producer or nonproducer status of the line.

3129 POLYOMA GENE FUNCTION REQUIRED FOR VIRAL DNA SYNTHESIS. *(E.)* Francke B. (Salk Inst., La Jolla, Ca.) and W. Eckhart. *Virology* 55(1):127-135, 1973.

The ts-a mutant of polyoma virus, and other mutants of the ts-a type, were previously shown to be defective in viral DNA synthesis, T-antigen synthesis and stable transformation at the restrictive temperature of 39 C. The present kinetic study, carried out with mutant-infected BALB/3T3 cells, showed that viral DNA synthesis ceases following a shift from the permissive (32 C) to the restrictive temperature. Inhibition of RNA synthesis by actinomycin D or of protein synthesis by cycloheximide during a shift up in temperature has no effect on the cessation of viral DNA synthesis. Pulse-chase experiments showed that viral DNA molecules already initiated at 32 C could be completed at 39 C and suggested that initiation of new rounds of viral DNA synthesis is blocked at the restrictive temperature. Completion of rounds of viral DNA synthesis at the restrictive temperature occurs at the same rate in mutant-infected cells as in wild-type infected cells. It appeared likely that a viral gene product is required for initiation of rounds of viral DNA replication in lytic infection and also for stable transformation.

3130 VARIOUS FORMS OF EPSTEIN-BARR VIRUS INFECTION IN MAN: ESTABLISHED FACTS AND A GENERAL CONCEPT. *(E.)* Epstein, M. A. (U. Bristol Med. Sch., England) and B. G. Achong. *Lancet* (7833):836-839, 1973.

Herpes viruses in general can infect in several ways. With productive infection there is virus replication and cell death, and with non-productive infection the viral genome may be unexpressed or expressed in malignant transformation. Clinical, epidemiological, and *in vitro* evidence shows that Epstein-Barr (E.B.) virus manifests all three modes of infection. Most primary infections by E.B. virus occur in childhood and early adolescence and are accompanied by permanent seroconversion and total immunity to infectious mononucleosis, and by lifelong harboring of the virus. This harboring is manifested by productive infection of cells somewhere in the oropharynx with release of infectious virus into the buccal fluid and by non-productive infection of peripheral lymphoid cells which therefore contain the unexpressed virus genome. Nasopharyngeal and Burkitt's lymphoma patients have antibodies to E.B. virus, and evidence suggests that E.B. virus is present in Burkitt's lymphoma cells in the form of non-productive infection in which the virus genome is expressed in malignant transformation. E.B. virus is clearly a potent stimulator of lymphoproliferation *in vivo* during infectious mononucleosis, and confers the power of unlimited growth on human lymphoid cells *in vitro*, accompanied by changes characteristic of malignant transformation brought about in normal cells in culture by known oncogenic animal viruses. E.B. virus shows absence of virus particles in the tumor; expression of specific viral genetic activity by the production of viral determined neoantigens in tumor cells and transformed culture cells; and ability of the virus genome to be activated to a productive infection by treatment with B.U.D.R. In light of this evidence it seems likely that E.B. virus has an oncogenic role in man. The virus might, in a rare individual, induce actual malignant lymphorpoliferation. In tropical regions where Burkitt's lymphoma is endemic some climate-dependent co-factor must be superimposed on this pattern.

3131 B-TYPE ONCORNAVIRUSES ISOLATED FROM CONTINUOUS HUMAN CANCER CELL LINES. *(E.)* Bykovsky, A. F. (N. F. Gamaleya Inst. Epidemiology Microbiol., Moscow, USSR), G. G. Miller, F. I. Yershov, K. V. Ilyin and V. M. Zhdanov. *Arch Gesamte Virusforsch* 42(1):21-35, 1973.

Eleven lines and sublines of larynx carcinoma (HEp-2), cervical cancer (HeLa) and dedifferentiated astrocytoma (DAPT) cells were examined and viruses were isolated from eight lines. Electronmicroscopic, biophysical and biochemical examinations of the viruses showed that they belong to the B-type oncornaviruses. The virions had a size of 100 to 150 nm, they banded at densities of 1.16 to 1.17 g/ml in sucrose gradients and RNA from the virions contained 60-70S component. The reproduction of the viruses was inhibited by actinomycin D, and the virions possessed reverse transcriptase (RNA-dependent DNA polymerase) activity. The isolated viruses differed antigenically from mouse mammary tumor virus. The cell cultures preserved the human karyotype and were not contaminated with foreign cells. Since all tissue cultures were maintained in the presence of bovine serum, it may be supposed that the virus isolates could be contaminants of bovine or swine origin. This possibility is not excluded by these experiments, although oncornaviruses of the B type have not yet been discovered in cattle or swine.

3132 RNASE H AND A: EFFECTS ON DNA TEMPLATE SPECIFICITY OF RNA POLYMERASE FROM NORMAL AND FRIEND VIRUS-INFECTED MOUSE SPLEEN. *(E.)* Munson, B. R. (Springville Lab., New York) and C. L. Tober. *J Natl Cancer Inst* 51(2):631-635, 1973.

Compared to normal spleens, spleens of mice with Friend virus (FV) leukemia were previously shown to show a 5- to 8-fold increase in RNA synthesis in either aggregate enzyme or nuclei. During the course of studies designed to understand the molecular events responsible for this difference, DNA-dependent RNA polymerase from each source was solubilized by sonic disruption of nuclei in the presence of 0.3M ammonium sulfate. The resulting enzyme activities were similar to those of RNA polymerases from other eukaryotic sources. Template specificities of the enzyme preparations from the 2 sources differed notably however. The normal tissue preparation strongly preferred denatured DNA as its template, whereas the FV-infected tissue preparation preferred native DNA as its template. Interfering proteins and degradatory enzymes were detected, which may explain the difference in the RNA polymerase activities from normal and leukemic cells. The normal enzyme preparation had significant amounts of RNase A activity, whereas the FV-infected tissue had none. In addition, the preparation from FV-infected tissue had a large amount of RNase H activity, whereas the corresponding normal tissue had much less. The net result was an apparent DNA template specificity which was really a specificity of product degradation.

3133 ATP REQUIREMENT FOR RELEASE OF ADENOVIRUS mRNA FROM ISOLATED NUCLEI. *(E.)* Raskas, H. J. (St. Louis U. Sch. Med., Mo.) and Y.-C. Rho. *Nature* [*New Biol*] 245(141):47-49, 1973.

The release of adenovirus RNA from human cell nuclei incubated with 1 mM ATP, 5 mM creatine phosphate, and 20 μg/ml creatine kinase was reported previously. In the present work it was found that increasing the concentration of ATP stimulated the reaction, abolished the requirement for creatine phosphate and kinase, and changed the properties of RNA produced from predominantly 4S-7S to predominantly 18S-28S (mRNA). AMP was only 5-10% as effective as ATP; ADP, 10-10% as effective. The extent of ATP-dependent RNA release was affected by concentration of Mg^{++} as shown by the observation that at 3 mM ATP, concentrations of Mg^{++} of 5 mM or higher were strongly inhibitory while concentrations below this level led to increasing production of RNA. Although the activity of ATP was thus antagonized by Mg^{++} it appeared unlikely that ATP functioned simply by chelating divalent ions from nuclei since pyrophosphate and EDTA failed to mimic the action of ATP. It was suggested that Mg^{++} may stabilize nuclear sites so that they are nonresponsive to ATP or may form Mg-ATP complexes which will not function in the RNA-release reaction. Both AMPPCP and AMPCPP (analogues of ATP containing methelene groups rather than oxygen atoms in the α-β or β-γ positions) were as effective as ATP, suggesting that the *in vitro* release reaction does not require hydrolysis of a specific high energy bond. It was pointed out that if, as indicated, RNA release does not require energy, the effect of ATP on nuclei would resemble the interaction of ATP with muscle. The interaction of ATP with isolated nuclei may, therefore, relax or dissociate a structure which retains mRNA in the nucleus. To reform this structure for reception of a subsequent mRNA molecule may then require the energy derived by hydrolysis of ATP. Although the present results provided no direct evidence that the interaction of nuclei with ATP yields a reaction analogous to mRNA transport *in vivo*, the observation that ATP stimulates mRNA release from nuclei, as well as from cores of the cytoplasmic vaccinia virus, as previously reported, suggests a common effect on all cellular components that export mRNA.

3134 MURINE C-TYPE RNA VIRUS FROM SPONTANEOUS NEOPLASMS: *IN VITRO* HOST RANGE AND ONCOGENIC POTENTIAL.

(E.) Peters, R. L. (Microbiol. Associates, Inc., Walkersville, Md.), G. J. Spahn, L. S. Rabstein, G. J. Kelloff and R. J. Huebner. *Science* 181(4100):665-667, 1973.

The incidence of murine C-type RNA leukemia viruses of each of the two tropisms present in a variety of spontaneous neoplasms of the BALB/cCr mouse strain were evaluated and the association between the virus type and its oncogenic potential were determined. One variant, which is host-cell tropic (B-tropic), is the predominant isolate from neoplastic tissues and produces lymphoreticular neoplasms when injected into BALB/c newborn mice. A second variant, which grows preferentially in NIA Swiss mice (N-tropic), is isolated throughout life, grows poorly in host embryonic cells in culture and is not associated with lymphoreticular neoplasm induction when injected into newborn BALB/c mice. The failure of the N-tropic variant to induce tumors in the BALB/c host might, however, have been due to use of too low a dose.

3135 PROPERTIES OF MOUSE LEUKEMIA VIRUSES. V. HEMAGGLUTINATION-INHIBITION AND INDIRECT HEMAGGLUTINATION TESTS.

(E.) Witter, R. (Max-Planck Inst. Virus Res., Tubingen, Germany), G. Hunsmann, J. Lange and W. Schäfer. *Virology* 54(2):346-358, 1973.

A specific hemagglutination-inhibition (HI) test system for mouse leukemia virus (MuLV) has been devised. The presence of nonspecific inhibitors precluded the use of untreated sera in HI tests with MuLV hemagglutinating antigens. However, preparations of immunoglobulin G (IgG) lacked nonspecific inhibitory activity and were suitable for use. Hemagglutination (HA) induced by intact, enzyme-treated MuLV and by hemagglutinating MuLV subunits was inhibited by IgG isolated from MuLV antisera. Low titer HI reactions were obtained with IgG from feline leukemia virus (FeLV) antisera, and IgG from a broad spectrum of control antisera was noninhibitory. The HI activities of three FMR (Friend-Moloney-Rauscher) antisera against FMR antigens were totally type-specific as revealed by absorption tests. Gross leukemia virus (GLV) antisera also appeared to react type-specifically against GLV antigen. However, the inhibition of FMR antigens by IgG from one nonneutralizing FMR (gs) antiserum, GLV antisera and FeLV antisera, all of which lacked FMR type-specific antibodies, indicated that gs-like reactivity also could be detected by the HI test in at least certain sera. In related studies, an indirect HA reaction employing heterotypic Friend virus antiserum (IgG) revealed a new, possibly monovalent hemagglutinin from degraded GLV. The HA activity of this GLV antigen could be reconstituted by most FMR sera tested, but not by sera directed against GLV, FeLV or other control antigens. The reconstituted hemagglutinin was specifically inhibited by homotypic GLV antiserum (IgG). This indirect HA test not only constitutes direct evidence for the existence of gs antigenic determinants on the surface of the virion but also provides a tool for the detection of such antigens and their corresponding antibodies.

3136 RD-114 VIRUS COMPARED WITH FELINE AND MURINE TYPE-C VIRUSES RELEASED FROM RD CELLS.

(E.) McAllister, R. M. (U. Southern California Sch. Med., Los Angeles), M. Nicolson, M. B. Gardner, S. Rasheed, R. W. Rongey, W. D. Hardy, Jr. and R. V. Gilden. *Nature* [*New Biol*] 242(116):75-78, 1973.

The morphological, growth, and immunological characteristics of cultured human rhabdomyosarcoma (RD) cells infected with RD-114 virus were compared with those of uninfected RD cells and of RD cells infected with feline leukemia virus (RD-FeLV) or Kirsten murine sarcoma virus (RD-KiMSV). Whereas RD and RD-114 cultures consisted of two types of cells, polygonal and strap, RD-FeLV and RD-KiMSV cultures consisted primarily of polygonal cells. The chromosome patterns of RD-FeLV and RD-KiMSV cells were similar to those of RD cells, whereas that of RD-114 cells consisted of a higher percentage of ring forms. The saturation densities and generation times of the four lines were similar. RD-114, RD-FeLV and RD-KiMSV, but not RD, cells shed C-type virus particles observable under the electron microscope. Particles of all 3 viruses incorporated ^{3}H-uridine, had a density of about 1.16 gm/cm^3, contained 70S RNA and RNA-dependent DNA polymerase activity, and possessed a capacity to rescue murine sarcoma virus. However, RD-114, RD-FeLV and RD-KiMSV cells reacted specifically in complement fixation tests to antisera prepared against their own respective gs-1 antigens. RD cells contained no gs-1 antigen. Furthermore, antisera to FeLV or MuLV polymerases failed to inactivate RD-114 virus polymerase and antisera to RD-114 virus polymerase failed to inhibit FeLV or MuLV polymerases. The findings indicated, therefore, that the RD-114 virus is not a feline or murine type-C virus contaminant.

3137 PLASMA MEMBRANE PROTEINS OF NORMAL AND ROUS SARCOMA VIRUS-TRANSFORMED CHICK-EMBRYO FIBROBLASTS.

(E.) Wickus, G. G. (Dept. Biol., Massachusetts Inst. Technol., Cambridge) and P. W. Robbins. *Nature* [*New Biol*] 245(142):65-67, 1973.

To compare plasma membrane proteins of normal and transformed cells, cultures of chick embryo fibroblasts (CEF) infected with the thermosensitive mutant (Ts-68) of the Schmidt-Ruppin strain of Rous sarcoma virus, subgroup A (SR-RSV-A), were incubated at a permissive temperature of 36 C to produce fully transformed cells or, subsequently, at a non-permissive temperature of 41 C to reverse the morphology to that of normal cells. The two different cultures were labeled with ^{35}S L-methionine and plasma membrane vesicles were prepared from each. The vesicle preparations were then disrupted in sodium dodecyl sulfate-mercaptoethanol and subjected to electrophoresis on polyacrylamide slab gels. Positions of the labeled polypeptides on the slabs were determined by autoradiography. Of 26 polypeptides seen on the gel for the cells with normal morphology, one was greatly reduced in the gel for the cells with transformed morphology. Additional studies were made with cell ghosts from the two different cultures and with membrane vesicles from uninfected cultures and cultures transformed by wild type RSV. The results all showed that the disappearance of only one polypeptide component was the major transformation specific alteration in plasma membrane preparations from RSV-transformed chick cells.

3138 CHARACTERIZATION OF HERPES SIMPLEX VIRUS-INDUCED DEOXYRIBONUCLEIC ACID POLYMERASE.

(E.) Weissbach, A. (Roche Inst. Molecular Biol., Nutley, N.J.), S.-C. L. Hong, J. Aucker and R. Muller. *J Biol Chem* 248(18):6270-6277, 1973.

In view of the importance that knowledge about herpes simplex virus (HSV)-induced DNA polymerase may have in understanding the regulatory events in infected cells or in the process of DNA replication, the purification of induced DNA polymerase and separation from host DNA polymerase and HSV-induced DNase was undertaken. Infection of HeLa cells with herpes simplex virus led to a large increase in a new DNA polymerase activity in the nucleus of infected cells. The HSV-induced DNA polymerase was purified 400- to 1300-fold from infected cells and shown to be different in its properties from the normal host cells DNA polymerases. The HSV-induced DNA polymerase appeared as two peaks of activity on DEAE-cellulose in the presence of glycerol. However, these two DNA polymerase activities seemed to have the same molecular weight (1.8 x 10^5) and exhibited similar properties after further purification on phosphocellulose. The HSV-induced DNA polymerase was markedly different from the host DNA polymerases in its high salt requirement for maximal activity. It showed a strong preference for an oligomer-homopolymer template containing deoxyguanylate and deoxycytidylate. A nuclease activity which degrades the polydeoxyadenylate-deoxythymidylate copolymer and the deoxyguanylate-deoxycytidylate homopolymer pair was found to co-chromatograph with the herpes simplex virus-induced DNA polymerase. The nuclease activity measured with these synthetic DNA's solubilized the substrate at about 5% the rate of the synthetic activity of the DNA polymerase with activated DNA.

3139 HUMAN PAPOVAVIRUS (JC): INDUCTION OF BRAIN TUMORS IN HAMSTERS.

(E.) Walker, D. L. (U. Wis. Med. Sch., Madison), B. L. Padgett, G. M. ZuRhein, A. E. Albert and R. J. Marsh. *Science* 181 (4100):674-676, 1973.

Fifty two of 63 hamsters inoculated intracerebrally or s.c. at birth with JC virus, a human papovavirus isolated from brain tissue of a case of progressive multifocal leukoencephalopathy, developed malignant gliomas within six months. All tumors appeared on the brain and none appeared at the site of s.c. inoculation. The majority of these gliomas were of types not previously reported to be induced by papovaviruses in *in vivo* experiments. Three of the tumors have been serially transplanted as subcutaneous tumors. JC virus was isolated from five of seven tumors tested. Cells from four tumors were cultivated *in vitro*. These cells contained an intranuclear antigen with the characteristics of a T antigen, and this antigen was antigenically related to SV40 T antigen. Although virus was not recovered from extracts of serially cultured tumor cells, JC virus was rescued when one tumor cell line was fused with permissive cells, giving evidence for the continued presence of the virus genome.

3140 DEPLETION OF SERUM GROWTH FACTORS BY 3T3 MOUSE FIBROBLASTS AND VIRAL TRANSFORMANTS.

(E.) Roehm, C. (Milton S. Hershey Med. Ctr., Hershey, Pa.) and A. Lipton. *Nature* [*New Biol*] 245(143): 115-116, 1973.

The depletion properties and growth factor requirements of 3T3 cells, SV-3T3 cells and other 3T3 virus-transformed cell lines were investigated. Media which were depleted by exposure to growing 3T3, SV-3T3, SV-Fl$_2$-101 and Py-3T3 mouse fibroblasts, were collected daily from the cultures, concentrated 10-fold by lyophilization and dialyzed against serum-free media. The media were then assayed for ability to support cell growth, to determine which factor was utilized, and to assess the degree of depletion by each cell line. All 3T3 virus-transformed cell lines studied were found to require and to deplete a SV-3T3 growth factor from serum-containing medium. The SV-3T3 growth factor was different from that serum factor which was most limiting for the growth of 3T3 mouse fibroblasts. Dialysis of depleted media against serum-containing media did not "recharge" the depleted media, showing that both the 3T3 growth factor and the SV-3T3 growth factor had large molecular wt.

3141 LOW-LEUKEMOGENIC VARIANTS OF RAUSCHER LEUKEMIA VIRUS OBTAINED IN LONG-TERM CULTURES AT SUPRA-OPTIMAL TEMPERATURE. *(E.)* Barski, G. (Inst. Gustave-Roussy, Villejuif, France), D. Barbieri, J. K. Youn and G. Hue. *Int J Cancer* 12(1)55-65, 1973.

The rate of replication and leukemogenic potential of Raushcer leukemia virus (RLV) were studied using chronically infected Balb/c thymus-spleen and C57BL/6 lung tissue cultures maintained for several months at either 37 C or 40.5 C. Cells maintained for up to 586 days at 40.5 C showed a decreased modal chromosome number, the appearance of a new long metacentric marker, and the loss of two other markers. Inoculation of newborn or weanling Balb/c mice with cell culture supernatants showed that supernatants from cultures maintained at 40.5 C had a much lower leukemogenic potential (11%) with a longer latent period (98 days) than did those from cultures maintained at 37 C (89%, 23 days). The late-appearing leukemias produced by material from the cultures at 40.5 C were of Rauscher erythroblastic type. Morever, actual counts performed on the supernatants showed that those from cultures maintained at 40.5 C were on the whole 6 times higher than those from cultures maintained at 37 C. Therefore, the differences between supernatants from cultures at 40.5 C and those from cultures at 37 C were not due to differences in infectivity or to the induction of a new type-C virus in the 40.5 C cultures.

3142 ELECTRON MICROSCOPIC IDENTIFICATION OF PAPOVA VIRUS IN LARYNGEAL PAPILLOMA. *(E.)* Boyle, W. F. (San Francisco Med. Ctr., Calif.) J. L. Riggs, L. S. Oshiro and E. H. Lennette. *Laryngoscope* 83(7):1102-1108, 1973.

A continuing effort to define the relationship of the papilloma virus to human laryngeal papilloma is described. Attempts to propagate a laryngeal papilloma virus in cell culture by the establishment of tissue lines from 27 papilloma specimens which were removed endoscopically from the larynx of 19 patients of all age groups were unsuccessful. Papillomas from two of the patients contained papova-like virus when examined by electron microscopy. A specimen from a 22-year-old Caucasian male revealed intranuclear viral particles which were in the shape of an icosahedron and measured approximately 45 mμ in diameter. The particles contained a densely staining core and were generally scattered throughout the nucleus. Occasionally, they were seen in crystalline arrays. The nuclear site of replication, their size and shape fit the description of the agents belonging to the papová virus group. Papova-like viruses were also seen in laryngeal papillomas from an 18-month-Negro child born of a mother with venereal warts. Clinical evidence of the transmissability of this disease from infected genital warts to the newbown was suggested by the development of laryngeal warts in the young child. The electron microscopic identification of virus in the laryngeal papilloma that is morphologically similar to the genital wart virus supports this theory of transmissability. The results of these studies add strong morphologic and clinical evidence of the viral etiology of laryngeal papilloma; however, the evidence is not conclusive.

3143 MAPPING OF BASE SEQUENCE HETEROLOGIES BETWEEN GENOMES FROM DIFFERENT ADENOVIRUS SEROTYPES. *(E.)* Garon, C. F. (Natl. Inst. Allergy Infectious Dis., Bethesda, Md.), K. W. Berry, J. C. Hierholzer and J. A. Rose. *Virology* 54(2):414-426, 1973.

An electron microscopic heteroduplex technique was used to physically map sequence heterologies between genomes from different human adenovirus serotypes. When DNA from serotypes was mixed, denatured, and renatured, characteristic heteroduplex molecules were formed. These molecules, composed of one strand from one serotype and one strand from the other serotype, contained one or more detectable regions of strand separation (or heterology), depending upon denaturing conditions and the combination of type-specific DNA preparations. By increasing denaturing conditions, regions of partial homology could be demonstrated in all interserotypic heteroduplexes. The heterology patterns in heteroduplexes constructed with DNA from nononcogenic serotypes (Ad 1, 2, and 5) were relatively simple and nearly similar, whereas both simple and complex patterns were seen in heteroduplexes prepared with DNA from weakly oncogenic serotypes (Ad 3, 7, and 21). Only complex heterology patterns were found in heteroduplexes prepared with DNA from highly oncogenic serotypes (Ad 12, 18, and 31). As expected, the most extensive heterologies were observed in heteroduplexes prepared with DNA from serotypes belonging to the different serologic groups. Within groups, heteroduplex molecules contained two specific regions in which heterologies were most marked. These regions appeared to map at similar locations among heteroduplexes from all groups. It was found that the integrated SV40 virus DNA in an Ad 7-SV40 hybrid genome was situated in one of these regions.

3144 THE EPSTEIN-BARR VIRUS CARRIER STATE IN INFECTIOUS MONONUCLEOSIS. *(E.)* Mycind, N. (Blegdams Hosp., Copenhagen, Denmark). *N Engl J Med* 289(16):867, 1973.

A lymphocyte-transforming particle, Epstein-Barr (EB) virus, has been observed in throat washes from patients with infectious mononucleosis. White cells in the peripheral blood were examined and serum oxalacetic transaminase and lactic dehydrogenase (LDH) isoenzymes were determined in 42 patients with infectious mononucleosis. Patients were followed with weekly, and later monthly, tests for six months. A sustained elevation of LDH was observed, which presumably originates from tissue leukocytes stimulated by EB virus, since leukocytes in peripheral blood are quantitatively normal 4 wk after the onset of disease. It appears that infectious mononucleosis occurs in three phases: the clinical disease with blood leukocytosis and massive proliferation in the lymphoid system in the whole body; a recovery state lasting for several weeks without blood leukocytosis, but with a slowly decreasing rate of proliferation in the lymphoid system; and a passive carrier state, lasting for several months, in which the only abnormal finding is a lymphoid-transforming principle, identified as EB virus, in the throat washes.

3145 SUPERINDUCTION OF ENDOGENOUS TYPE C VIRUS BY 5-BROMODEOXYURIDINE FROM TRANSFORMED MOUSE CLONES.

(E.) Lieber, M. M. (Natl. Cancer Inst., Bethesda, Md.), D. M. Livington and G. J. Todaro. *Science* 181(4098):443-444, 1973.

Certain transformed subclones of the mouse cell line BALB/3T3 release 5 to 15 times more type C virus cell than the parent cell line after treatment with 5-bromodeoxyuridine (BrdU). Amount of virus produced was measured by assay for viral reverse transcriptase. Virus release begins within 8 hrs and increases exponentially for the first 24 to 48 hr. Superinducibility is associated with the transformed phenotype. The rapidity with which virus particles can be secreted from such cells after treatment with BrdU suggests that the viral information is present in the cells in a form that allows for prompt expression. Virus production by the superinducible clones is comparable in magnitude to productive infections with exogenously added laboratory strains of murine leukemia virus; however, the latent period is markedly shorter than that found with exogenous infection by RNA tumor viruses.

3146 COMPARATIVE CHARACTERIZATION OF A C-TYPE VIRUS-PRODUCING CELL LINE (VSW) AND A VIRUS-FREE CELL LINE (VH2) FROM *VIPERA RUSSELLI.*

(E.) Clark, H. F. (Wistar Inst., Philadelphia, Pa.), M. M. Cohen and P. D. Lunger. *J Natl Cancer Inst* 51(2):645-657, 1973.

A C-type virus isolated from a cultured cell line (VSW) of a myxofibroma of the spleen of a Russell's viper (*Vipera russelli*) had been found to be antigenically distinct from known mammalian and avian C-type viruses and represented the only poikilothermic vertebrate oncornavirus yet propagated *in vitro*. In the present work a nonvirogenic cell line (VH2) of the homologous species was established from the heart of a tumor-free viper and its properties were compared with those of the VSW line. VSW cells adapted rapidly to continuous subculture *in vitro*, with a stable, small, epithelial-like cell morphology and a stable aneuploid karyotype established by cell passage 10. The VH2 cell line was established more slowly; the emergent cell type was larger and fibroblast-like with a quasidiploid karyotype. Chromosomal abnormalities were common in VSW cells only. Electron microscopy revealed that VSW cells had a higher nuclear-cytoplasmic ratio and a less differentiated cytoplasmic appearance than the apparently more metabolically active VH2 cells. Both cells types grew optimally at an incubation temperature near 30 C, but VH2 cells grew better at suboptimal and supraoptimal temperatures. VH2 cells also had a superior tolerance to growth in the presence of dextran sulfate. VSW cells supported the replication of rabies and vesicular stomatitis viruses more efficiently than did VH2 cells. However, vaccinia, herpes simplex, pseudorabies, Sendai, and Newcastle's disease viruses replicated in VH2, but not in VSW cells.

3147 TRANSFORMATION OF RAT CELLS BY DNA OF HUMAN ADENOVIRUS 5.

(E.) Graham, F. L. (Lab. Physiological Chem., State U. Leiden, The Netherlands) and A. J. Van Der Eb. *Virology* 54(2):536-539, 1973.

Primary rat embryo and baby rat kidney cells were transformed by human adenovirus 5 DNA (Ad 5 DNA). Approximately two to three wk after exposure of rat embryo cells to Ad 5 DNA, foci of small, densely packed, round or cuboidal cells were observed over a background of normal fibroblasts. In baby rat kidney cultures, however, foci could often be observed within as little as 10 to 12 days after exposure to DNA, and after 3 wk many colonies were as large as 2-3 mm. As in the case of the rat embryo cells, these foci contained small, round, densely packed cells characteristic of adenovirus transformation. From morphological and immunological evidence it could be shown that the transformation in DNA treated cultures was adenovirus specific. Transforming activity was resistant to heating (1 hr at 56 C), and to pronase, but was sensitive to DNase. The efficiency of transformation was approximately 1 transformed focus/μg DNA.

3148 CLONAL VARIABILITY OF ADENOVIRUS-TRANSFORMED CELLS.

(E.) Weber, J. (Central U. Hosp., Sherbrooke, Quebec, Canada). *J Cell Sci* 13(2): 421-427, 1973.

Cells transformed by DNA oncogenic viruses acquire characteristic properties which have been considered criteria for transformation. SV40- and polyoma virus-transformed cells are capable of selective reversion for several of these properties. Until now, reversion of transformed cell properties has not been reported in the adenovirus system. The stability of several transformed cell properties was examined in a line (HTC) of Ad. 12-transformed (*in vivo*) cells after several hundred generations of *in vitro* passage. From a large number of clones isolated by the soft-agar suspension technique, 7 were selected for detailed examination. The clones, as the parental cells, were free of infectious virus and type-specific antigen, but contained intranuclear Ad. 12 T antigen. Cell growth profiles with 0, 1 and 10% serum in the culture medium revealed that while 4 clones retained the parental characteristic of growth in the absence of serum, 3 lost this ability and grew only in serum concentrations greater than 1%. Agglutination profiles with 4-fold dilutions of concanavalin A (Con A) and wheat germ agglutinin (WGA) directly or after trypsin treatment revealed 2 clones which failed to agglutinate with Con A; trypsin treatment reversed this effect. Each clone agglutinated with WGA directly, though one clone showed reduced agglutination. On the basis of superinfection with Ad. 2 the clones could be grouped into low, intermediate and high yielders with 10-fold intervals. All clones were transplantable into random-bred adult Syrian hamsters. There was no marked correlation between the reversion or modification of individual transformed cell properties among the clones. The phenotypic stability of these transformed cell properties was confirmed bv re-cloning the tumors induced by one of the revertant clones. It was concluded that some of these properties are not essential for the maintenance of oncogenic potential.

3149 TRANSFORMATION OF RAUSCHER VIRUS-INFECTED CELL CULTURES AFTER TREATMENT WITH HYCANTHONE AND LUCANTHONE. *(E.)* Hetrick, F. M. (Department Microbiol., U. Maryland, College Park) and W. L. Kos. *J Pharmacol Exp Ther* 186(3):425-429, 1973.

Rat embryo cell cultures (F 1706 line) chronically infected with Rauscher murine leukemia virus were transformed morphologically after treatment with low levels of the antischistosomal drugs hycanthone and lucanthone. Neither infection with the virus alone or treatment with the chemical alone resulted in the transformation. The foci of transformed cells appeared on the first subcultivation after chemical treatment and became the predominant cell as the cultures were passaged. In addition to their morphological change, the transformed cells no longer exhibited contact inhibition and they produced tumors within three to five weeks after implantation into newborn rats. The predictive value of this cotransformation system has not yet been firmly established but of the chemicals tested to date there is a good correlation between their ability to transform the virus-infected cells and their ability to produce tumors in animals. The fact that hycanthone behaves similarly to known carcinogens in this cell culture system is particularly troubling because it is being administered on a large scale to humans in various countries to control schistosomiasis.

3150 PILOT EXPERIMENTS WITH EB VIRUS IN OWL MONKEYS (*AOTUS TRIVIRGATUS*). I. RETICULOPROLIFERATIVE DISEASE IN AN INOCULATED ANIMAL. *(E.)* Epstein, M. A. (U. Bristol Med. Sch., England), R. D. Hunt and H. Rabin. *Int J Cancer* 12(2):309-318, 1973.

Epstein-Barr (EB) virus-containing cultured lymphoblasts were inoculated i.p. into three adult owl monkeys in a dose of 1.5 ml (6×10^7 cells/animal). Virion-free cells were inoculated into three other monkeys, and four further animals were kept as uninoculated controls. One of the animals receiving the virus died after 14 wk with a striking reticuloproliferative disease very compatible with certain forms of malignant lymphoma. The findings included extensive cellular infiltration in the liver and kidney, small collections of lymphocytes in the myocardium and meninges, and a large mass invading the wall of the cervix. A unique feature of the cellular infiltrate and proliferation, especially in lymph nodes, was the heterogeneity of cell type. The presence of large numbers of plasma cells, eosinophils, and large reticulum cells is not usual in spontaneous lymphoproliferations and lymphomas of man or lower animals. On the other hand, an essentially identical histologic pattern is occasionally seen in *Herpes saimiri* malignant lymphoma in owl monkeys and marmosets, and a mixed cell population is typical of Hodgkin's disease and some malignant lymphomas of mice. The results point to a possible oncogenic capacity for EB virus.

3151 EARLY RESPONSE OF LYMPH NODE CELLS TO ABELSON LEUKEMIA VIRUS. *(E.)* Siegler, R. (Children's Cancer Res. Fdn., Boston, Mass.), I. Lane, Y. Frosch and S. Moran. *Lab Invest* 29(3): 273-277, 1973.

The early ultrastructural responses of cervical and auxillary lymph node cells of outbred CD-1 mice infected with the murine C-type Abelson leukemia virus were studied. Virions could be seen budding from lymphoblasts, reticulum cells, capillary endothelial cells, and after their appearance, Abelson tumor cells. Between 10-20 days after infection, before proliferating tumor cells could be found in the nodes, most of the cortical reticulum cells underwent morphologic changes with the appearance of numerous microvilli and ruffles on their membrane surfaces. Many of these microvilli coalesced at the cell surface to form vacuoles which contained adherent C-type particles. Within the cytoplasm, these virion-containing vacuoles often joined to and fused with lysosomes, resulting in progressive destruction of the virions. Some cells indistinguishable from tumor cells, as well as an occasional lymph node cell from control mice, also showed these changes. On the basis of these observations, it is speculated that the leukemia in these infected mice resulted from an unsuccessful vigorous host effort to limit the spread of virus.

3152 THE FRAGMENTATION OF CELLULAR DNA AND THE FORMATION OF PSEUDOVIRIONS DURING SV40 INFECTION OF AFRICAN GREEN MONKEY KIDNEY CELLS. *(E.)* Ritzi, E. M. (Dept. Biochem. Sci., Princeton U., N.J.) and A. J. Levine. *J Gen Virol* 20(3): 353-367, 1973.

Infection of African green monkey kidney cells (AGMK) with simian virus 40 (SV40) results in a fragmentation of cellular DNA to a small molecular wt form and the production of pseudovirions. The small molecular wt cellular DNA was first detectable at about 45 to 60 hr after infection and continued to increase in amount up to 105 hr. Pseudovirions were preferentially formed at very late times also (60 to 96 hr after infection). Type 2 adenovirus does not cause cellular DNA fragmentation in infected AGMK cells and even prevents SV40 breakdown of cellular DNA in co-infected cells. DNA and protein synthesis were both required for the conversion of high molecular wt cellular DNA to the smaller molecular wt form. DNA replication was required up to 30 to 40 hr after SV40 infection, while protein synthesis was required up to 48 to 60 hr after infection for the efficient fragmentation of cellular DNA. These very late events could be due to the necessity to accumulate (with time) some virus or cellular gene products which only act efficiently at high concentrations. Alternatively, some specific gene product (virus or cellular) might be synthesized (prior to 50 hr) at late times in an infected cell.

Finally, it remains possible that the large molecular wt DNA is packaged into pseudovirions and the small molecular wt DNA fraction detected in these experiments is a by-product or abortive product of this packaging process. Extensive single-stranded breaks were detected in some 60 to 75% of the cellular DNA late in SV40 infection. Under these conditions some 20 to 45% of the double stranded cellular DNA was present in a low molecular wt form. Preferential levels of highly repetitive sequences were not detected in the fragmented cellular DNA. It appears that this DNA represents a random collection of cellular DNA molecules with respect to the frequency of repetitive DNA sequences.

3153 PILOT EXPERIMENTS WITH EB VIRUS IN OWL MONKEYS (*AOTUS TRIVIRGATUS*). II. EB VIRUS IN A CELL LINE FROM AN ANIMAL WITH RETICULOPROLIFERATIVE DISEASE.

(E.) Epstein, M. A. (U. Bristol Med. Sch., England), H. Rabin, G. Ball, A. B. Rickinson, J. Jarvis and L. V. Melendez. *Int J Cancer* 12(2): 319-332, 1973.

A continuous cell line has been established in culture from a pathological lymph node of an owl monkey with reticuloproliferative disease after inoculation with Epstein-Barr (EB) virus. The cells grow in suspension in clumps or as single individuals and show the characteristic features of lymphoblasts with some differentiation to reticulum cells; their karyotype is typical of one sub-species of owl monkey. A herpes virus found in the cells has been identified as EB virus in two types of immunofluorescence test using two different specific antisera; this identification was confirmed by the ability of the virus to transform normal human cord-blood lymphocytes and make them grow as a continuous cell line. Infection by a carcinogenic simian virus, *Herpesvirus saimiri*, has been excluded by tests for neutralizing antibodies and virus growth in susceptible cells. The continuous growth of the monkey cells, the presence in them of EB virus, and the possible relationship of the virus to the original reticuloproliferative disease of the inoculated monkey are considered and discussed.

3154 INCOMPLETE VIRUS PARTICLES OF ADENOVIRUS TYPE 16.

(E.) Wadell, G. (Karolinska Inst. Sch. Med., Stockholm, Sweden), M-L. Hammarskjöld and T. Varsanyi. *J Gen Virol* 20(3):287-302, 1973.

Hemagglutinating populations of virus particles of adenovirus type 16 (I-VIII) were separated by isopycnic centrifuging in CsCL. One of these populations (V) contained complete virus particles. Fresh preparations of virus particles from all populations studied had a morphology indistinguishable from that of complete virus particles. The five lightest populations were shown to contain [^{3}H]-thymidine and to be infective; dose-response relationships indicated the occurrence of a multiplicity-dependent infectivity of the incomplete virus particles. SDS-polyacrylamide gel electrophoresis of incomplete virus particles of the lightest population demonstrated that they were deficient in three polypeptides, but contained three other polypeptides which were not detectable in complete particles. One characteristic polypeptide of incomplete particles and another characteristic polypeptide of complete particles displayed high ratios of arginine over threonine. Although no experimental evidence has indicated a precursor-product relationship between polypeptides of incomplete virus and those assembled into intact particles, the occurrence of different polypeptides in the four incomplete virus particle populations could correspond to precursor stages during development of an intact particle.

3155 INTERFERON MEDIATES ENHANCEMENT OF TUMOUR GROWTH AND VIRUS-INDUCED SARCOMAS IN MICE.

(E.) Gazdar, A. F. (Natl. Cancer Inst., Bethesda, Md.), H. Sims, G. J. Spahn and S. Baron. *Nature [New Biol]* 245(142):77-78, 1973.

Although interferon and its inducers have been reported to inhibit the growth of tumors *in vivo* or of tumorigenic and diploid embryonic cells *in vitro*, interferon inducers have also been reported to accelerate the growth of transplanted and virus-induced tumors and to enhance spontaneous and virus-induced transformation *in vitro*, particularly when there is pretreatment with the inducers. In the present work, weanling Balb/c mice and suckling Osborne-Mendel rats were used. The interferon used was prepared from Newcastle disease virus-infected Moloney sarcoma virus transformed S+L- cells. Pretreatment of mice with a single dose of interferon or poly (I) poly (C) 6-24 hr before infection with MSV enhanced tumor size and incidence. Simultaneous administration of virus and interferon or poly (I) poly (C) gave more variable and less significant results, while administration of interferon or poly (I) poly (C) 24 hr after virus had no significant effect. The tumor enhancing effect of both substances was strongly dose-dependent. Pretreatment of rats with interferon had no effect on tumor induction while poly (I) poly (C) pretreatment had a moderate effect. It was suggested that the paradoxical effects of interferon and its inducers on tumor growth may be associated with their effects on the immune system. Thus, under certain circumstances, interferon and poly (I) poly (C) act as immunological adjuvants, while pretreatment may depress immunological responsiveness.

3156 GROWTH AND ANTIGENIC CHARACTERISTICS OF SUBCLONES OF HAMSTER CELLS TRANSFORMED BY THE SCHMIDT-RUPPIN STRAIN OF ROUS SARCOMA VIRUS.

(E.) Bataillon, G. (Radium Inst., Fac. Sci., Orsay, France). *J Gen Virol* 20(2):257-260, 1973.

Four subclones of hamster cells transformed by Schmidt-Ruppin strain of Rous sarcoma virus (SR-RSV) were compared with respect to growth and antigenic properties. A positive correlation was found between the growth potential of the cells in soft agarose containing dextran sulphate, and the degree to which they expressed the virus-induced surface antigen (VISA), as determined by an adaptation of the colony-inhibition test. This is contrary to most observations made *in vivo* with RSV-induced tumor-specific transplantation antigen (TSTA). The apparent discrepancy may result from the use in other laboratories of more heterogeneous material. Another explanation could be that RSV-VISA, as detected *in vitro*, is not identical with RSV-TSTA studied in isograft rejection experiments and that antisera prepared by different investigators may show slightly different antigenic determinants of group-specific antigen or VISA complexes.

3157 MUTANTS OF NONPRODUCER CELL LINES TRANSFORMED BY MURINE SARCOMA VIRUS. II. RELATIONSHIP OF TUMORIGENICITY TO PRESENCE OF VIRAL MARKERS AND RESCUABLE SARCOMA GENOME.

(E.) Hatanaka, M. (Flow Lab., Inc., Rockville, Md.), R. Klein, C. W. Long and R. Gilden. *J Exp Med* 138(2):364-372, 1973.

Tumorigenic and nontumorigenic cell mutants induced by a single 5'-bromodeoxyuridine (BrdU) treatment of a nonproducer (NP) tumorigenic cell line were isolated by cloning and characterized. Among the cloned derivatives were examples of virus-free and sarcoma virus-producing cell lines. Oncogenicity did not correlate with production of virus or ease of rescue of the sarcoma genome. All lines, including nononcogenic derivatives, retained the sarcoma genome. Phenotypic reversion of some cell mutants was observed after *in vivo* inoculation or long term *in vitro* cultivation. The M-50T cell line, obtained from a tumor induced by M-50 mutant cells, had a sarcoma genome rescuable by direct superinfection; this was only achieved with parental M-50 cells by a cell fusion rescue technique. The M-43-2T cell, obtained from a single small static tumor induced by otherwise nononcogenic M-43-2 mutant cells, shed sarcoma virus and became tumorigenic. M-58-4-48 became tumorigenic after passage 48 of the M-58-4 mutant line, which was originally nontumorigenic. These observations of phenotypic reversion demonstrate that the presence of the sarcoma gene in cells is an essential but not sufficient condition of tumorigenesis.

3158 ISOLATION OF TYPE C ONCORNA VIRUS FROM CATTLE WITH SPONTANEOUS AND EXPERIMENTALLY INDUCED LYMPHOID LEUKEMIA.

(Rus.) Kukain, R. A. (August Kirchenstein Inst. Microbiol., Riga, USSR), L. I. Nagaeva, S. V. Chapenko, O. I. Bratsslavskaia, M. A. Aleksandrova, E. M. Ustinkova, A. Ia. Runtsis, G. V. Kudeleva, T. F. Shitova and S. S. Chashina. *Vopr Onkol* 19(7):44-49, 1973.

A study was made of morphological and physicochemical properties of virus particles found in the lymph nodes, cultures of peripheral WBC, and plasma from Latvian brown cattle with chronic leukemia or leukemia induced by inoculation of newborn calf WBC and cell-free homogenates from cattle with lymphoid leukemia. Electron microscope examination of ultrathin sections of lymph nodes from animals with both spontaneous and induced leukemia revealed the presence of virus particles measuring 900-1200 Å with an electron-dense nucleoid measuring 600 Å. Most viral particles were found inside the cells, near the cell membrane. Large masses of virus particles measuring 1000-1200 Å with electron-dense nucleoids measuring 600-800 Å were detected intracellularly in 72-hr cultures of WBC from cattle with spontaneous lymphoid leukemia; morphologically they can be classified as type C oncorna viruses. Many free type C virus particles were also found in 72-hr WBC cultures from calves with experimental leukemia. Virus particles were isolated in a peak at 1.16 g/ml from plasma by ultracentrifugation in a linear potassium citrate gradient. This peak, which is characteristic for oncorna viruses, was found in plasma from animals with spontaneous and induced leukemia. The reverse transcription reaction showed the presence of a relatively high revertase activity which was linear for 1 hr.

3159 SPONTANEOUS REGRESSION OF LEUKEMIA IN CHICKENS INFECTED WITH AVIAN MYELOBLASTOSIS VIRUS.

(E.) Silva, R. F. (VA Hosp., Gainsville, Fla.) and C. Moscovici. *Proc Soc Exp Biol Med* 143(3):604-611, 1973.

The spontaneous regression of avian myeloblastosis virus (AMV) induced leukemia was observed in two chicken lines. When the virus was injected intravenously into 17 day old Kimber embryos, 15% of the resultant leukemias regressed spontaneously. If a relatively resistant chicken line (C/AB) was used, 62% of the leukemias regressed. Nearly one-third of the regressed chickens experienced a fatal relapse. Over 30% of the bone marrow in both regressed Kimber and C/AB chickens consisted of abnormal blast-like cells. Nearly 40% of the regressed birds evidenced lymphoma-like tumors upon autopsy. Kimber chickens were viremic during both the acute phase and regression, while infectious AMV could not be recovered from the C/AB at any time. Buffy coat cultures from regressed Kimber and C/AB always resulted in the reappearance and proliferation of myeloblasts. Splenic lymphocytes from regressed Kimber chickens were found to inhibit colony formation of leukemic myeloblasts in semisolid medium. Evidence indicates that a host function is responsible for the spontaneous regression. A good explanation is not yet available for the reappearance of myeloblasts in buffy coat cultures obtained from regressed chickens.

3160 EB VIRAL GENOMES IN EPITHELIAL NASOPHARYNGEAL CARCINOMA CELLS.

(E.) Wolf, H. (Inst. Clin. Virology, Erlangen, Germany), H. Zur Hausen and V. Becker. *Nature* [*New Biol*] 244(138):245-247, 1973.

Although the EB viral DNA present in Burkitt lymphomas occurs within the tumor cells, it has been obscure as to whether the EB viral DNA present in nasopharyngeal tumors occurs within the epithelial tumor cells or within the infiltrating lymphocytes. To answer this question, DNA of frozen sections of 28 nasopharyngeal tumors was annealed with EBV-specific complementary RNA (cRNA). Most of the nasopharyngeal carcinoma specimens hybridized with EBV-cRNA at levels clearly above those of control DNA preparations from other carcinomas, chronic lymphatic leukemias and healthy lymphocytes. Those nasopharyngeal tumors which annealed in the highest range were predomonantly epithelial when examined histologically, while low hybridizing preparations consisted mainly of lymphatic cells or paratumorous tissue. Autoradiographic study of hybridized preparations of nasopharyngeal carcinoma tissues and of control preparations of an adenoma and salivary glands showed clustering of label and accumulation of grains over nuclei for the former, none for the latter. The arrangement of labeled clusters of nuclei in sections of nasopharyngeal carcinoma corresponded to similar clusters of epithelial cells in non-denatured sections of the same tumor, while nuclei of lymphocytes seemed to be unlabeled. Finally, tests for EBV-specific complement-fixing antigen-antibody complexes by anticomplement immunofluorescence were applied to frozen sections of biopsy material from a nasopharyngeal carcinoma and three other tumors, with cells of the Raji line from a Burkitt lymphoma serving as a positive control. No anticomplement fluorescence was observed in the control tumors, whereas Raji cells and apparently epithelial cells of the nasopharyngeal carcinoma revealed a clearly positive reaction. In Raji cells this fluorescence was mainly confined to the nuclear regions, whereas in the nasopharyngeal carcinoma cells it appeared to be more granular and was also observed in the cytoplasm. The data thus provide evidence that the epithelial cells of nasopharyngeal carcinomas harbor the EB viral genomes which were demonstrated by nucleic acid hybridizations. This tumor thus represents the first example of an infection of human non-lymphatic cells with EBV. The presence of EBV DNA within the epithelial cells substantially supports the circumstantial evidence for a causal role of EBV in this malignancy.

3161 DETERMINANTS OF THE HOST RANGE OF FELINE LEUKAEMIA VIRUSES. *(E.)*

Jarrett, O. (U. Glasgow, Leukaemia Res. Unit, Scotland), H. M. Laird and D. Hay. *J Gen Virol* 20(2):169-175, 1973.

The host range of feline leukemia virus (FeLV) was examined as well as the factors necessary for FeLV isolates to initiate successful infections in human and canine cells. FeLV of subgroups B and C multiply in human and canine cells, while subgroup A viruses do not. This host range restriction is determined by the virus envelope and operates at the level of virus entry into the cell. Subgroup A virus genomes are expressed and replicated when they are introduced within B subgroup envelopes into human or dog cells. Therefore, since they are phenotypic mixtures of A and B subgroup viruses, the majority of feline leukemia virus isolates will infect human and canine cells with the subsequent production of FeLV of each subgroup.

3162 AGGLUTINATION WITH CON A DEPENDENT ON CELL CYCLE. *(E.)*

Smets, L. A. (Netherlands Cancer Inst., Amsterdam). *Nature (New Biol)* 245(143):113-115, 1973.

The assumption that the membrane of transformed cells does not permanently display the mitotic configuration as suggested by some workers but rather is modulated during the cell cycle so that in G2 phase the transformed cell has a similar surface architecture to the normal cell during interphase was examined and confirmed in long-term cultures of human lymphocytes transformed by Epstein-Barr virus. Agglutination with con A was studied for cultures synchronized in various parts of the cell cycle. The results indicate that agglutinability decreases when cells progress through the cycle from G1 to G2 phase but rises abruptly at the transition from late G2 to metaphase. High agglutinability lasts for at least the whole G1 period and may decrease abruptly when cells enter S phase. The results confirm that, in an established line of human lymphoblasts, the postmitotic modulation of the mitotic membrane occurs much later in the cell cycle than in untransformed cells.

3163 MURINE MAMMARY TUMOR CELL CLONES WITH VARYING DEGREES OF VIRUS EXPRESSION. *(E.)*

Parks, W. P. (Natl. Cancer Inst., Bethesda, Md.) and E. M. Scolnick. *Virology* 55(1):163-173, 1973.

Viral expression by murine mammary tumor virus (MTV) may be detected by electron microscopy, tumor formation, antigen induction or nucleic acid hybridization. Using some of these methods a comparison was made of two single cell clones derived from Sykes' CCL-51, a type-B particle positive, type-C virus negative murine mammary tumor cell line. The clones had similar growth properties—they formed "dome-like" structures, grew in soft agar, and had abundant microvilli by electron microscopy—but differed markedly in their expression of type-B viral information. One clone, clone 11, had morphological type-B particles, type-B specific s1 antigen (3500 ng/mg protein), and cellular RNA which hybridized with MTV[^{3}H]DNA product. The other clone, clone 6, did not have detectable particles by electron microscopy, antigen levels were below the limits of sensitivity or barely detectable (~30 ng/mg protein), and RNA hybridizable to MTV DNA product was less than 5% of the reaction noted with the positive clone. Measurement of the cellular MTV-related DNA sequences by DNA-DNA hybridization using the same single-stranded MTV DNA product revealed comparable levels of MTV DNA sequences in both clones. Type-C viral expression (gs antigen and RNA levels) in both clones was comparable and not significantly higher than noted in other "normal" murine cell lines. The findings thus showed that there was a correlation between levels of viral antigen and hybridizable total cellular or polysomal RNA in these cells and that clones which contained equivalent amounts of MTV-related cellular DNA sequences had markedly different levels of hybridizable RNA, antigen, and virus particles. The results suggest that the mechanism of viral control in these cells is at the level of DNA to RNA transcription and is not necessarily dependent on presumable differentiated characteristics of mammary cells such as "doming".

3164 ANALYSIS OF MINIMAL FUNCTIONS OF SIMIAN VIRUS 40. I. ONCOGENIC TRANSFORMATION OF SYRIAN HAMSTER KIDNEY CELLS *IN VITRO* BY PHOTODYNAMICALLY INACTIVATED SV40. *(E.)*

Seemayer, N. H. (Wistar Inst., Philadelphia, Pa.), K. Hirai and V. Defendi. *Int J Cancer* 12(2):524-531, 1973.

The viral DNA of SV40 is sufficient to code for 6-8 enzymes or proteins, but it is not known which of these functions is essential for oncogenicity.

One approach to genetic analysis of viral functions is the method of serial inactivation of the virus. In the present work, serial inactivation of SV40 was accomplished by irradiation with white light in the presence of Toluidine blue O. Similar studies had previously been carried out using inactivation by UV. It was found that different functions of SV40 were inactivated at different rates. The most sensitive function was the capacity of the virus to replicate followed by the capacity to induce T antigen and cell DNA synthesis. The transformation capacity was the most resistant. No evidence was obtained that the survival of trasformation activity after large decrease in infectivity was due to multiplicity reactivation. Syrain hamster kidney cells transformed *in vitro* by photodynamically inactivated SV40 had the same properties as those transformed by control virus. They showed identical morphology, were T-antigen positive, contained the same number of SV40 DNA genome equivalents integreated in their DNA and were oncogenic when inoculated into adult hamsters.

165 DISCONTINUOUS ELONGATION OF BOTH STRANDS AT THE REPLICATION FORKS IN POLYOMA DNA EPLICATION.

(E.) Pigiet, V. (Dept. Biochem., arolinska Inst., Stockholm, Sweden), E. L. Winnacker, . Eliasson and P. Reichard. *Nature* [*New Biol*] 45(146):203-205, 1973.

he mechanism of replication of the double-stranded ircular DNA of polyoma virus was studied *in vitro*. S replicative DNA intermediates were density abeled with bromodeoxyuridine triphosphate and urified by chromatography on benzoylated naphthoy-ated cellulose followed by treatment with NaOH to emove RNA. The labeled daughter strands were hen separated according to size by alkaline su-rose and alkaline Cs_2SO_4 equilibrium density cen-rifugation and the capacity of the long and short ragments to self-anneal was then investigated. etween 1.5 and 5.0 min after labeling, DNA frag-ents banding in light density positions shifted o higher densities, a finding consistent with he interpretation that heavier 4S fragments formed nitially are subsequently added on to preexisting ight strands. Self-annealing experiments showed high degree of complementarity between the short eplicative intermediates, strongly suggesting hat polyoma DNA replication involves discontinu-us synthesis of both daughter strands at the re-lication forks.

66 SENSITIVITY TO EBV SUPERINFECTION AND IUdR INDUCIBILITY OF HYBRID CELLS FORMED BETWEEN SENSITIVE AND A RELATIVELY RESISTANT BURKITT LYMPH-MA CELL LINE.

(E.) Nyormoi, O. (Dept. Zoology, Indi-na U., Bloomington), G. Klein, A. Adams and L. Dombos. *t J Cancer* 12(2):396-408, 1973.

he sensitivity of Burkitt lymphoma-derived cell lines Epstein-Barr virus (EBV) superinfection and the nducibility of the latent EBV genome in these lines ere studied by somatic cell hybridization. Two non-BV producer lines -- AG R3, an adherent, 8-azaguanine-esistant variant of the Raji line, and Namalwa, a nonadherent, 8-azaguanine-sensitive line -- were fused with the aid of β-propiolactone-inactivated Sendai virus. The resident EBV genome in Raji is inducible with 5-iododeoxyuridine (IUdR) and the line is also sensitive to EBV superinfection. Namalwa is relatively resistant to both superinfection and induction, and synthetizes surface-associated IgM-lambda. Cytological studies showed that hybrid cells are much larger than either parent and attach to culture plates less firmly than the adherent Raji variant parent. Karyological analyses showed that hybrids contain the expected sum of the parental chromosome markers. Membrane immunofluorescence tests also showed that hybrids synthetize IgM. All the hybrid cells appear to be non-EBV producers, but they are sensitive to both EBV superinfection and induction of latent EBV. These findings suggest the following explanations: (1) there is no evidence of any complementation between the two non-EBV producer lines (Raji and Namalwa) to elicit spontaneous EBV production in hybrid cells; (2) Namalwa is deficient in some factors required for the synthesis of EBV-specified early antigens after EBV superinfection and after induction of latent EBV by IUdR; these factors are supplied by the Raji parent in the hybrids; or (3) Raji, Namalwa, and hybrid cells or EBV all produce a substance which inhibits activation of a productive viral cycle, but whose action is antagonized in Raji and hybrid cells to allow the synthesis of EBV-specific early antigens.

3167 VIRUS MORPHOGENESIS IN A MOUSE LYMPHOMA.

(E.) Petrea, I. (Yale U. Sch. Med., New Haven, Conn.) and W. U. Gardner. *Cancer Res* 33(10): 2291-2299, 1973.

A primary pituitary tumor (PPT) developed in one of four isologous pituitary glands that were transplanted s.c. in a hybrid mouse (C57 X BALB/c). The mammary glands of the host were thick and lactating; its spleen and liver were enlarged; and the lower uterine horns and the cervix were also enlarged, firm, and fibrous. The PPT was transplanted s.c. in isologous hosts, six female and six male, and 170 to 190 days later, five female and two male hosts had transplanted pituitary tumors and five female and six male hosts had ascites and generalized lymphoma. In the subsequent transfer generations, the mice bearing transplants survived only 4 wk or less. The PPT grew at the site of transplantation and also involved remote nodes and the liver and the spleen. The tumor after the first transfer contained medium and large lymphoblastic cells with multiple nucleoli and mitotic figures. Examination by electron microscopy revealed virus-like particles in juxtaposition to chromatin and within the rough endoplasmic reticulum (ER) surrounding the nucleus. Morphogenically, the ribosomes successively aggregated and fused on the ER membrane to form long and short osmiophilic bars that bulged into the ER cisternae and then separated to form viral particles (VP's) of the C-type (oncornaor leukoviruses) measuring about 100 nm in diameter. VP's were not found outside the ER. The distribution of RNA and DNA in the nuclei and cytoplasm of the lymphoma cells was revealed by the acriflavine-phosphotungstate technique. The provirus bars and the VP's were electron opaque after RNase treatment and slightly less dense after DNase treatment. Only the protein capsids remained after incubation with both DNase and RNase.

3168 E. B. VIRUS AND AMERICAN AND AFRICAN BURKITT'S LYMPHOMA. *(E.)* Levine, P. H. (Natl. Inst. Hlth., Bethesda, Md.) and G. O'Conor. *Lancet* (7833):850, 1973.

In a letter to the editor, the authors refute the implication that Epstein-Barr (EB) virus is apparently not an etiologic agent of American Burkitt's lymphoma as it seems to be for African Burkitt's lymphoma. They point out that most of the serologic studies showing a poor correlation between the presence of antibodies to EB virus and Burkitt's lymphoma have used small series of patients, thus increasing the likelihood of experimental bias. The author's studies, which have used relatively large series (73 Burkitt's lymphoma patients), have shown a significantly higher EB virus antibody titer in sera from American Burkitt's lymphoma patients compared with leukemic and normal age-matched controls. Thus, the importance of large numbers of patients is stressed. Although the authors agree that it is still premature to assume that EB virus is the etiologic agent of Burkitt's lymphoma in any country, they believe it is also premature to state that EB virus does not have this role in America.

3169 FREE REVERSE TRANSCRIPTASE IN THE PLASMA OF LEUKEMIC MICE AND CHICKENS. *(Fr.)* Burny, A. (Inst. Cancer Res., Columbia U., New York, N.Y.), A. Yaniv and S. Spiegelman. *Arch Int Physiol Biochim* 81(2):362-363, 1973.

Leukemia was induced in BALB/c mice by injection of Rauscher leukemia virus (RLV). The activity of DNA polymerase increased linearly in the plasma of injected mice for about 3 wk and then reached a plateau. This DNA polymerase activity was contained entirely in a fraction precipitated with 30% saturated ammonium sulfate and centrifuged in a glycerol gradient; the enzyme had an apparent molecular weight of 68,000. Purified ^{3}H-labeled DNA was specifically hybridized to the RNA used as a model. Thus, the enzyme catalyzing DNA synthesis is a reverse transcriptase. It sediments in the same way as reverse transcriptase from solubilized RLV. Plasma from chickens injected with avian myeloblastosis virus also contained reverse transcriptase. The enzyme sedimented in a gradient in the same way as viral reverse transcriptase and could not be differentiated from it immunologically. Preliminary studies indicate that reverse transcriptase was also present in the plasma of patients with leukemia. It is suggested that radioimmunoassay of this enzyme or enzymes would be useful in the diagnosis of some cancers.

3170 PHOSPHORYLATION OF SIMIAN VIRUS 40 PROTEINS IN A CELL-FREE SYSTEM. *(E.)* Tan, K. B. (Wistar Inst. Anatomy, Biol., Philadelphia, Pa.) and F. Sokol. *J Virol* 12(4):696-703, 1973.

3171 TRANSFER OF THE B77 VIRUS GENOME FROM VIROGENIC MAMMALIAN CELLS TO OTHER MAMMALIAN CELLS BY CELL FUSION. *(E.)* Altanar, C. (Cancer Res. Inst., Slovak Acad. Sci., Bratislava, Czechoslovakia), A. Lizonova and M. Hladka. *Neoplasma* 20(2):125-132, 1973.

3172 EVIDENCE FOR DIFFERENT GLYCERALDEHYDE-3-PHOSPHATE DEHYDROGENASES IN WI-38 AND SV40-TRANSFORMED WI-38. *(E.)* Dunaway, G. A. Jr. (Dept. Chem., U. Oklahoma, Norman) and E. C. Smith. *Int J Biochem* 4(21):245-247, 1973.

3173 COMBINED IMMUNOTHERAPY AND CHEMOTHERAPY OF MOLONEY SARCOMA VIRUS-INDUCED TUMORS IN MICE. *(E.)* Giuliani, F. (Natl. Inst. Study Cure Tumors, Milan, Italy), A. M. Casazza and A. Di Marco. *Biomedicine* 18(5):387-392, 1973.

3174 AUTORADIOGRAPHY OF THE TISSUES OF CHICKS INFECTED WITH MAREK'S DISEASE VIRUS USING ^{3}H-THYMIDINE. *(E.)* Iwa, N. (Res. Fdn. Microbial Diseases, Osaka U., Japan), K. Ono, M. Naito and S. Kato. *Biken J* 16(2):85-90, 1973.

3175 CULTIVATION OF GANGLIA ON MONOFIL FABRIC, A SUITABLE METHOD FOR DEMONSTRATION OF LATENT HERPESVIRUS INFECTION. *(E.)* Benda, R. (Military Inst. Hygiene, Epidemiology, Microbiol., Prague, Czechoslovakia), J. Cinatl, S. Petrovic, J. Roubal and V. Plaisner. *Acta Virol* 17(4):305-309, 1973.

3176 SYNTHESIS OF PROTEINS IN CELLS INFECTED WITH HERPESVIRUS. IX. SULFATED PROTEINS. *(E.)* Erickson, J. S. (Vanderbilt U. Sch. Med., Nashville, Tenn.) and A. S. Kaplan. *Virology* 55(1):94-102, 1973.

3177 EFFECT OF 2-DEOXY-D-GLUCOSE ON CELL FUSION INDUCED BY NEWCASTLE DISEASE AND HERPES SIMPLEX VIRUSES. *(E.)* Gallaher, W. R. (Presbyterian-U. Pennsylvania Med. Ctr., Philadelphia), D. B. Levitan and H. A. Blough. *Virology* 55(1):193-201, 1973.

3178 FRIEND LEUKEMIA VIRUS (FLV) ACTIVITY IN CERTAIN ARTHROPODS. I. INTRODUCTION, PARAMETERS AND CONTROL MEASURES NECESSARY FOR EVALUATION OF EXTRINSIC INCUBATION POTENTIAL. *(E.)* Luecke, D. H. (Dept. Microbiol., U. North Dakota, Grand Forks), J. Rehacek and R. G. Fischer. *Neoplasma* 20(2):133-145, 1973.

3179 FRIEND LEUKEMIA VIRUS (FLV) ACTIVITY IN CERTAIN ARTHROPODS. II. QUANTITATIVE INFECTIVITY DETERMINATIONS. *(E.)* Rehacek, J. (Dept. Microbiol., U. North Dakota, Grand Forks), R. G. Fischer and D. H. Luecke. *Neoplasma* 20(2):147-158, 1973.

3180 RNA SYNTHESIS IN CELLS INFECTED WITH HERPES SIMPLEX VIRUS. VII. CONTROL OF TRANSCRIPTION AND OF TRANSCRIPT ABUNDANCIES OF UNIQUE AND COMMON SEQUENCES OF HERPES SIMPLEX VIRUS 1 AND 2. *(E.)* Frenkel, N. (Dept. Microbiol., U. Chicago, Ill.), S. Silverstein, E. Cassai and B. Roizman. *J Virol* 11(6):886-892, 1973.

3181 ELECTROPHORETIC DIFFERENCES IN THE CAPSID PROTEINS OF SIMIAN VIRUS 40 PLAQUE MUTANTS. *(E.)* Barban, S. (Natl. Inst. Hlth., Bethesda, Md.). *J Virol* 11(6):971-977, 1973.

3182 PARTICIPATION OF EARLY PROTEINS IN ADENOVIRUS 2 DNA TRANSCRIPTION. *(E.)* D'Halluin, J. C. (I.N.S.E.R.M. Virology Res. Unit., U. Lille, France), C. Chauvet, A. Sergeant and K. Krsmanovic. *Arch Int Physiol Biochim* 81(2):370, 1973.

3183 EB VIRUS AND CHILDHOOD DISEASES. *(E.)* Arai, M. (First Natl. Hosp. Tokyo, Japan), M. Nakamura, Y. Fukuda and M. Yamaguchi. *Asian Med J* 16(5):5-16, 1973.

3184 REASSORTMENT OF SIMIAN VIRUS 40 DNA DURING SERIAL UNDILUTED PASSAGE. *(E.)* Martin, M. A. (Natl. Inst. Hlth., Bethesda, Md.), L. D. Gelb, G. C. Fareed and J. B. Milstien. *J Virol* 12(4):748-757, 1973.

3185 INDUCTION OF CELLULAR DNA SYNTHESIS BY THE NONDEFECTIVE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID VIRUSES. *(E.)* Schnipper, L. E. (Natl. Cancer Inst., Bethesda, Md.), A. M. Lewis, Jr. and A. S. Levine. *J Virol* 12(4):940-943, 1973.

3186 STUDIES ON HERPES SIMPLEX VIRUS DNA: DENATURATION PROPERTIES. *(E.)* Gordin, M. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel), U. Olshevsky, H. S. Rosenkranz and Y. Becker. *Virology* 55(1):280-284, 1973.

3187 NONPRODUCER CLONES OF MURINE SARCOMA VIRUS TRANSFORMED GUINEA PIG EMBRYO CELLS. *(E.)* Rhim, J. S. (Natl. Cancer Inst., Bethesda, Md.), H. Y. Cho and F. G. Duh. *Virology* 54(2):547-551, 1973.

3188 FRIEND LEUKEMIA VIRUS (FLV) ACTIVITY IN CERTAIN ARTHROPODS. III. TRANSMISSION STUDIES. *(E.)* Fischer, R. G. (Dept. Microbiol., U. North Dakota, Grand Forks), D. H. Luecke and J. Rehacek. *Neoplasma* 20(3):255-260, 1973.

3189 BIOGENESIS OF POXVIRUSES: INACTIVATION OF HOST DNA POLYMERASE BY A COMPONENT OF THE INVADING INOCULUM PARTICLE. *(E.)* Pogo, B. G. T. (Public Health Res. Inst., New York City, N.Y.) and S. Dales *Proc Natl Acad Sci USA* 70(6):1726-1729, 1973.

3190 ANTITUMOR ACTIVITY OF SILICA GEL PF 254 ELUATE. *(E.)* De Clercq, E. (Rega Inst. Med. Res., U. Leuven, Belgium). *Cancer Res* 33(9): 2173-2180, 1973.

3191 *HERPESVIRUS SAIMIRI*: EXPERIMENTAL INFECTION OF SQUIRREL MONKEYS (*SAIMIRI SCIUREUS*). *(E.)* Falk, L. A. (Rush-Presbyterian-St. Luke's Hosp., Chicago, Ill.), L. G. Wolfe and F. Deinhardt. *J Natl Cancer Inst* 51(1):165-170, 1973.

3192 BURSA-DEPENDENT AND THYMUS-DEPENDENT "SURVEILLANCE" OF A VIRUS-INDUCED TUMOUR IN THE CHICKEN. *(E.)* Thompson, K. D. (Temple U. Sch. Med., Philadelphia, Pa.) and T. J. Linna. *Nature [New Biol]* 245(140):10-12, 1973.

3193 FUCOSYLGLYCOLIPID METABOLISM IN ONCORNAVIRUS-TRANSFORMED CELL LINES. *(E.)* Steiner, S. (Baylor Coll. Med., Houston, Tex.), P. J. Brennan and J. L. Melnick. *Nature [New Biol]* 245(140):19-21, 1973.

3194 FURTHER EVIDENCE FOR VIRAL ETIOLOGY OF HUMAN MELANOMA. *(E.)* Balda, B.-R. (Dept. Dermatology, Cell Biol., U. Munich, West Germany) and G. D. Birkmayer. *Naturwissenschaften* 60(6): 304-305, 1973.

3195 B-TROPIC FRIEND VIRUS: A HOST-RANGE PSEUDOTYPE OF SPLEEN FOCUS-FORMING VIRUS (SFFV). *(E.)* Lilly, F. (Albert Einstein Coll. Med., New York, N.Y.) and R. A. Steeves. *Virology* 55(2):363-370, 1973.

3196 5-BROMODEOXYURIDINE INCORPORATION INTO SIMIAN VIRUS 40 DEOXYRIBONUCLEIC ACID. EFFECTS ON SIMIAN VIRUS 40 REPLICATION IN MONKEY CELLS. *(E.)* Calothy, G. (Wistar Inst., Philadelphia, Pa.), K. Hirai and V. Defendi. *Virology* 55(2):329-338, 1973.

3197 STRUCTURE OF CRITICAL POINT DRIED ONCORNAVIRUSES. *(E.)* De Harven, E. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), D. Beju, D. P. Evenson and S. Basu. *Virology* 55(2):535-540, 1973.

3198 CHARACTERIZATION OF TWO SIMIAN VIRUS 40-SPECIFIC RNA MOLECULES FROM INFECTED BS-C-1 CELLS. *(E.)* Warnaar, S. O. (St. U., Leiden, Holland) and A. W. de Mol. *J Virol* 12(1):124-129, 1973.

3199 HYDROXYAPATITE CHROMATOGRAPHY AND FORMAMIDE DENATURATION OF ADENOVIRUS DNA. *(E.)* Tibbetts, C. (Wallenberg Lab., Uppsala U., Sweden), K. Johansson and L. Philipson. *J Virol* 12(2):218-225, 1973.

3200 REACTION OF MICE ON INFECTION WITH MYCOPLASMA AND LEUKOSIS VIRUSES. *(E.)* Kagan, G. Y. (Acad. Med. Sci. U.S.S.R., Moscow), Z. A. Postnikova, T. D. Morgunova and I. V. Rakovskaya. *J Hyg Epidemiol Microbiol Immunol (Praha)* 17(2):226-236, 1973.

3201 FINE STRUCTURE OF AN AMPHIBIAN LEUKOCYTE VIRUS. *(E.)* Briggs, R. T. (Dept. Physiology, Cell Biol., U. Kansas, Lawrence) and P. R. Burton. *J Submicrosc Cytol* 5(2):71-78, 1973.

3202 ALTERATIONS IN THE SURFACE OF GLYCOPROTEINS OF CHICKEN ERYTHYROCYTES FOLLOWING TRANSFORMATION WITH ERYTHROBLASTOSIS STRAIN R VIRUS. *(E.)* Barbarese, E. (Jewish Gen. Hosp., Montreal, Quebec, Canada), H. Sauerwein and H. Simpkins. *J Membr Biol* 13(2):129-142, 1973.

3203 TRANSFORMED RNA METHYLASES AND TRANSFER RNA-METHYLATED BASES OF POLYOMA VIRUS-TRANSFORMED AND SPONTANEOUSLY TRANSFORMED MOUSE CULTURE CELLS. *(E.)* Wright, S. E. (Natl. Cancer Inst., Bethesda, Md.), R. E. Gallagher, R. C. Ting and R. C. Gallo. *Cancer Res* 33(10):2513-2517, 1973.

3204 PRESENCE OF TWO "8 S" RNA COMPONENTS IN MOUSE SARCOMA VIRUS (MOLONEY). *(E.)* Larsen, C. J. (Saint-Louis Hosp., Paris, France), R. Emanoil-Ravicovitch, A. Samso, J. Robin, A. Tavitian and M. Boiron. *Virology* 54(2):552-556, 1973.

3205 THE EFFECT OF pH ON THE MORPHOLOGY, ELECTROPHORETIC MOBILITY, AND INFECTIVITY OF THE MOUSE MAMMARY TUMOR VIRUS. *(E.)* Sarkar, N. H. (Inst. Med. Res., Camden, N.J.), D. H. Moore and J. Charney. *Cancer Res* 33(10):2283-2290, 1973.

3206 INCORPORATION OF 2-DEOXY-D-GLUCOSE INTO GLYCOPROTEINS OF NORMAL AND SIMIAN VIRUS 40-TRANSFORMED HAMSTER CELLS. *(E.)* Steiner, S. (Baylor Coll. Med., Houston, Tex.), R. J. Courtney and J. L. Melnick. *Cancer Res* 33(10):2402-2407, 1973.

3207 THE EPSTEIN-BARR VIRUS (EBV) IN HUMAN PATHOLOGY. RESULTS OF A SEROLOGICAL INVESTIGATION OF A HOSPITAL POPULATION. *(E.)* Dalens, M. (U. Paul Sabatier, U.E.R. Med. Rangueil, Toulouse, France), J. Didier, J. Pris and L. Enjalbert. *Biomedicine* 18(4):336-345, 1973.

3208 INHIBITORY EFFECT OF STREPTONIGRIN ON A MURINE SARCOMA VIRUS-INDUCED TUMOR CELL LINE (MSC) AND SELECTION OF DRUG-RESISTANT CLONES. *(E.)* Woods, W. A. (Natl. Cancer Inst., Bethesda, Md.), J. G. Massicot, J. H. Webb and M. A. Chirigos. *In Vitro* 9(1):24-30, 1973.

3209 ELECTROPHORETIC SEPARATION OF ALL COMPONENTS OF THE DOUBLE-STRANDED RNA OF WOUND TUMOR VIRUS. *(E.)* Reddy, D. V. R. (Dept. Botany, U. Illinois, Urbana) and L. M. Black. *Virology* 54(2):557-562, 1973.

3210 DNA DAMAGE AND BIOLOGICAL FUNCTION OF HUMAN ADENOVIRUS AFTER U.V.-IRRADIATION. *(E.)* Rainbow, A. J. (Dept. Biol., McMaster U., Hamilton, Ontario, Canada) and S. Mak. *Int J Radiat Biol* 24(1):59-72, 1973.

3211 SIMIAN VIRUS 40-INDUCED RETINOPATHY IN THE RAT. *(E.)* Friedman, A. H. (Albert Einstein Coll. Med./Montefiore Hosp. Med. Ctr., New York, N.Y.), R. W. Bellhorn and P. Henkind. *Invest Ophthal* 12(8):591-594, 1973.

3212 EFFECT OF FRIEND VIRUS ON SERUM ALBUMIN SYNTHESIS IN MOUSE LIVER. *(E.)* Szafarz. D. (Curie Fdn., Radium Inst., Orsay, France), O. Gallien-Lartigue, D. Huaulme and F. Zajdela. *J Natl Cancer Inst* 51(2):465-472, 1973.

3213 SUSCEPTIBILITY OF GUINEA PIG CELL CULTURES TO INFECTION WITH CELL-BOUND AND CELL-FREE HERPESVIRUS OF TURKEYS. *(E.)* Bedigian, H. G. (Dept. Vet. Animal Sci., U. Massachusetts, Amherst) and M. Sevoian. *Infect Immun* 8(3):482-487, 1973.

3214 INHIBITORY EFFECT OF MYCOSTATIN ON THE PLAQUE FORMATION BY MAREK'S DISEASE HERPESVIRUS. *(E.)* Cho, B. K. (Dept. Vet. Sci., Washington Agricultural Experiment Station, Pullman) and W. R. Bryson. *Am J Vet Res* 34(9):1211-1212, 1973.

3215 ONCOGENICITY OF *HERPESVIRUS ATELES* IN MONKEYS. *(E.)* Laufs, R. (Hygiene-Inst., U. Göttingen, West Germany) and L. V. Melendez. *J Natl Cancer Inst* 51(2):599-608, 1973.

3216 ISOLATION OF GLUCOSAMINE FROM THE CAPSIDS OF A PICORNAVIRUS. *(E.)* Halperen, S. (E. I. du Pont de Nemours Co., Wilmington, Del.), H. O. Stone and B. D. Korant. *J Gen Virol* 20(3):267-276, 1973.

3217 EPIDEMIOLOGY OF MAREK'S DISEASE (MD): STUDIES ON THE INCIDENCE OF MD PRECIPITINS IN SOME ZOO BIRDS. *(E.)* Mohanty, G. C. (Indian Vet. Res. Inst., Izatnagar), L. N. Acharjyo and B. S. Rajya. *Poult Sci* 52(3):963-966, 1973.

3218 THE INFLUENCE OF MATERNAL EFFECTS ON THE RESPONSE OF FAST AND SLOW GROWING CHICKENS TO A MAREK'S DISEASE VIRUS. *(E.)* Han, P. F.-S. (Dept. Vet. Animal Sci., U. Massachusetts, Amherst) and J. R. Smyth, Jr. *Poult Sci* 52(3):909-915, 1973.

3219 GROWTH IN SPINNER CULTURE OF AKR MOUSE CELLS, THE K3b, INDUCED BY THE SCHMIDT-RUPPIN STRAIN OF ROUS SARCOMA VIRUS. *(E.)* Okazaki, T. (Sch. Med., Chiba U., Japan), B. Simizu and T. Kuwata. *Folia Biol (Praha)* 19(4):293-295, 1973.

3220 ATTEMPTED INDUCTION OF SARCOMA IN A CAPE CHACMA BABOON WITH THE SNYDER-THEILEN FELINE SARCOMA VIRUS. *(E.)* Van Der Riet, F. De St. J. (U. Rand, Coll. Med., Johannesburg, South Africa) and L. B. Kahn. *S Afr Med J* 47(22):943-944, 1973.

3221 *HERPVESVIRUS HOMINIS* INFECTION IN NEWBORN MICE. I. AN EXPERIMENTAL MODEL AND THERAPY WITH IODODEOXYURIDINE. *(E.)* Kern, E. R. (U. Utah, Coll. Med., Salt Lake City), J. C. Overall, Jr. and L. A. Glasgow. *J Infect Dis* 128(3):290-299, 1973.

3222 LYMPHOSARCOMAS IN TWO GIBBONS *(HYLOBATES LAR)* WITH ASSOCIATED C-TYPE VIRUS. *(E.)* Snyder, S. P. (Sch. Vet. Med., U. California, Davis), D. L. Dungworth, T. G. Kawakami, E. Callaway and D. T.-L. Lau. *J Natl Cancer Inst* 51(1):89-94, 1973.

3223 THE ACTION OF CRUDE NUCLEIC ACID PREPARATIONS EXTRACTED FROM HUMAN LEUKEMIC OR NEOPLASTIC TISSUES ON VARIOUS MOUSE STRAINS. *(E.)* Nastac, E. (S. S. Nicolau Inst. Virology, Bucharest, Romania), T. Ionescu, M. Hozoc, E. Ursu, P. Athanasiu, M. Lungu and M. Stoian. *Rev Roum Virol* 10(2):127-138, 1973.

3224 APPEARANCE OF CARCINOMA IN MICE INOCULATED WITH ROUS SARCOMA OR RNA SARCOMA EXTRACT. *(Fr.)* Athanasiu, P. (S. S. Nicolau Virology Inst., Bucharest, Romania), M. Lungu and E. Nastac. *Rev Roum Virol* 10(2):105-108, 1973.

3225 SINUS HISTIOCYTOSIS WITH MASSIVE LYMPHADENOPATHY. REPORT OF A CASE ASSOCIATED WITH ELEVATED EBV ANTIBODY TITERS. *(E.)* Lober, M. (Tulane U. Med. Ctr., New Orleans, La.), W. Rawlings, G. R. Newell and R. J. Reed. *Cancer* 32(2):421-425, 1973.

3226 ABELSON VIRUS ACTIVITY IN MICE AND RATS NEUTRALIZED WITH ANTI-RAUSCHER VIRUS SERUM. *(E.)* Cox, K. O. (Dept. Microbiol., U. Western Australia, Perth) and D. Keast. *J Natl Cancer Inst* 51(2):671-673, 1973.

See also:

* (Rev): 3004, 3005, 3013, 3017, 3018, 3022
* (Chem): 3072
* (Immun): 3231, 3233, 3234, 3235, 3238, 3242, 3244, 3246, 3248, 3251, 3257, 3263, 3267, 3273, 3276, 3281

3227 CRITICAL ROLE OF DETERMINANT PRESENTATION IN THE INDUCTION OF SPECIFIC RESPONSES IN IMMUNOCOMPETENT LYMPHOCYTES.

(E.) Katz, D. H. (Harvard Med. Sch., Boston, Mass.) and E. R. Unanue. *J Exp Med* 137(4):967-990, 1973.

Analysis of the role of determinant presentation in the process of triggering immunocompetent lymphocytes has been made utilizing cell-bound hapten-carrier conjugates to elicit secondary anti-hapten antibody responses, primarily *in vitro*. The hapten-conjugates were 2,4-dinitrophenyl (DNP) bovine gamma globulin or keyhole limpet hemocyanin. Cells used for binding the hapten-conjugates were mouse macrophages or mouse embryo fibroblasts. The *in vitro* immune responses were observed with spleen cell cultures of mice which had been primed previously with conjugated hapten, and were measured by a modified homolytic plaque technique for IgM and IgG anti-DNP plaque-forming cells. The results of these experiments demonstrate that: (a) hapten-protein conjugates will attach to the surface membranes of macrophages directly, in the absence of specific antibodies, in a highly immunogenic form; (b) such macrophage-bound conjugates serve as remarkably efficient stimuli to trigger both thymus-derived (T) and bone marrow-derived (B) cells in a specific manner, lowering the optimal threshold antigen dose (in molar terms) by several logs as compared with soluble antigen; (c) the macrophage is not unique in this regard, since fibroblasts are essentially comparable in the capacity to present antigen in highly immunogenic form; (d) cell surface-bound antigen clearly favors secondary *in vitro* responses of the IgG as compared with the IgM antibody class; (e) in terms of triggering B or T cells, antigen bound to macrophages in the form of immune complexes does not appear to possess any appreciable advantage over equimolar quantities of directly attached antigen; (f) the increased immunogenicity of cell-bound antigen appears to reflect certain crucial, and undefined, features of cell surface membranes and not merely the stabilization of determinants on a relatively immobile surface; and (g) although the efficiency of lymphocyte triggering is markedly enhanced by cell-bound antigen, the presence of macrophages is apparently not an absolute requirement for eliciting secondary *in vitro* antibody responses to soluble hapten-protein conjugates. The relevance of these observations to the nature of the signal induced upon antigen interaction by specific lymphocytes and the sequential cellular events involved in the regulatory influence of activated T cells on B cell responses to antigen is discussed. It is postulated that T lymphocytes are best triggered by cell-bound antigen and that after this step the activated T lymphocytes regulate the triggering B cells with antigen.

3228 CELL-SURFACE IMMUNOGLOBULINS IN CHRONIC LYMPHOCYTIC LEUKEMIA AND ALLIED DISORDERS.

(E.) Aisenberg, A. C. (Huntington Mem. Hosp., Boston, Mass.), K. J. Bloch and J. C. Long. *Am J Med* 55(2):184-191, 1973.

Immunoglobulin on the surface of peripheral blood lymphocytes from 57 patients with chronic lymphocytic leukemia (CLL) and allied disorders was investigated by fluorescence microscopy and correlated with circulating immunoglobulin. In 38 of 48 patients with CLL, the predominant surface immunoglobulin identified on peripheral blood lymphocytes was M (IgM) of either kappa or lambda light chain type. In five patients, the predominant surface protein was immunoglobulin G (IgG) of either kappa or lambda type. The finding of predominantly one type of immunoglobulin heavy chain and one type of light chain on the neoplastic lymphocytes of each patient established the monoclonal nature of this neoplastic proliferation. In three of these five patients no surface immunoglobulin was detected, and the lymphocyte surface immunoglobulin could not be definitely identified in the other two. The presence of immunoglobulin on the CLL cells supported the view that they originate from immune cells of the B type. Circulating immunoglobulin levels, particularly IgM, were depressed in the majority of patients with CLL, perhaps through a displacement of normal precursor cells by the effete neoplastic clone. In three subjects with IgM-bearing lymphocytes the serum contained a circulating IgM M component, and three of the five subjects with IgG-bearing cells had a circulating IgG M component. In three patients with CLL, immunoglobulin disappeared from the cell surface with progession of the disorder, although neoplastic cells remained in the circulation. The amount of immunoglobulin on the surface of cells from patients with chronic lymphosarcoma cell leukemia was much greater than that on cells from patients with CLL, and the surface immunoglobulin pattern in hairy cell leukemia also appeared distinctive. Study of immunoglobulin on the surface of lymphocytes has helped to define the cellular origin and monoclonal nature of CLL, the source of circulating M components in this disease, and the relationship of CLL to other lymphoproliferative disorders.

3229 STUDIES ON THE DISTRIBUTION OF SURFACE IMMUNOGLOBULINS ON HUMAN B-LYMPHOCYTES.

(E.) Ault, K. A. (Harvard Med. Sch., Boston, Mass.), M. J. Karnovsky and E. R. Unanue. *J Clin Invest* 52(10): 2507-2516, 1973.

Normal human peripheral blood lymphocytes were studied with fluorescent anti-immunoglobulin antibodies and shown to have a patchy distribution of immunoglobulin (Ig) on their surfaces that does not form a cap after complexing with antibody. Use of freeze-etch electron microscopy confirmed the distribution of immunoglobulin in isolated patches on the membrane. It appeared that the several thousand angstroms separating the groups of Ig prohibited cross-linking by either antibody or by anti-antibody sandwich. This wide separation of groups of Ig in human lymphocytes seemed to be an adequate, of not complete, explanation for lack of capping as compared with mouse B-lymphocytes. Studies of the metabolism of antibody bound to the cell surface revealed rapid shedding of complexes from the cell and also rapid endocytosis with subsequent degradation of the antibody. Several attempts to alter the distribution of immunoglobulin on the surface were unsuccessful. The fact that most human lymphocytes do not cap makes it unlikely that capping is an essential part of triggering the cell in the immune response. Possible mechanisms by which cell surface elements may be organized are discussed, as well as the significance of the results in terms of the immune response and the classification of certain lymphoproliferative diseases.

3230 NASOPHARYNGEAL CARCINOMA. IV. EVOLUTION OF COMPLEMENT-FIXING ANTIBODIES DURING THE COURSE OF THE DISEASE.

(E.) De-Thé, G. (Int. Agency Res. Cancer, Lyons, France), R. Sohier, J. H. C. Ho and R. Freund. *Int J Cancer* 12(2):368-377, 1973.

Sequential sera from 37 patients (26 males, 11 females; aged 25-62 yr) with nasopharyngeal carcinoma (NPC), with either controlled or uncontrolled tumors after radiotherapy, were studied for complement-fixing (CF) antibodies against a soluble antigen extracted from a leukemia-derived and virus-producing lymphoblastoid line (QIMR-WIL). When compared with results obtained previously with the same antigen on sequential Burkitt's lymphoma (BL) sera, the NPC sera exhibited (1) approximately tenfold higher CF antibody titers than the BL sera; (2) an overall stability of CF titers in the uncontrolled tumor groups contrasting with the situation in BL; (3) a two- to four-fold decrease in CF titers in the controlled tumor groups; (4) similar CF titers in patients at stage I or IV of the disease. The above suggests an active and long-standing Epstein-Barr virus infection before and during the development of the clinical NPC disease.

3231 EXPRESSION OF THE MAJOR GROUP-SPECIFIC ANTIGEN (GS-A) OF AVIAN TYPE-C VIRUSES IN NORMAL CHICKEN CELLS AND TISSUES.

(E.) Vaheri, A. (Dept. Virol., U. Helsinki, Finland) and E. Ruoslahti. *Int J Cancer* 12(2):361-367, 1973.

The major group-specific antigen (gs-a) of avian type-C viruses was detected by radioimmunoassay in all tissue samples and cultured cells from leukosis-free Brown Leghorn chickens. Visceral tissues of individual embryos contained 7-36 ng antigen/mg protein. The mean values in adult tissues were 2-5 times lower than those of the embryos, except for spleen which gave exceedingly high values of about 50 ng/mg protein. The gs-a levels of tissues and cells producing avian type-C viruses were 100-1000 times higher. In such cells up to 1% of cellular protein can be gs-a. The gs-a in normal tissues was indistinguishable from the viral gs-a: cellular and viral gs-a inhibited chicken and rabbit anti-gs serum to the same degree and gave parallel inhibition curves. Differences in expression of gs-a and other malignancy-associated proteins in normal tissues may prove useful indicators of loosened gene regulation as an initial step towards malignancy.

3232 INCREASE OF LYMPHOCYTE-MEDIATED TUMOR-CELL DESTRUCTION BY CERTAIN PATIENT SERA.

(E.) Hellström, I. (U. Washington Sch. Med., Seattle), K. E. Hellström and G. A. Warner. *Int J Cancer* 12(2):348-353, 1973.

Sera from one patient with an ovarian carcinoma, one with sarcoma, one with a breast carcinoma, and four patients with melanoma were found to potentiate (i.e., increase) the cytotoxic effect of blood lymphocytes reactive to the respective type of tumor target cells. Of these patients, four were without evidence of growing tumor at time of serum sampling. Furthermore, sera from the breast cancer and sarcoma patients and from one of the melanoma patients "armed" (i.e., made cytotoxic) lymphocytes from nonreactive donors, not having (had) the respective tumor studied. Both the potentiating and arming serum effects were specific. The possible relevance, *in vivo*, of these findings, and the frequency with which they occur, need further clarification. It is suggested that the potentiating molecules are antigen-antibody complexes and that the cells on which they act are of non-T origin. It is possible that potentiating serum factors act by unblocking tumor antigen-antibody complexes or by arming reactive lymphoid cell populations in a way similar to that observed for nonimmune lymphocytes.

3233 STUDY BY THE ISOTOPIC ANTIGLOBULIN TECHNIQUE OF A CROSS-REACTING MURINE ANTIBODY PRODUCED BY IMMUNIZATION WITH A SYNGENEIC SV40 TUMOR.

(E.) Burdick, J. F. (Natl. Cancer Inst., Bethesda, Md.), D. Stemple and S. A. Wells, Jr. *Int J Cancer* 12(2):474-484, 1973.

A sarcoma virus (SV) tumor line, SV-AL/N, derived from SV40 transformation of an embryonic cell line, was used to immunize syngeneic mice, and the resultant antisera were investigated with the modified isotopic antiglobulin assay. A single specificity was detected in the antiserum, which reacted strongly with an antigen present on SV40-transformed lines. However, the antigen was also present on cells derived from the same embryonic line as SV-AL/N but transformed instead either spontaneously or by polyoma virus. The antigen was present on one nontransformed syngeneic embryonic line and may have been present in low concentration in a nontransformed syngeneic fibroblast line; it was not detected in several methylcholanthrene-induced lines. Antiserum derived from multiple immunizations with irradiated syngeneic fetuses reacted weakly in a similar pattern. The results suggest that the antigen detected is present in the host genome in occult form, and is derepressed by several processes, perhaps including embryogenesis.

3234 CELL-SURFACE AND VIRION-ENVELOPE ANTIGENS SHARED BY RADIATION LEUKEMIA VIRUS (RadLV) AND OTHER MURINE C-TYPE VIRUSES.

(E.) Ferrer, J. F. (Sch. Vet. Med., U. Pennsylvania, Kennett Square). *Int J Cancer* 12(2)?378-388, 1973.

To test for a specific transplantation resistance induced by immunization with tumor-cell suspensions (i.p. and s.c., every 7-20 days), young adult W/Fu rats were exposed to a total-body dose of 400-500 R prior to challenge with a selected syngeneic lymphoma. The results demonstrate that rats recognize a tumor-specific transplantation antigen(s) shared by tumors induced by Radiation Leukemia Virus (RadLV), Gross Leukemia Virus (GLV), Moloney Leukemia Virus (MLV) or Moloney Sarcoma Virus (M-MSV), which is different from the type-specific transplantation antigen(s) detected by mice. Direct cytotoxic tests and absorption experiments with specific antisera confirm and extend previous studies indicating that rats also form antibodies to a cell-surface cross-reacting antigenic determinant present in tumors induced by these viruses. Results of cytotoxic tests with a monospecific guinea-pig antiserum for the intraspecies group-specific (gs-1) antigen of murine leukemia viruses suggest that this antigen is present at the cell surface of tumors induced by RadLV, GLV, of MLV, and thus may be responsible for the cross-reactions observed between these tumors in the present study. The data indicate that the rat-typing system also recognizes a type-specific MLV antigen(s) and support the idea that some MLV-induced leukemias may be superinfected and antigenically converted by GLV. Virus neutralization studies show that rats form antibodies to virion-envelope antigens shared by RadLV, GLV and MSV.

3235 SPONTANEOUS BREAKDOWN OF IMMUNOLOGICAL TOLERANCE TO GROSS-VIRUS-INDUCED CELL-SURFACE ANTIGENS IN THE RAT. *(E.)* Kuzumaki, N. (Hokkaido U. Sch. Med., Sapporo, Japan), N. Takeichi, T. Kodama and H. Kobayashi. *Int J Cancer* 12(2):389-395, 1973.

Inbred Wistar-King-Aptekman (WKA/Mk) rats injected, by a modification of Chenaille's method, with undiluted Gross virus at birth demonstrated loss of resistance to transplanted Gross lymphoma (WGT-4), and also during later life did not contain detectable cytotoxic antibody to Gross virus-specific cell-surface antigen. These rats were considered to be immunologically tolerant to the Gross-virus-induced cell-surface antigens, and all of them developed primary Gross lymphoma about 3 months after birth. Some rats injected s.c. or i.p. with diluted virus at birth or undiluted virus at the age of 1 or 2 weeks also continued to be immunologically tolerant to Gross-virus-induced cell-surface antigens for a certain period in proportion to the amount of virus administered, or age at the time of exposure to the virus. Thereafter, this tolerant state was spontaneously broken down and the rats developed transplantation resistance to WGT-4, and produced cytotoxic antibody to Gross antigens for a considerable period. These rats did not develop Gross lymphoma. These facts suggest that susceptibility to leukemogenesis by the Gross virus in WKA/Mk rats might be ascribed to the immunologically tolerant state to the Gross-virus-induced cell-surface antigens.

3236 TUMOR IMMUNITY *IN VIVO*: EVIDENCE THAT IMMUNE DESTRUCTION OF TUMOR LEAVES "BY-STANDER" CELLS INTACT. *(E.)* Weissman, I. L. (Dept. Path., Stanford U. Sch. Med., Cal.). *J Natl Cancer Inst* 51(2):443-448, 1973.

Certain previous studies of the mechanism of cell-mediated immunity (CMI) revealed a nonspecific component of the effector limb in *in vitro* experiments, indicating that although specificity resides in the recognition of target cells by immune lymphoid cells, the actual destruction process involves the release of nonspecific mediators. This conclusion appeared to be supported by *in vivo* experiments, wherein the induction of a delayed-hypersensitivity reaction to a third-party antigen in a cutaneous neoplasm led to local tumor regression. In contrast, other studies did not reveal syngeneic tumor destruction in a local CMI reaction to an allogeneic tumor in nonimmune hosts. Since using nonimmune hosts allowed the syngeneic cells to form micronodules before the effector phase of CMI developed, the present experiments were designed with the use of hosts actively immune to tumor-associated transplantation antigens. Moloney sarcoma (MS) regressors were highly immune to a transplantable MS tumor cell line but not to a syngeneic, undifferentiated mammary tumor, EMT6. Limiting numbers of EMT6 cells in suspension were intermixed with 1- to 50-fold more MS cells and transplanted into MS regressors or nonimmune hosts. The incidence of EMT6 tumor growth in mixed tumor sites in MS-immune hosts did not differ significantly from that in sites without admixed MS cells. It is concluded that the effector arm of cell-mediated immunity to MS cells involves specificity in both the recognition and target-cell destruction processes. It is suggested that there may be a fundamental difference between tumor target-cell destruction on the one hand and cutaneous delayed hypersensitivity on the other. Apparently *in vivo* testing of specificity of CMI effector functions should parallel *in vitro* analysis whenever possible.

3237 IMMUNE RESPONSE IN FAMILIES OF CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKAEMIA. *(E.)* Evans, D. I. K. (Dept. Child Health, U. Manchester, England). *Arch Dis Child* 48(6):441-445, 1973.

The immune status of 33 mothers, 17 fathers, and 14 sibs of children with acute lymphoblastic leukemia was studied with the following tests: peripheral blood lymphocyte counts, lymphocyte transformation with phytohemagglutinin, serum immunoglobulin levels, isohemagglutinin titers, and levels of antibody to herpes simplex and Epstein-Barr viruses. No significant abnormality was shown by these tests, with the exception of a lymphocytosis detected in parents at an early stage of their child's disease. The theory that maternal viral infection relates to childhood acute lymphoblastic leukemia is given indirect support by this unexpected finding. It is concluded that there is no defect of immunity in these families.

3238 ANTIBODIES REACTING TO SIMIAN VIRUS 40 T ANTIGEN IN HUMAN SERA. *(E.)* Shah, K. V. (Johns Hopkins U. Sch. Hyg. Pub. Hlth., Baltimore, Md.), R. W. Daniel and G. Murphy. *J Natl Cancer Inst* 51(2):687-690, 1973.

In a program involving periodic examination of urine specimens for papovavirus and of sera for antibody titer, 1 of 19 renal transplant recipients showed in 13 successive serum specimens the presence of antibodies reacting to simian virus 40 (SV40) T antigen in fluorescent antibody tests. These sera had minimal or undetectable levels of SV40-neutralizing antibodies but high titers of antibodies to B.K. virus, an SV40-related papovavirus infection of man. There was no clinical or laboratory evidence that the patient had a tumor. In addition, SV40 T antigen-reactive antibodies were found in the sera of two of 30 Maryland children with B.K. hemagglutination inhibition antibody titers of 1:640 or above but in none of 75 children with titers ranging from < 1:20 to 1:320. The two SV40 T antigen-reactive sera were negative for SV40-neutralizing antibodies. Hamster sera containing only SV40 T antibodies specifically stained the nuclei of B.K. virus-infected vero cells. These data suggest that the observed reaction of human sera with SV40 T antigen is the result of a minor antigenic relationship between T antigens of SV40 and B.K. virus.

3239 THE EFFECTS OF TUMOR BEARING UPON CHANGES IN CELL DISTRIBUTION AND MEMBRANE ANTIGEN CHARACTERISTICS IN MURINE SPLEEN AND THYMUS CELL SUBPOPULATIONS. *(E.)* Konda, S. (Coll. Med., U. Florida, Gainesville) and R. T. Smith. *Cancer Res* 33(8):1878-1884, 1973.

A model system was developed for examining the impact of transplanted syngeneic methylcholanthrene-induced tumors upon the various lymphoid cell subpopulations of mouse spleen and thymus. Recipients of all tumor implants were 7- to 8-wk old female C57BL/6 and CBA mice. Fibrosarcomas were produced by injecting 0.5 mg methylcholanthrene in 0.1 ml olive oil i.m. in four sites. The data define qualitative aspects of the 10-fold increase in numbers of spleen cells, and the equivalent reduction in thymus cells, during the 40- to 50-day course of tumor bearing in C57BL/6 or CBA mouse strains. The proportion of spleen cells susceptible to anti-θ or anti-H-2 plus complement is reduced, but the absolute numbers increased progressively. The proportion of anti-H-2 susceptible thymus cells increased markedly. These changes are interpreted as reflecting a net increase in T-cells in the spleen during tumor bearing. The proportional reduction in T-cells in the spleen, therefore, appears to represent a dilution effect, whereas, in fact, a net increase in total spleen T-cells occurs. This study provides no explanation for the increased numbers of T-cells present. It is plausible, however, that a proportion of the increased T-cells in the system carries specificities directed against the tumor. On the other hand, there is evidence that enhanced competence with respect to T-dependent nontumorous antigens is also characteristic under these circumstances. The increased T-cell population is probably not exclusively tumor directed.

3240 SURFACE IMMUNOGLOBULINS OF LEUKAEMIC CELLS. *(E.)* Foulis, A. K. (Dept. Path., U. Glasgow, Scotland), A. J. Cochran and J. R. Anderson. *Clin Exp Immunol* 14(4):481-490, 1973.

In view of the available evidence that leukemic and lymphomatous lymphoid cells show some of the characteristics of thymus-dependent (T) or bone marrow (B) lymphocytes, depending, presumably, on their histogenesis, a study was made of the presence of surface immunoglobulins in leukemic cells from 28 patients: 19 chronic lymphatic leukemia; 7 chronic myeloid leukemia; 1 acute myeloid leukemia; 1 acute lymphoblastic leukemia. Chronic lymphatic leukemia cells were classified on the basis of their reactivity with anti-immunoglobulin sera in a leukemia cell migration inhibition test (MIT) and by their responsiveness to phytohemagglutinin. Immunoglobulins in eluates or lysates were detected by agglutination and MIT tests. Immunoglobulins were identified on the cells of 14 out of 19 patients with chronic lymphatic leukemia but were not present on cells from patients with chronic myeloid leukemia or acute leukemia. The results obtained with the migration technique were confirmed by identification of immunoglobulins in eluates and lysates of the leukemia cells. Serial elution and lysis studies indicated that in some cases the immunoglobulins were produced by the leukemia cells at a low but steady rate, possibly indicating a relationship between those cells and B cells. In other cases the immunoglobulins were readily removed from the cells and were present only in the initial eluates from the cells, suggesting that they had been absorbed and might possibly be specific antibody. The immunoglobulin negative chronic lymphatic leukemia cells may be related to T cells, a possibility supported by a high level of phytohemagglutinin induced transformation in one case examined by this technique.

3241 DISTRIBUTION OF A CROSS-REACTIVE IDIOTYPIC SPECIFICITY IN INBRED STRAINS OF MICE. *(E.)* Pawlak, L. L. (U. Illinois Coll. Med., Chicago) and A. Nisonoff. *J Exp Med* 137(4):855-869, 1973.

The expression of an idiotype characteristic of anti-*p*-azophenylarsonate (anti-Ar) antibodies of all A/J mice was explored in F_1 progeny, in 16 other inbred strains, and in 4 cogenic strains of mice. Of the strains tested only those closely related to A/J produced antibodies with the cross-reactive idiotype (CRI). None of the mice synthesized intermediate levels of CRI. No relationship between *H-2* type and idiotype was noted. Congenic mice with a strain A background but a different *H-2* type produced CRI in amounts quantitatively equivalent to those of strain A mice. Conversely, the presence of the *H-2* genotype of strain A on an unrelated background was not associated with the formation of CRI. Nearly all F_1 progeny of strain A mice formed CRI, indicating that failure of the other (non-A) parental strain to produce CRI is not attributable to the presence of a gene controlling the synthesis of a suppressor of CRI. NZB mice, which have the same heavy chain allotype as strain A, but are unrelated in origin failed to produce CRI, although allotype has been shown to be linked to idiotype in congenic strains. One interpretation of these patterns of cross-reactivity is that different strains of mice have different sets of germ line V genes. An alternative possibility is that different strains have similar germ line genes but that a regulatory gene controls the production of CRI. The results suggest, however, that the failure of BALB/c or C57BL mice to express the cross-reactive anti-Ar idiotype is attributable to a difference, as compared with A/J, in germ line V genes rather than to the presence of a regulatory gene which prevents the expression of the cross-reactive anti-Ar idiotype.

3242 CELL SURFACE PROPERTIES AND THE EXPRESSION OF SV40-INDUCED TRANSFORMATION. *(E.)*

Smith, H. S. (Sch. Pub. Hlth., U. California, Berkeley), A. J. Hiller, E. W. Kingsbury and C. Roberts-Dory. *Nature* [*New Biol*] 245(142):67-69, 1973.

Different clones of SV40 transformed mouse embryo Balb/3T3 line of cells express transformation in different ways. To examine the correlation between type of transformation and surface properties said to be characteristic of transformed cells, the different clones were tested for agglutinability by concanavalin A (con A) and for membrane staining by ruthenium red as seen in the electron microscope. The different clones consisted of untransformed Balb/3T3, standard transformants (growing in multiple layers), flat transformants (growing in factor-free medium but not in multiple layers), and flat revertants (similar to flat transformants but arising from standard transformants). The standard SV40 transformed lines agglutinated with con A much more extensively than did non-transformed controls or flat transformants. However, a flat revertant, selected for growth to high density, was agglutinated by con A. With regard to staining with ruthenium red, standard transformants were positive, albeit to varying degrees, as also were those flat transformants and revertants which continued to synthesize DNA at confluence, while the one flat transformant which did not synthesize DNA at confluence stained weakly like the non-transformed control. It was pointed out that although most of the flat transformants and revertants are not contact inhibited and thus differ from control cells, they neither agglutinate with con A nor have transplantation immunity antigen. Thus the loss of contact inhibition *per se* is not sufficient to express other alterations associated with tumorigenicity. The increased staining of the flat transformants and revertants by ruthenium red suggests that an increase of acid mucopolysaccharide on the cell membrane is related to loss of contact inhibition.

3243 LYMPHOCYTE BLASTOGENIC RESPONSES TO AUTOCHTHONOUS VIABLE AND NONVIABLE HUMAN TUMOR CELLS. *(E.)*

Mavligit, G. M. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.), E. M. Hersh and C. M. McBride. *J Natl Cancer Inst* 51(2):337-343, 1973.

The finding that mitomycin C-treated human tumor cells can stimulate a weak blastogenic response among autologous lymphocytes and that treatment of the tumor cells with neuramidase augmented this response suggested that the nature and physiologic state of the tumor cells determine the nature and degree of the blastogenic response. In the present study, *in vitro* lymphocyte blastogenic response to autochthonous tumor cells was studied in 11 patients with solid tumors. After viable cells were separated from nonviable tumor cells by differential centrifugation on Ficoll-Hypaque density solution, they were exposed to irradiation, mitomycin C, or no treatment. These conditions produced tumor-cell preparations with various degrees of stimulatory ability. Viable tumor cells have some advantage over nonviable tumor cells, and irradiated tumor cells were superior to those treated with mitomycin C in terms of tumor-induced blastogenesis among autologous lymphocytes. Viable tumor cells may be superior because they furnish a better source of tumor-associated antigen than nonviable cells. Treatment of dying and disintegrating cells with irradiation or mitomycin C probably removes more antigen or further reduces their ability to synthesize new antigen. Conversely, cell viability and continued cell-surface antigen production may be a requirement for vigorous stimulator cell activity. Diminished antigenicity in mitomycin C-treated cells may be due to changes in membrane properties caused by the drug.

3244 EXPRESSION OF FETAL ANTIGENS AND TUMOR-SPECIFIC ANTIGENS IN SV40-TRANSFORMED CELLS. II. TUMOR TRANSPLANTATION STUDIES. *(E.)*

Ting, C.-C. (Natl. Cancer Inst., Bethesda, Md.), D. Rodrigues and R. B. Herberman. *Int J Cancer* 12(2):519-523, 1973.

In previous work, immunization with fetal tissues produced resistance to challenge of SV40 or adenovirus-induced tumors in hamsters, but such immunization failed to give protection against polyoma or SV40 tumors in mice. The latter negative results did not appear to be due to the use of non-irradiated cells since X-irradiated cells also failed to protect. In the present study, the effect of immunization with fetal antigens and tumor-specific antigens on the transplantation of SVT2, a BALB/c mouse tumor induced by Simian virus 40 (SV40), has been studied. Hyperimmunization with 5,000 R X-irradiated, syngeneic fetuses of 1-2 weeks gestation or syngeneic spleen cells gave no protection to SVT2 cell challenge. In contrast, immunization with SV40 or irradiated SV40-transformed cells gave a 100- to 1,000-fold protection. These results gave additional evidence that fetal antigens are different from tumor-specific transplantation antigen (TSTA). Apparent conflict with the findings obtained previously with hamster SV40 tumors was suggested to have been due to elimination of weak or non-specific immunization by pre-irradiation of the mice before challenge, by the possible presence of more transplantation-type fetal antigens in SV40-induced hamster tumors, or by greater responsiveness of hamsters to fetal antigens.

3245 TRANSMISSION OF RESISTANCE TO TRANSPLANTED TUMORS IN THE RAT. *(Ger.)*

Matthies, E. (Hyg. Inst., Martin Luther U., Halle-Wittenberg, Germany) and W. Ponsold. *Z Krebsforsch* 80(1):27-30, 1973.

An inbred rat line was developed by breeding only litter mates who were resistant to Jensen sarcoma and who had survived a test transplantation before they were bred. From the seventh filial generation, rats were bred without first transplanting the tumor to develop tumor resistance. At present, observations are being carried out on the 35th filial generation. All of the rats from the fifth filial generation on were resistant to Jensen sarcoma, regardless of whether they received transplants when young or after they stopped breeding. Although the virulence of the Jensen sarcoma did vary somewhat during the observation period, highly significant differences were found in all generations between the tumor resistance of the inbred line and that of controls. In subsequent generations the inbred line of rats also became resistant to Yoshida sarcoma and Guerin carcinoma. The tumor resistance of these inbred rats was not transmitted passively from the tumor resistance of the mother since, from the seventh generation on, rats were mated without first being given a test transplant. It is suggested that tumor resistance in these rats results from an inherited ability to develop immunity to certain transplanted tumors.

3246 IMMUNOLOGICAL RELATIONSHIP OF DNA POLYMERASE FROM HUMAN ACUTE LEUKAEMIA CELLS AND PRIMATE AND MOUSE LEUKAEMIA VIRUS REVERSE TRANSCRIPTASE.

(E.) Todaro, G. J. (Natl. Cancer Inst., Bethesda, Md.) and R. C. Gallo. *Nature* 244(5413): 206-209, 1973.

The biochemical and immunological properties of reverse transcriptase from human acute myeloblastic leukemia cells have been studied and compared with those from known primate and mouse C-type viruses. Human transcriptase activity banded in glucose gradients at a density of 1.16 gm/cm^3, identical to that at which Rauscher leukemia virus was found. Like feline and woolly monkey type-C transcriptases, human leukemic transcriptase preferred poly $(rA)\cdot(dT)_{12-18}$ as compared to poly $(dA)\cdot(dT)_{12-18}$ as template. Human transcriptase, like the viral enzymes, showed an endogenous ribonuclease-sensitive reaction, could make a DNA product using avian myeloblastosis virus 70S RNA, and was able to transcribe poly (rC) in the presence of $(dG)_{12}$. Antisera prepared against reverse transcriptase from gibbon virus strongly inhibited reverse transcriptase from human leukemia cells, followed in effectiveness by antisera against enzymes from woolly monkey sarcoma virus and mouse leukemia virus. Antisera prepared against reverse transcriptases from feline leukemia virus, RD114 virus, Rous sarcoma virus and Mason-Pfizer monkey virus had low inhibitory activities on human leukemia cell enzyme. Thus the human leukemia cell reverse transcriptase is most closely related to primate type-C viruses, especially that of the gibbon ape.

3247 IMPAIRED RESPONSIVENESS OF LYMPHOCYTES AND SERUM-INHIBITORY FACTORS IN PATIENTS WITH CANCER.

(E.) Suciu-Foca, N. (Coll. Phys. Surg., Columbia U., New York, N.Y.), J. Buda, J. McManus, T. Thiem and K. Reemtsma. *Cancer Res* 33(10): 2373-2377, 1973.

Cellular immune responsiveness, as measured by lymphocyte transformation in one-way mixed-leukocyte cultures and in phytohemagglutinin (PHA)-stimulated cultures was evaluated in 60 patients with cancer and in 81 normal controls. The effect produced by sera from these subjects on *in vitro* transformation of lymphocytes was tested in autologous cells and on homologous responding cells from a constant panel of 10 healthy volunteers. The responsiveness of lymphocytes from cancer patients to PHA and to a battery of mitomycin-treated allogeneic cells was significantly lower than that of normal controls. Lymphocyte reactivity in the presence of autologous cancer serum was inversely related to the extent of the disease. Pooled normal serum enhanced the responsiveness of lymphocytes from cancer patients but had no such effect on control cultures from healthy subjects. Sera from cancer patients reduced the PHA and mixed-leukocyte culture responsiveness of normal panel lymphocytes to a level that was significantly lower than that found in the presence of sera from healthy controls. The degree of inhibition produced by cancer sera on the blastogenic response of normal lymphocytes increased with advanced disease. Suppression of lymphocyte transformation was markedly greater in mixed-leukocyte cultures than in cultures exposed to PHA. Out of 60 cancer sera investigated, 23 possessed *HL-A* reactivity against a random panel of lymphocytes. The findings demonstrate that serum-inhibitory factors are at least partially responsible for impairment of T-cell responsiveness in cancer patients. It is suggested that isoantigenic modifications on the surface of cancer cells cause the appearance of blocking antibody, with broad cross-reactivity that might bind to the surface of responding and/or target cells.

3248 ANTIBODIES TO EPSTEIN-BARR VIRUS-RELATED ANTIGENS IN NASOPHARYNGEAL CARCINOMA. COMPARISON OF ACTIVE CASES WITH LONG-TERM SURVIVORS.

(E.) Henle, W. (Children's Hosp., Philadelphia, Pa.), H.-C. Ho, G. Henle and H. C. Kwan. *J Natl Cancer Inst* 51(2):361-369, 1973.

Differentiation between EBV-related antigens has provided specific procedures for the detection and titration of the corresponding antibodies. Application of these techniques to longitudinal study of Burkitt's lymphoma (BL) patients revealed that changes in the spectra and titers of antibodies to EBV-related antigens may be referable to preceding or succeeding clinical events. Since similar studies of nasopharyngeal carcinoma (NPC) patients had not yet been performed, sera from nasopharyngeal carcinoma (NPC) patients before treatment (BT) and from those who had survived 4-8 years after radiation therapy were titrated for antibodies to Epstein-Barr virus (EBV) capsid antigens (VCA) and to the D (diffuse) and R (restricted) components of the early antigen (EA) complex. The geometric mean anti-VCA and anti-D titers in the BT group increased substantially with progression of NPC from stage I to V. Anti-D was almost exclusively the dominant antibody to the EA complex, preventing detection of low anti-R levels which probably were also present. The long-term survivors (LTS), who initially had NPC in stages I-IV, had generally lower anti-VCA titers than the BT group. Compared to BT patients, fewer patients in the LTS group showed anti-D, usually at low titers, and many more revealed low to moderate levels of anti-R as the dominant or sole antibody to the EA complex. The apparent declines in anti-VCA and anti-D in LTS were seen regardless of the stage of disease before radiation therapy. Thus, in contrast to the BT group, no significant stage-related increments in serologic activities were evident in LTS, except that those with initially stage IV NPC tended to have somewhat higher titers than those treated at an earlier stage. These results suggest that the anti-VCA and anti-D titers decline gradually in apparently tumor-free LTS and that anti-D eventually becomes nondetectable.

3249 EVIDENCE FOR COMPROMISE OF TUMOUR IMMUNITY IN RATS BY A NON-SPECIFIC BLOCKING SERUM FACTOR THAT INACTIVATES MACROPHAGES. *(E.)* Keller, R. (Immunobiol. Res. Group, U. Zurich, Switzerland) *Br J Exp Pathol* 54(2):298-305, 1973.

Serum from rats infected 30 days previously with the nematode parasite, *Nippostrongylus brasiliensis*, was able to fully reverse the macrophage-mediated inhibition of growth of Walker 256 carcinosarcoma ascites cells in other *N. brasiliensis*-infected rats. Control serum from normal uninfected rats or rabbits had no such effect. A similar enhancement of tumor cell growth was observed in rats injected s.c. with carcinosarcoma cells preincubated with serum from mematode-infected animals, indicating that the enhancing factor was probably adsorbed onto the tumor cells. Direct observation of serum-treated cells indicated that the serum factor was active against activated peritoneal macrophages and not against the tumor cells *per se*. Studies on the effect of the serum factor on peptone-induced migration of macrophages into the peritoneal cavity *in vivo* showed that this process was unaffected; however, the motility of these macrophages and their ability to adhere to a glass culture dish were greatly reduced compared to controls. Results from studies of the effects of the serum blocking factor on two rat strains differing in major histocompatibility antigens indicated that the effects were nonspecific.

3250 MECHANISMS IN THE SUPPRESSION OF DELAYED HYPERSENSITIVITY IN THE GUINEA PIG BY 6-MERCAPTOPURINE. *(E.)* Phillips, S. M. (Peter Bent Brigham Hosp., Boston, Mass.) and B. Zweiman. *J Exp Med* 137(6):1494-1510, 1973.

The mechanism of suppression of delayed hypersensitivity to tuberculoprotein by 6-mercaptopurine (6-MP) was studied in guinea pigs. Under the conditions of the protocol, suppression of tuberculin delayed skin test reactivity was not associated with a significantly altered end-organ response to mediators of permeability. No significant alteration of *in vivo* lymphoid activity, as measured by reconstitution studies, was found. In addition, lymphoid cells from 6-MP-treated animals reacted in a fashion similar to those of placebo-treated animals with respect to (a) antigen-induced lymphocyte proliferation, (b) antigen-induced liberation of macrophage inhibitory factor activity, (c) direct inhibition by antigen of peritoneal exudate cell migration. Conversely, suppression was seen in levels of blood monocytes and *in vitro* function of macrophages from 6-MP-treated animals in several respects: (a) adherence to glass, (b) migratory rate, (c) phagocytic capacity. Therefore, it would appear that a major mechanism of 6-MP-induced suppression of delayed hypersensitivity is through its action on effector cells.

3251 IMMUNOGENICITY AND IMMUNOSENSITIVITY OF SV40-TRANSFORMED CELLS WITH DIFFERENT ONCOGENIC POTENTIALS. *(E.)* Stillström, J. (Dept. Virol., U. Uppsala, Sweden). *Int J Cancer* 12(2):493-501, 1973.

In vitro SV40-transformed cell lines appear at first to be non-oncogenic, but after repeated *in vitro* propagation become oncogenic in their species of origin, mainly in animals with depressed immunological reactivity. To find out whether the increase in oncogenic potential is accompanied by any change in the immunogenicity and immunosensitivity of the cells, ME1 C9, a cloned line of mouse embryo cells, transformed by SV40 *in vitro*, and its more malignant progeny, ME1 C9-V15, derived from a tumor of the 15th consecutive passage of MEI C9 *in vivo*, were compared in syngeneic CBA mice. The cells used for immunization were X-irradiated as also were the immunized mice prior to challenge. A threshold zone of immunization with ME1 C9 and ME1 C9-V15 cells in the range of 10^3-10^5 irradiated cells was indicated, after which the immunized animals were 1,000 times more resistant. There was no significant difference in immunizing potential between ME1 C9 and ME1 C9-V15 cells at any dose level. A heterologous line of SV40-transformed rat cells was significantly less immunogenic than the syngeneic cells. The growth of ME1 C9-V15 cells could be enhanced after immunization with spontaneously transformed ME3 cells or with a small dose of ME1 C9 cells. The immunosensitivity of the ME1 C9 and ME1 C9-V15 cells was measured as a sensitivity index in uniformly immunized animals. Both lines appeared immunosensitive if the animals were sufficiently immunized. The less malignant cells, ME1 C9, appeared more immunosensitive than the more malignant ME1 C9-V15 cells in seven out of nine experiments, but only once was the difference between the lines significant. In view of the fact that no differences in immunogenicity between cell lines were found, the differences in immunosensitivity could not be interpreted as a loss of transplantation antigens in the more malignant cells. The interpretation of differences of sensitivity indices is further discussed, and a possible role of blocking factors is considered.

3252 SPECIFICITY, CROSS-REACTIVITY, AND AFFINITY OF ^{125}I-LABELED ANTIGLIOMA ANTIBODIES FOR MONOLAYERS OF CULTURED GLIOMA CELLS. *(E.)* Day, E. D. (Duke U. Med. Ctr., Durham, North Carolina) and D. D. Bigner. *Cancer Res* 33(10):2362-2372, 1973.

The sequential method of adsorption analysis was applied to antiglioma radioantibodies adsorbed by intact monolayer cultures of the C6 rat astrocytoma cell line. A noncentrifugal technique was devised to permit transfer of radioantibody solutions from one culture to the next in a sequence and to effect removal of unbound radioglobulins. Primary binding of ^{125}I-labeled antibody immunoglobulin G (IgG) was readily measured in a particularly sensitive and reproducible manner over and above control IgG. With proportions varying from one serum to the next, several individual antiglioma radioantibody preparations, after preadsorption three times with rat liver, still retained cross-reactivity for fibroblast cultures and, after preadsorption three times with fibroblasts, still retained selective reactivity for glioma cells. When adsorbed with glioma cells first, the bulk of all binding activity remained with the glial cells. When unlabeled antibody IgG in varying amounts was mixed with fixed quantities of labeled antibody IgG and glioma cells, an equivalence point was established by competitive binding curves and, independently, by microscopic examination of the monolayer-antibody complexes after light trypsinization: cell clumps in excess antibody, free cells otherwise. Parameters of IgG binding in one antibody-cell combination were $K_a = 2.1 \times 10^6 \ M^{-1}$; a = 0.75; and s = 6.9×10^7 antigenic sites/cells. When two cloned sublines of the S-77 line of the C6 astrocytoma were used sequentially along with fibroblasts to adsorb a radioantibody preparation from one particular anti-S-77 antiserum in nine steps, four different antigenic moieties were clearly distinguished; a cross-reaction between fibroblasts and glioma cells; a cross-reaction between the two sublines that was not shared by fibroblasts; and a distinctive reaction in each of the two sublines that was not shared by the other subline or by fibroblasts.

3253 TUMOR IMMUNITY TO MURINE PLASMA CELL TUMORS. II. ESSENTIAL ROLE OF T LYMPHOCYTES IN IMMUNE RESPONSE. *(E.)* Rouse, B. T. (Walter and Eliza Hall Inst. Med. Res., Melbourne, Australia), M. Röllinghoff and N. L. Warner. *Eur J Immunol* 3(4):218-224, 1973.

Spleen cells, thoracic duct lymphocytes and adherent peritoneal exudate cells from mice immunized to syngeneic plasma cell tumors were capable of transferring specific protective immunity to these tumors. Plasma cell tumors were induced with mineral oil, and BALB/c, NBZ and (BALB/c x NBZ)F_1 mice were immunized by injection of cultured tumor cells into a limb. The limbs were removed 10 days after injection, and, 10 to 20 days after limb removal, immune lymphoid and peritoneal exudate cells were collected. Pretreatment of these cells with AKR anti-θ C3H serum or goat anti-mouse lymphocyte serum, but not with rabbit anti-mouse K serum, effectively abolished the ability of lymphoid cells to transfer this immune response. These studies demonstrate that thymus-derived lymphocyte (T) cells are essential in the adoptive transfer of immunity. Furthermore, immunization of athymic (nude) mice to plasma cell tumors was not achieved. Attempts to activate normal macrophages *in vitro* with sensitized lymphoid cells were unsuccessful. Admixture of immune lymphoid cells and target tumor cells to unrelated tumor cells did not inhibit the growth of the unrelated tumor cells. It is proposed that the immune response to syngeneic tumor associated antigens of plasma cell tumors not only involves essential sensitization of T lymphocytes, but that activated cytotoxic T lymphocytes are directly responsible for inhibiting target tumor growth.

3254 CELLULAR IMMUNITY IN CANCER PATIENTS AGAINST EMBRYONAL CELLS. *(E.)* Fossati, G. (Natl. Inst. Study Cure Tumors, Milan, Italy), S. Canevari and G. Della Porta. *J Natl Cancer Inst* 51(2):667-669, 1973.

Previous reports showed that tumors possess embryo-specific components not expressed in normal adult cells and that peripheral lymphocytes from patients with colonic carcinoma inhibited colony formation of plated cells of fetal intestine and liver as well as of colonic carcinoma. In the present work, the cellular-immune reaction of patients with different types of tumor against cells from 2 human embryos and 2 fetuses was studied by *in vitro* cytotoxic microassay. Peripheral blood lymphocytes from 13 of 19 patients with malignant tumor (melanoma, breast carcinoma, reticulosarcoma, trichoepithelioma, and carcinomas of ovary, rhinopharynx and tongue) and 1 of 6 patients with benign tumor (breast, parotid gland and tongue) showed a killing effect on 1- to 2-month-old embryo cells, whereas no cytotoxicity was demonstrated against 4- to 5-month-old fetal cells and adult skin fibroblasts. These data suggest that patients with malignant tumor are sensitized against antigen(s) in embryo cells.

3255 INHIBITION OF THE IMMUNE RESPONSE BY 7S ANTIBODY. MECHANISM AND SITE OF ACTION. *(E.)* Abrahams, S. (Ontario Cancer Inst., Toronto, Canada), R. A. Phillips and R. G. Miller. *J Exp Med* 137(4):870-892, 1973.

A cell culture system was used to investigate the mechanism of action of the feedback inhibition caused by specific 7S antibody. It was found that preincubation of spleen cells with specific 7S antibody led to a marked reduction in the *in vitro* response of the treated spleen cells to the antigen used to prepare the antibody. The inhibition was not caused by a carry-over of free antibody nor by the release of 7S antibody from the cells. Rather, the preincubation appeared to specifically inactivate one of the cells required for initiation of an *in vitro* response. Since the suppression could be reversed by addition of untreated cells, it was possible to characterize the properties of the reconstituting cell. This cell is identified as the nonlymphoid accessory cell (A cell) by several criteria: (a) suppression can be demonstrated only in assay systems requiring functional A cells; (b) the most active sources for reconstitution are also good sources for A cells; (c) the sedimentation velocity of the reconstituting cell is identical with that for A cells; (d) like A cells, the reconstituting cell is resistant to high doses of ionizing radiation; and (e) the reconstituting ability is not affected by anti-θ antibody. Additional experiments suggest that the Fc portion of 7S antibody binds to the surface of A cells. Thus, fluorescein isothiocyanate-labeled 7S antibody binds specifically to cells with properties similar to those described above, and $F(ab')_2$ fragments, lacking Fc portion, are unable to cause immunosuppression when they are preincubated with spleen cells. It is possible that this binding is related to the specific suppression caused by 7S antibody molecules.

3256 THE STIMULATORY EFFECT OF TUMOR BEARING UPON THE T- AND B-CELL SUBPOPULATIONS OF THE MOUSE SPLEEN.

(E.) Konda, S. (Coll. Med., U. Florida, Gainesville), Y. Nakao and R. T. Smith. *Cancer Res* 33(10):2247-2256, 1973.

The numbers and certain functions of T- and B-cell subpopulations of the spleen have been assessed in C57BL/6 and CBA mice over the course of bearing transplanted, syngeneic methylcholanthrene fibrosarcomas. The level of proliferative activity in this population increases progressively over the 5- to 8-week course of tumor bearing. The size of the θ-antigen-bearing subpopulation and the T-cell functions of graft-*versus*-host reactivity, *in vitro* responses to phytohemagglutinin, and primary stimulation by alloantigen in one-way mixed leukocyte cultures were found to increase significantly, provided that the data are expressed on the basis of total numbers of cells or degree of total function in the spleen. Numbers of plaque- or rosette-forming cells per spleen also increased over control values, both after direct immunization with sheep erythrocytes and when the primary response of spleen cells to sheep red blood cells was assayed adoptively in an irradiated host. The subpopulation responding to the mitogenic effect of lipopolysaccharide *in vitro* was greatly increased during tumor bearing. The data suggest that both T- and B-cell subpopulations and their functions are not diminished but are augmented significantly by tumor bearing.

3257 CELL SURFACE ANTIGEN AND VIRAL ANTIGEN OF MAREK'S DISEASE VIRUS IN LYMPHOID CELLS OF THE SPLEEN FROM CHICKENS INOCULATED WITH VIRULENT STRAINS.

(E.) Akiyama, Y. (Res. Inst. Microbiol. Dis., Osaka U., Japan), M. Naito and S. Kato. *Biken J* 16(2):91-94, 1973.

A preliminary report on a state of dissociation of antigen expression in lymphoid cells from chicks infected with Marek's disease virus (MDV) is presented. A virulent strain of MDV, BC-1, or JM, was inoculated intramuscularly into one-day-old chicks. After appropriate periods, viral antigen (VA) from various organs of the chicks was examined by the direct fluorescent antibody technique. Sera for this technique were obtained from chickens infected with MD and the standard ammonium sulfate method was used for separation of globulins from the sera. Material was labeled with fluorescein isothiocyanate. Conjugates were subjected to chromatography on DEAE-cellulose. Normal tissues and lymphoid cells were not stained with these labeled sera. Some cells with VA were found in the bursa of Fabricius (BF), spleen and thymus. In some chicks, a very few cells with VA were also found in the liver at an early period. Some lymphoid cells in the spleen of these chicks showed both VA and cell surface antigen (CSA) of MDV. However, 3 wk or more after infection, some lymphoid cells still showed CSA of MDV but no cells with VA of MDV could be seen. When cells with CSA were cultured for 11-48 hr, the VA of MDV reappeared and CSA bearing cells increased in number. The CSA and VA of lymphoid cells constituting a lymphoma of MD were examined. Most of the cells had neither CSA nor VA, but some cells had both VA and CSA. On treatment with concanavalin A at a concentration of 50 μg/ml these lymphoid cells in tumors were always agglutinated clearly, while normal lymphocytes were not. This work demonstrates a state of dissociation of CSA from VA in lymphoid cells *in vivo* which suggests that MDV may be intrinsically involved in chicken lymphoid cells.

3258 A COMPARATIVE STUDY OF THE INTERACTION BETWEEN CONCANAVALIN A AND MITOCHONDRIA FROM NORMAL AND MALIGNANT CELLS.

(E.) Glew, R. H. (U. Pittsburgh Sch. Med., Pa.), J. B. Zatzkin and S. C. Kayman. *Cancer Res* 33(9):2135-2141, 1973.

The concanavalin A (con A)-binding capacity of mitochondria isolated by sucrose density gradient methods from host livers, from Morris hepatomas 7800 and 7777, and mouse myeloma MOPC-46 were compared. Con A binding to the various mitochondrial preparations was quantitated using an isotopic assay procedure in which con A-acetyl-^{3}H is used and by a spectrophotometric agglutination method using native con A. Tumor-derived mitochondria bound significantly less con A than did the corresponding host liver preparations, indicating an alteration in the glycoproteins of tumor mitochondria. Triton X-100-treated hepatoma 7800 and 7777 mitochondria were capable of binding only 50 and 70% as much con A-acetyl-^{3}H, respectively, when compared with mitochondria isolated from the host liver controls. The data suggest either that there is a decrease in the concentration of glycoproteins in tumor mitochondria or that there are qualitative changes in the structure of their oligosaccharide side chains. The altered activity of tumor mitochondria to con A cannot be accounted for on the basis of differential growth rates of tumor and host liver tissue since mitochondria isolated from regenerating rat liver did not differ from normal rat liver mitochondria in their con A-binding properties.

3259 THE POSSIBLE PRESENCE OF RIBONUCLEIC ACID-DEPENDENT DEOXYRIBONUCLEIC ACID POLYMERASE IN THE IMMUNE RESPONSE.

(E.) Kurashige, S. (Sch. Med., Gunma U., Maebashi, Japan) and S. Mitsuhashi. *Japan J Microbiol* 17(2):105-109, 1973.

Immune ribonucleic acid (*i*RNA) preparations were extracted from spleens of rabbits immunized either with *Salmonella* flagella or f_2 phage. The enzyme extract prepared from popliteal lymph node cells of immunized rabbits induced DNA synthesis in a cell-free system in the presence of *i*RNA. The enzyme activity was demonstrated only after stimulation with a corresponding antigen and was inhibited by large amounts of rifampicin, rifampicin derivatives (known inhibitors of reverse transcriptase) or by treatment of the *i*RNA with ribonuclease, but neither actinomycin D nor mitomycin C affected it. Pretreatment of *i*RNA with deoxyribonuclease did not affect the enzyme activity. This DNA polymerase was immunologically specific and could induce DNA synthesis only by using the corresponding *i*RNA but not by using either normal RNA or *i*RNA prepared against another antigen. This new DNA presumably represents an intermediate in the antibody formation process specified by this enzyme using *i*RNA as a template. By contrast, the RNA dependent DNA polymerase demonstrated in oncogenic viruses, in human leukemic cells and in human lymphocytes stimulated by phytohemagglutinin, were able to copy even non-specific RNA(s) such as viral RNA of a different species, rat liver RNA or poly A.

3260 HIGH LEVEL OF ALPHA-FETOPROTEIN IN SERA OF ADULT MICE.

(E.) Pihko, H. (Dept. Serol. Bacteriol., U. Helsinki, Finland) and E. Ruoslahti. *Int J Cancer* 12(2):354-360, 1973.

Mouse α-fetoprotein (AFP) was purified by making use of its immunological cross-reaction with rabbit AFP. A radioimmunoassay, developed for mouse AFP, was used to measure AFP concentrations in the sera of adult mice of inbred IAH, C58B1, C3H, and CBA strains. Basal AFP levels were in the range of 30-350 μg/ml, which is about 10 times higher than has been reported for humans and rats. An association seems to exist between the high basal AFP levels and susceptibility of mice to spontaneous hepatomas. The mean AFP level was significantly higher in IAH and C57B1 males than in females of the same strains, who have a lower incidence of hepatoma.

3261 DELAYED CUTANEOUS HYPERSENSITIVITY REACTIONS TO EXTRACTS OF MALIGNANT AND NORMAL HUMAN BREAST CELLS.

(E.) Alford, C. (George Washington U. Med. Ctr., Washington, D.C.), A. C. Hollinshead and R. B. Herberman. *Ann Surg* 178(1):20-24, 1973.

Six of 13 patients with mammary cancer had a delayed hypersensitivity skin reaction to autologous and allogeneic mammary cancer membrane extracts but not to normal breast membranes derived from either the same mastectomy specimen or from breast tissue of patients with fibrocystic disease. Three of these patients had disseminated mammary cancer and their clinical course indicated that host reponses were significant. Five patients with other types of malignancies did not react to any of these preparations. This suggests that a common tumor associated membrane antigen is being detected in the mammary cancer patients. However, the low frequency of positive responses makes diagnostic application unlikely. Autologous or allogeneic solubilized fractions from mammary cancer membranes also elicited a delayed hypersensitivity skin reaction in a higher percentage of mammary cancer patients than in patients with other types of cancer. However, the soluble fractions derived from either normal breast of the mastectomy specimen or from fibrocystic disease were just as reactive in mammary cancer patients or patients with benign breast disease as the soluble tumor fractions. Therefore, the ability to diagnose or prognosticate the outcome of mammary cancer with skin tests with soluble fractions also does not appear feasible. The results suggest that the reactions observed with membrane preparations were tumor specific but that those with soluble fractions detected only organ specific antigen.

3262 EFFECTS OF HUMORAL AND CELLULAR IMMUNE REACTIONS ON TUMOR TARGET CELLS *IN VITRO*. THEIR DEPENDENCE ON ISO-, ALLO- OR XENOGENIC ORIGIN OF LYMPHOCYTES AND ANTISERA.

(E.) Warnatz, H. (U. Hosp., Erlangen, Germany), F. Scheiffarth and J. Rüters. *Z Krebsforsch* 79(3):176-184, 1973.

The effects of humoral and cellular immune reactions or tumor target cells and their dependance on the iso-, allo- or xenogenic origin of lymphocytes and antisera were studied. Lymph node cells and antisera of isogenic origin were obtained from AKR-mice immunized to Ehrlich ascites tumor cells (EAT-cells). Allogenic antisera to EAT-cells were produced in CB57B1 mice, and xenogenic antisera in rabbits. The EAT-cell specific immune reactions of antisera and lymph node cells were measured 1) by a cytotoxic test using ^{51}Cr-labeled EAT-cells as target cells and 2) by inhibition of ^{3}H-thymidine incorporation into nuclear DNA of EAT-cells. Lymph node cells from immunized AKR-mice in combination with xenogenic as well as allogenic antisera show an additive cytotoxic effect on EAT-cells. The cytotoxic activity of lymph node cells is reduced by the antiserum to EAT-cells produced in AKR-mice. The specificity of the antisera was demonstrated by absorption experiments. Lymph node cells from immunized AKR-mice and xenogenic as well as allogenic antisera to EAT-cells inhibit the *in vitro* DNA synthesis of EAT-cells significantly. No additive effect of lymph node cells and the antisera was observed. The inhibitory effect of AKR-lymph node cells on the DNA synthesis of EAT-cells is abrogated by antiserum produced in AKR-mice, particularly after long period cultivation. The specificity of these enhancing antibodies was demonstrated in absorption experiments. Lymph node cells of non-sensitized mice do not impair the tumor cell growth; the inhibitory effect of xeno- and allogenic antisera to EAT-cells is reduced by the non-sensitized lymph node cells. The data indicate that the additive effect or the enhancement by humoral and cellular immune reactions is dependent on the origin of lymph node cells and antisera.

3263 IMMUNE RESPONSE TO ADENOVIRUS 12-INDUCED TUMOUR ANTIGENS, AS MEASURED *IN VITRO* BY THE MACROPHAGE MIGRATION INHIBITION TEST.

(E.) Rees, R. C. (Dept. Med. Microbiol., U. Sheffield, England) and C. W. Potter. *Eur J Cancer* 9(7): 497-502, 1973.

Immunity produced by cell-free tumor extracts against challenge with adenovirus 12-induced tumor cells was shown to be cell mediated by adaptive transfer of spleen cells from immunized animals but could not be demonstrated by *in vitro* colony inhibition tests. In the present work cellular immunity to an adenovirus 12-induced CBA mouse tumor was demonstrated *in vitro* by the macrophage migration inhibition test. The specific production of migration inhibition factor (MIF) was demonstrated for sensitized spleen cells incubated for 3 days at 37 C in the presence of cell-free tumor extract, and was detected by the ability of supernatant from such cultures to inhibit the migration of guinea pig peritoneal macrophages. In addition, cellular immunity was demonstrated in mice bearing large transplantable tumors, 28 days following tumor transplant. Spleen cells from tumor-bearing mice taken 14 and 24 days after tumor transplant did not, however, produce MIF in the presence of tumor extract. Serum from mice bearing large tumors was shown to inhibit the production of MIF from sensitized spleen cells incubated with tumor extract. The nature of inhibitory factors in such serum and their mechanism of action is not yet known, but it is likely that they are either antibody or antigen-antibody complexes.

3264 ISOLATION AND IMMUNOCHEMICAL CHARACTERIZATION OF RABBIT 7S ANTI-IgG WITH RESTRICTED HETEROGENEITY. *(E.)* Bokisch, V. A. (Rockefeller U., New York, N.Y.), J. W. Chiao and D. Bernstein. *J Exp Med* 137(6):1354-1368, 1973.

7S anti-IgGs were isolated from four rabbit antistreptococcal antisera by the use of immunoabsorbent columns. The isolated proteins were of restricted molecular heterogeneity; they formed a monodisperse band on microzone electrophoresis and had a limited number of light chain bands when analyzed on urea polyacrylamide gel. The binding site of the 7S anti-IgGs was detected in the $F(ab')_2$ portion of the molecule. The binding site has antibody specificity for the Fc portion of IgG. For one 7S anti-IgG the combining site on the Fc portion could further be defined. A pepsin fragment of Fc, described as Pep-III', was a potent inhibitor of the coprecipitation of 7S anti-IgG with antigen-antibody complexes. An idotypic cross-reaction was detected between the 7S and 19S anti-IgGs isolated from the same rabbit with anti-idiotype sera prepared in guinea pigs. This idiotypic specificity was not detected in the 7S anti-IgGs of 20 other rabbits.

3265 SURFACE CHANGES IN TRANSFORMED CELLS DETECTED BY LECTINS. *(E.)* Burger, M. M. (Dept. Biochem. Sci., Princeton U., N.J.). *Fed Proc* 32(1):91-101, 1973.

The use of plant agglutinins in assessing surface differences in transformed cells is reviewed including discussion of such topics as available lectins, lectin receptor sites and their respective topography, a quantitative assay for cell agglutination, the possible significance of surface alterations detected with lectins, and the possible consequences of these surface alterations for growth control. Increased agglutinabilities with lectins have been observed for many transformed tissue cultures as well as native tumor cells when compared with their normal parent cells. Only some lectins, however, are able to distinguish between transformed and untransformed cell surfaces and even these lectins are not able to detect differences between all pairs of normal and transformed cells. The positive correlation between increased lectin agglutinability and several aspects of transformation may be summarized as follows: 1) some 40 to 60 lines of transformed cells have been found to agglutinate well with various lectins; 2) tumor cells that were selected for reversion to good density-dependent inhibition of growth lose their agglutinability; 3) tumor cells that were selected for a loss of agglutinability regain to some extent their normal growth control; 4) conditionally nontransforming viral mutants and temperature sensitive mutants cannot achieve the agglutinable state under nonpermissive conditions; and 5) temperature-sensitive host mutants that lose some of their transformed properties at the nonpermissive temperatures lose their agglutinability simultaneously.

3266 GLOMERULONEPHRITIS ASSOCIATED WITH IMMUNE COMPLEX DEPOSITS AND VIRAL PARTICLES IN SPONTANEOUS MURINE LEUKEMIA. AN ELECTRON MICROSCOPIC STUDY WITH IMMUNOFLUORESCENCE. *(E.)* Pascal, R. R. (Coll. Phys. Surg., Columbia U., New York, N.Y.), M. N. Koss and R. L. Kassel. *Lab Invest* 29(2): 159-165, 1973.

Fifty-four AKR mice, including treated and untreated leukemic and aleukemic animals, showed a 65 percent incidence of subendothelial glomerular basement membrane deposits in addition to other glomerular alterations. Immunofluorescence detected the presence of IgG and β1C in locations corresponding to the deposits seen by electron microscopy. There was no correlation between the incidence of deposits and leukemic infiltration of the kidney, response to antitumor therapy, or presence or absence of overt leukemia. There was significant correlation, however, between the presence of deposits and the presence of C-type viral particles in those glomeruli. It is proposed that the combination of Gross leukemia virus and antibody within the glomerular basement membrane is one mechanism producing the nephritis of AKR mice.

3267 TESTING BY THE "PAIRED-LABEL" ANTIBODY BINDING TECHNIQUE FOR FELINE LEUKEMIA VIRUS-INDUCED CELL SURFACE ANTIGENS (FeLV-CSA) ON THE SURFACE OF HUMAN RHABDOMYOSARCOMA CELLS RELEASING RD-114 VIRUS. *(E.)* Boone, C. W. (Natl. Cancer Inst., Bethesda, Md.), E. C. Church and R. M. McAllister. *Virology* 55(1):157-162, 1973.

It has been questioned whether the C-type virus (RD-114) found in a human rhabdomyosarcoma line RD after heterotransplantation into fetal cats originated from RD or represented a passenger feline leukemia virus (FeLV). In the present work, studies using the sensitive and quantitative paired-label antibody binding technique gave no evidence that RD cells productively infected with RD-114 virus possessed feline leukemia virus-induced cell surface antigen (FeLV-CSA). The paired-label technique readily detected FeLV-CSA on RD cells intentionally infected with the feline leukemia virus. In addition, rabbit antibody against RD-114 virus which bound to the surface of RD-114 cells did not bind to the surface of FeLV-CSA-positive feline leukemia cells. Electron microscopy confirmed that a type-C virus was present in the RD cells studied. These findings support other biological studies indicating that the RD-114 virus is an endogenous feline type-C virus that is distinct from previously described feline type-C viruses.

3268 DETECTION OF HAMSTER TYPE-C VIRUS ANTIGENS BY THE PASSIVE HEMAGGLUTINATION TECHNIQUE. *(E.)* Hatch, G. G. (Baylor Coll. Med., Houston, Tex.), K. J. McCormick and J. J. Trentin. *J Natl Cancer Inst* 51(2):519-524, 1973.

To provide a simple means for studying the distribution of Syrian hamster type-C antigens in various tissue extracts, an assay was developed using the passive hemagglutination (PHA) and passive hemagglutination inhibition (PHAI) techniques previously worked out for antigens of murine and feline leukemia viruses and mammary tumor virus. These methods, unlike complement fixation, would not be troubled by anticomplementarity of sera. Rabbits immunized with density gradient purified type-C virus (from plasma of Syrian hamsters bearing the D-9 lymphoma) developed antibodies that specifically hemagglutinated sheep erythrocytes sensitized with the hamster virus. The PHA reaction was inhibited by density gradient purified type-C particles, tumor extracts, plasma, and organ extracts from hamsters bearing the D-9 lymphoma. Inhibition of passive hemagglutination was not obtained with density gradient material from normal hamster plasma. Both ether-soluble external viral antigens and water-soluble internal viral components inhibited the agglutination of sensitized cells. Extracts of normal Syrian hamster tissues or hamster embryos harvested during the last five days of gestation exhibited low levels of antigenicity. After sedimentation of type-C virions from infected plasma, large amounts of "soluble" viral antigens remained. These antigens could be removed by prolonged centrifugation at 115,000 X g. In some experiments, low levels of antigenicity were demonstrated in sera of newborn or weanling hamsters given injections of hamster type-C virus. The PHAI technique was more applicable to extracts of tissues than to small amounts of materials from sources *in vitro*, such as supernatants from cultured D-9 cells. The antigen(s) obtained by homogenization of tissue was sensitive to heat but not to freeze-thawing.

3269 RELEASE OF ANTIGEN-SPECIFIC IMMUNOGLOBULIN FROM CYTOTOXIC EFFECTOR CELLS AND SYNGENEIC TUMOUR IMMUNITY *IN VITRO*. *(E.)* Röllinghoff, M. (Royal Melbourne Hosp., Victoria, Australia), H. Wagner, R. E. Cone and J. J. Marchalonis. *Nature* [*New Biol*] 243(122):21-23, 1973.

Cortisone-resistant thymus (T) cells were purified from cortisone-treated Balb/c mice and were sensitized *in vitro* to a tumor-associated transplantation antigen (TATA) of a syngeneic plasma tumor cell line (HPC) 108. Following ^{125}I labeling of the activated T cell surface proteins, the proteins were released from the cells by incubation in culture medium at 37 C and purified by dialysis. They were identified as IgM by polyacrylamide gel electrophoresis after precipitation with specific antisera. Twenty to 25% of the precipitated ^{125}I labeled surface protein from activated T cells would bind to HPC 108 cells. This binding was eliminated if the immunoglobulin was removed by specific coprecipitation before the binding assay. Cell-surface immunoglobulin from non-activated T cells did not bind to HPC 108 cells. These results suggested that membrane bound immunoglobulin may function as the receptor for antigen on TATA activated T cells.

3270 THE EFFECTS OF IMMUNE DEPRESSION ON CHEMICAL CARCINOGENESIS IN THE RAT. I. REGIONAL LYMPHADENECTOMY. *(E.)* Bolton, P. M. (Dept. Surg., U. Queensland, Brisbane, Australia). *Oncology* 27(5):430-436, 1973.

Comparison was made between groups of female rats which underwent unilateral axillary and inguinal lymphadenectomy, bilateral lymphadenectomy, or sham unilateral lymphadenectomy 5 days before the first of 4 doses of 7, 12-dimethylbenz(A)anthracene (DMBA). The results showed that among the different groups the number of animals developing tumors were similar, there were no significant differences between the mean tumor/animal ratios, the histological patterns and sizes of tumors were closely comparable, differences in site of tumors were not statistically significant, and metastasis was not found in any animal. A comparison made between the lymphadenectomized and normal sides of animals undergoing unilateral axillary and inguinal lymphadenectomy showed that statistical significance was not reached on any of the parameters measured. It is suggested that immunological concepts should not be invoked to justify sparing possibly involved regional lymph nodes during cancer surgery.

3271 BLOCKING AND REDISTRIBUTION ("CAPPING") OF ANTIGEN RECEPTORS ON T AND B LYMPHOCYTES BY ANTI-IMMUNOGLOBULIN ANTIBODY. *(E.)* Roelants, G. (Basel Inst. Immunology, Switzerland), L. Forni and B. Pernis. *J Exp Med* 137(4):1060-1077, 1973.

In an effort to distinguish the receptors on T and B lymphocytes, antigen-binding of the two types of cells was studied by combined autoradiography and immunofluorescence. Mouse spleen lymphocytes binding the antigens, [^{125}I]MSH or [^{125}I]TIGAL, were incubated with rhodamine-labeled anti-Ig reagents or with a rhodamine-labeled IgG fraction of anti-θ serum. B cells could thus be identified as Ig+ or θ-, T cells as Ig- or θ+. It was found that 20% (1-2 months after priming) to 30% (3.5-4 months after priming) of the antigen-binding cells were T cells. The range of antigen molecules bound by B and T cells was similar; binding of antigen to B and T cells was inhibited by polyvalent anti-Ig, anti-μ, or anti-L reagents. Binding to T cells was more readily inhibited than to B cells. Normal rabbit serum, antimouse lymphocyte serum, or anti-θ did not inhibit antigen binding. When Ig at the surface of B cells was induced, by noninhibiting concentrations of anti-Ig reagents, to redistribute into polar caps and the cells subsequently exposed to [^{125}I]antigen under noncapping conditions, the [^{125}I]antigen silver grains were distributed in caps superimposed on the Ig fluorescent cap. Of crucial importance, antigen was found in cap in the same proportion of T cells as B cells. Significant capping of antigen receptors was not induced in B or T cells with normal rabbit serum or by anti-Ig reagents absorbed with mouse Ig. The main conclusions of this series of experiments using direct visualization of antigen-binding B and T lymphocytes are that T cells have antigen-specific receptors, probably of IgM nature, and that the number of these receptors appears to range in the order of thousands.

3272 CHANGE IN THE NUMBER OF ANTIBODY-PRODUCING CELLS IN THE SPLEEN OF MICE DURING CARCINOGENESIS.

(Rus.) Iakimenko, L. V. (Inst. Oncol. Problems, Kiev, USSR) and Iu. A. Umanskii. *Vopr Onkol* 19(7):49-52, 1973.

Changes in the number and functional activity of cells producing 19S and 7S antibodies were investigated in the spleens of 2-2.5-month-old BALB/c mice given methylcholanthrene (MC; 1 mg/mouse in 0.1 ml peach oil). Within 30-90 days after injection of MC, mice received an i.p. injection of 5 x 10^8 sheep RBC and were killed 4 days later. The Jerne reaction showed large numbers of cells producing 19S antibodies were present in the spleens of untreated controls. Progressive decreases occurred in the number of these cells in animals given MC: after one month they had decreased by 54.2% and after two months, by 70.3%. However, the number of cells producing 19S antibodies was 65-66% that of untreated controls in mice who failed to develop tumors within six months. In mice who had been given MC, progressive, but less pronounced, decreases occurred in cells producing 7S antibodies. By measuring the mean diameter of the hemolysis zone around the cells, the functional activity of cells producing 19S antibody was found to decrease progressively during this study, while the activity of cells producing 7S antibody was not impaired by MC.

3273 EXPRESSION OF FETAL ANTIGENS AND TUMOR-SPECIFIC ANTIGENS IN SV40-TRANSFORMED CELLS. I. SEROLOGICAL ANALYSIS OF THE ANTIGENIC SPECIFICITIES.

(E.) Ting, C.-C. (Natl. Cancer Inst., Bethesda, Md.), J. R. Ortaldo and R. B. Herberman. *Int J Cancer* 12(2):511-518, 1973.

The fetal antigens expressed in a variety of tumors have previously been shown to be different from tumor-specific antigens. In the present study the expression of fetal antigens in various SV40-transformed mouse cells and the correlation of this expression with the function of the viral genome were investigated. The antisera used were antifetal (AFS) and anti-SV40 tumor (ASVS). The test cells were standard SV40-transformed lines, "cryptic" transformants (cells exposed to SV40 and containing the genome but not expressing its function), spontaneously transformed lines and others. The reaction between antisera and cells was measured by absorption as detected by an isotopic antiglobulin technique. The ASVS reacted only with SV40-transformed cells, not with "cryptic" transformants or spontaneously transformed cells. The AFS reacted with all the cell lines; however, the lines did not all contain the same antigenic specificities, at least three different ones being expressed. Thus, the expression of fetal antigens was not directly related to the function of the SV40 genome.

3274 AN IMMUNOLOGICAL STUDY OF HUMAN BRAIN TUMORS CONCERNING THE BRAIN SPECIFIC PROTEINS S-100 AND 14.3.2.

(E.) Haglid, K. (Inst. Neurobiol., U. Göteborg, Sweden), C.-A. Carlsson and D. Stavrou. *Acta Neuropathol (Berl)* 24(3):187-196, 1973.

The distribution of the brain specific proteins S-100 and 14.3.2 was studied in 80 human brain tumors by means of immunofluorescence and immunoprecipitation techniques. Both S-100 and 14.3.2 proteins were shown to be present within nerve cells of human frontal lobes and in the cells of such gliomas as glioblastomas, astrocytomas, spongioblastomas and acoustic neurinomas and some oligodendrogliomas. Although the S-100 protein has been shown to be synthesized in glial cells, experiments indicate that it can be bound to different subcellular structures, not extractable with aqueous solutions. This fact could explain the presence of S-100 also in cells which are not capable of its synthesis, such as mouse neuroblastoma cells in tissue culture. The 14.3.2 protein, but not the S-100 protein, could be demonstrated in ependymomas, plexus papillomas and chromophobe adenomas of the pituitary and in three polymorphous oligodendrogliomas. Neither protein was found to be present in necrotic areas of brain tumors or in meningiomas and brain metastases.

3275 HETEROGENEITY OF THE CARCINOEMBRYONIC ANTIGEN.

(E.) Coligan, J. E. (City Hope Natl. Med. Ctr., Duarte, Cal.), P. A. Henkart, C. W. Todd and W. D. Terry. *Immunochemistry* 10(9):591-599, 1973.

Fractionation of carcinoembryonic antigen (CEA) by ECTEOLA-cellulose chromatography and by isoelectric focusing indicated that the antigen was heterogeneous. The fractions obtained were variable in their content of sialic acid, amino sugars, and amino acids. All fractions were antigenically active and exhibited immunologic identity with respect to one another. Removal of the sialic acid with neuraminidase altered the electrophoretic migration and slightly decreased the heterogeneity of CEA as revealed by isoelectric focusing, but did not alter the activity of the material in the radioimmune assay. Thus, while the variation in sialic acid content is responsible in part for the electrophoretic heterogeneity of CEA, other sources of heterogeneity must also exist. It is possible that some heterogeneity may be found in the protein structure or be caused by alterations arising during the isolation procedure.

3276 DETECTION OF A SPECIFIC SURFACE ANTIGEN(S) IN ADENOVIRUS TYPE 12-TRANSFORMED CELLS BY FLUORESCENT ANTIBODY TECHNIQUE.

(E.) Hamada, C. (Inst. Virus Res., Kyoto U., Japan), S. Nakajima and H. Uetake. *Jap J Microbiol* 17(4):297-302, 1973.

A surface antigen(s) was detected in adenovirus type 12 (Ad12)-transformed cells by fluorescent antibody technique. The antigen was revealed by antisera of mice and rabbits immunized with the virus, and by those of rabbits immunized with conditioned medium from cell cultures carrying the surface antigen. The antigen was specific for the Ad12-transformed cells, since it was found only in the Ad12-transformed cells regardless of the animal species and not in any other cells, including SV40- and polyoma virus-transformed cells. The antigen was distinct from the Ad12-specific viral and T antigens, since the sera of rabbits immunized with the conditioned medium contained little or no detectable antibodies to the latter two antigens. On the contrary, the surface antigen was not detected by the above antisera in any tumor cells induced by Ad12, and no detectable antibody to the surface antigen was produced in mice immunized with the tumor cells.

3277 PRIMARY IMMUNODEFICIENCY DISEASES AND CANCER: THE IMMUNODEFICIENCY-CANCER REGISTRY. *(E.)* Kersey, J. H. (Dept. Pathol., U. Minnesota, Minneapolis), B. D. Spector and R. A. Good. *Int J Cancer* 12(2):333-347, 1973.

Data from the newly established Immunodeficiency-Cancer Registry show 151 tumors in 145 patients with primary immunodeficiency syndromes, including: sex-linked (Bruton's) agammaglobulinemia, 6 patients; severe combined system immunodeficiency, 9 patients; Wiskott-Aldrich syndrome, 24 patients; ataxia-telangiectasia, 52 patients; common variable (late onset) immunodeficiency, 41 patients; isolated IgA deficiency, 7 patients; and isolated IgM deficiency, 6 patients. Most of the primary immunodeficiency syndromes occur in children, resulting in early death from severe infections; however, these individuals have a 2-10% chance of developing cancer during their short life span. Tumors are divided into major histologic types: lymphoreticular, 58% of total; leukemias, 17% of total; epithelial, 18% of total; mesenchymal, 3% of total; and nervous system, 4% of total. Data are reported on 14 families in which 12 siblings had the same tumors and identical primary immunodeficiency diseases. The finding that the majority of tumors are lymphoreticular or leukemias is consistent with the hypothesis that individuals with primary immunodeficiency syndromes have intrinsic abnormalities of the lymphoid system which result in increased frequency of malignant transformation and inability to eliminate transformed cells. Further reporting of tumors is urged.

3278 COMPLEMENT FACTORS AND THE GROWTH OF EHRLICH'S CARCINOMA. *(E.)* Thunold, S. (Dept. Pathol., U. Bergen, Norway), V. Horseide and F. Hartveit. *Acta Pathol Microbiol Scand* [*B*] 81(3):373-381, 1973.

3279 CELLULAR IMMUNITY AND SERUM FACTORS IN PLASMACYTOMAS OF MICE. *(Fr.)* Lespinats, G. (Sci. Res. Inst. Cancer, Villejuif, France), M.-F. Poupon and J.-P. Kolb. *Bull Cancer (Paris)* 59(4): 353-362, 1972.

3280 THE AUTOIMMUNE ACTIVITY OF LEUKEMIA PATIENTS. *(Ger.)* Richter, R. (Polish Acad. Sci., Wroclaw), S. Kotlarek-Haus, A. Harlozinska and W. Brodzka. *Folia Haematol (Leipz)* 99(2/3):144-151, 1973.

3281 ROUS VIRUS-INDUCED SURFACE ANTIGEN IN HAMSTER CELLS: SENSITIVITY OF A DETECTION METHOD BASED ON HUMORAL CYTOTOXICITY. *(Fr.)* Bataillon, G. (Curie Fdn., Orsay, France). *C R Acad Sci* [*D*] *(Paris)* 276(11):1797-1800, 1973.

3282 THE MECHANISM OF THE INHIBITORY ACTION OF *CORYNEBACTERIUM PARVUM* ON THE DEVELOPMENT OF MALIGNANT SYNGENEIC TUMORS. *(Fr.)* Halpern, B. (Natl. Inst. Hlth. Med. Res. (Immunol. Inst., Paris, France), A. Fray, Y. Crepin, O. Plactica, L. Sparros, A. M. Lorinet and A. Rabouroin. *C R Acad Sci* [*D*] *(Paris)* 276(12):1911-1915, 1973.

3283 ABO SYSTEM TRANSPLANTATION ANTIGEN ALTERATIONS PRODUCED BY NORMAL LYMPHOCYTES OR BY PARALYMPHOBLASTS DERIVED FROM LEUKEMIC PATIENTS. *(Fr.)* Mousseron-Canet, M. (Natl. Sch. Chem., Montpellier, France), R. Magous, C. Lecou and C. Levallois. *C R Acad Sci* [*D*] *(Paris)* 276(13):2109-2112, 1973.

3284 THE MEANING OF THE ALTERED EXPRESSION OF CERTAIN ERYTHROCYTIC, LEUKOCYTIC AND SERUM ALLOTYPES OCCURRING IN MALIGNANT HEMATOLOGIC DISEASES. *(Fr.)* Ducos, J. (Natl. Inst. Hlth. Med. Res., Toulouse, France), E. Ohayon, M. Blanc and Y. Marthy. *Bull Acad Natl Med (Paris)* 156(16/17):511-527, 1972.

3285 CYTOCHEMICAL AND IMMUNOLOGIC FINDINGS IN CHRONIC LYMPHOID LEUKEMIA. *(Fr.)* Westerhausen, M. (Freiburg U. Dept. Clin. Immunopathol., W. Germany). *Nouv Rev Fr Hematol* 13(3):398-400, 1973.

3286 PROLYMPHOCYTIC LEUKAEMIA OF B AND T CELL TYPE. *(E.)* Catovsky, D. (Royal Postgraduate Med. Sch., London, England), A. Okos, E. Wiltshaw, J. Galetto, D. A. Galton and G. Stathopoulos. *Lancet* 7823:232-234, 1973.

3287 THE DETECTION OF CARCINOEMBRYONIC ANTIGEN IN WHOLE SERUM FROM PATIENTS WITH MALIGNANT AND NONMALIGNANT DISEASE. *(E.)* MacSween, J. M. (Walter and Eliza Hall Inst. Med. Res., Melbourne, Australia), N. L. Warner and I. R. MacKay. *Clin Immunol Immunopathol* 1(3):330-345, 1973.

3288 EFFECT OF PHYTOHEMAGGLUTININ R AND CONCANAVALIN A ON EMBRYONIC CELLS DURING EMBRYOGENESIS. *(Fr.)* Aubery, M. (Fac. Med., Paris, France) and R. Bourrillon. *C R Acad Sci (Paris)* 276(24):3187-3190, 1973.

3289 CYTOTOXICITY AGAINST HUMAN LEUKAEMIC CELLS. I. DEMONSTRATION OF ANTIBODY-DEPENDENT LYMPHOCYTE KILLING OF HUMAN ALLOGENEIC MYELOBLASTS. *(E.)* Hersey, P. (Radcliffe Infirmary, Oxford, England), I. C. M. MacLennan, A. C. Campbell, R. Harris and C. B. Freeman. *Clin Exp Immunol* 14(2):159-166, 1973.

3290 PERIPHERAL BLOOD T- AND B-LYMPHOCYTES IN PATIENTS WITH LYMPHOMA AND ACUTE LEUKEMIA. *(E.)* Chin, A. H. (Bernalillo Cty. Med. Ctr., Alburquerque, New Mexico), J. H. Saiki, J. M. Trujillo and R. C. Williams. *Clin Immunol Immunopathol* 1(4):499-510, 1973.

3291 THE MEASUREMENT AND MANIPULATION OF HEMOLYTIC COMPLEMENT LEVELS IN TUMOR BEARING $C_{57}BL/6$ MICE. *(E.)* Drake, W. P. (Natl. Cancer Inst., Baltimore, Md.), P. C. Ungaro and M. R. Mardiney, Jr. *Biomedicine* 18(4):284-289, 1973.

3292 ALLOGENEIC MARROW GRAFTING IN ACUTE LYMPHOID LEUKEMIA DURING MARROW REMISSION. *(E.)* Buckner, C. D. (U. Washington Sch. Med., Seattle), F. L. Johnson, E. D. Thomas, R. Storb, R. A. Clift, R. L. Chard, D. D. Funk, P. Neiman and A. Fefer. *Biomedicine* 18(4):295-303, 1973.

3293 MIXED LYMPHOCYTE REACTIONS: ABSENCE OF STIMULATION BY IRRADIATED ALLOGENEIC CELLS. *(E.)* Gebhardt, B. M. (Coll. Med., U. Florida, Gainesville). *Cell Immunol* 8(2):290-298, 1973.

3294 THE MACROPHAGE ELECTROPHORETIC MIGRATION TEST IN CANCER. *(E.)* Goldstone, A. H. (U. Edinburgh, Scotland), L. Kerr and W. J. Irvine. *Clin Exp Immunol* 14(3):469-472, 1973.

3295 LYMPHORETICULAR SARCOMAS IN IMMUNO-DEFECT MICE. *(Ger.)* Grundmann, E. (Inst. Pathol. U. Munster, W. Germany) and H. P. Hobik. *Z Krebsforsch* 79(11):298-303, 1973.

3296 STANDARDIZATION OF LYMPHOCYTE TRANSFORMATION TO PHYTOHAEMAGGLUTININ. *(E.)* Yamamura, M. (Westminster Hosp. Med. Sch., London, England). *Clin Exp Immunol* 14(3):457-467, 1973.

3297 ON INTESTINAL TUMORS INDUCED BY BENZIDINE IN RATS. *(Rus.)* Pliss, G. B. (N. N. Petrov Res. Inst. Oncology USSR Ministry Public Hlth., Leningrad), N. J. Volfson and M. I. Jogannsen. *Vopr Onkol* 19(6):75-79, 1973.

3298 RADIOIMMUNOLOGICAL DETERMINATION OF CIRCULATING CARCINOEMBRYONIC ANTIGEN:IMMUNOPRECIPITATION FOLLOWED BY PERCHLORIC ACID EXTRACTION OF THE SERUM. *(Fr.)* Martin, F. (Fac. Med., Dijon, France), C. Klepping and J. Guerrin. *Arch Fr Mal App Dig* 61(12):725-733, 1972.

3299 THYMUS-DERIVED LYMPHOCYTE (T-CELL) DEPLETION IN HODGKIN'S DISEASE. *(E.)* Ramot, B. (Chiam Sheba Med. Ctr., Tel Hashomer, Israel), A. Many, M. Biniaminov and E. Aghai. *Isr J Med Sci* 9(5):657-659, 1973.

3300 THE INDUCTION OF IMMUNE TOLERANCE IN MICE DURING RETICULOENDOTHELIAL SYSTEM BLOCKADE. *(E.)* Neill, V. M. (U. Oklahoma Health Sci. Ctr., Oklahoma City) and R. M. Hyde. *J Reticuloendothel Soc* 13(5):449-458, 1973.

3301 SEROLOGICAL INVESTIGATIONS WITH CLOSTRIDIAL SPORES FOR TUMOUR DIAGNOSIS. I. INVESTIGATIONS ON TRANSPLANTABLE TUMOURS IN RATS AND HAMSTERS. *(Ger.)* Kretschmer, H. (Fac. Med. Karl-Marx-U., Leipzig, E. Germany) and A. Grässer. *Arch Geschwulstforsch* 41(1):12-27, 1973.

3302 STUDY OF ANTI-TYROSINASE ANTIBODY PRODUCTION IN IMMUNE RESPONSE IN THE RABBIT. II. STUDY OF CIRCULATING ANTIBODIES. *(Fr.)* Antoine, J.-C. (Pasteur Inst., Paris, France) and S. Avrameas. *Ann Immunol (Inst Pasteur)* 124 C:221-234, 1973.

3303 IMMUNOMORPHOLOGICAL INVESTIGATION OF THYROGLOBULIN IN EXPERIMENTAL TUMORS OF THE THYROID. *(Rus.)* Okulov, V. B. (N. Petrov Res. Inst. Oncology USSR Ministry Public Hlth., Leningrad). *Vopr Onkol* 19(6):69-75, 1973.

3304 RECOMBINATION EXPERIMENTS WITH AN UNUSUAL L-CHAIN RELATED MYELOMA PROTEIN. *(E.)* Wetter, O. (Internol. Med. Clinic. Hosp. Tumor Res., General Coll. Esser, Germany) and R. Pfeiffer. *Acta Haematol* 49(3):166-175, 1973.

3305 LEUKEMOID REACTION IN BALB/c MICE BEARING TRANSPLANTABLE TUMOR. *(E.)* Sendo, F. (Hokkaido U. Sch. Med., Japan), T. Kodama and H. Kobayashi. *Gann* 64(3):297-300, 1973.

3306 THE DISTRIBUTION OF HISTOCOMPATIBILITY ANTIGENS ON T AND B CELLS IN THE GUINEA PIG. *(E.)* Shevach, E. M. (Natl. Insts. Health, Bethesda, Md.), D. L. Rosenstreich and I. Green. *Transplantation* 16(2):126-133, 1973.

3307 EFFECT OF *PROTEUS VULGARIS* LIPOPOLYSACCHARIDE ON RESISTANCE OF MICE INOCULATED WITH TUMOR CELLS SENSITIZED TO EHRLICH CARCINOMA TRANSPLANTATION. *(E.)* Kato, N. (U. Tokyo, Japan), S. Ito, M. Yamazaki and D. Mizuno. *Gann* 64(2):111-120, 1973.

3308 NON-IMMUNOGLOBULIN-BEARING 'B' LYMPHOCYTES IN CHRONIC LYMPHATIC LEUKAEMIA? *(E.)* McLaughlin, H. (Dept. Chem. Path., Westminster Med. Sch., London, England), G. Wetherly-Mein, C. Pitcher and J. R. Hobbs. *Br J Haematol* 25(1):7-14, 1973.

3309 MONOCLONAL GAMMOPATHIES ASSOCIATED WITH MALIGNANT LYMPHOMAS. *(E.)* Palutke, M. (VA Hosp., Allen Park, Michigan) and J. M. McDonald. *Am J Clin Pathol* 59(20):157-165, 1973.

3310 IMMUNOHISTOCHEMICAL STUDY OF GLOMERULAR LESIONS IN RETROPERITONEAL LYMPHOMAS. *(E.)* Gupta, R. K. (St. Catherines Gen. Hosp., Ontario, Canada). *Am J Pathol* 71(3):427-434, 1973.

3311 IMMUNOLOGY AND COLONIC CANCER: FURTHER EVALUATION OF THE RADIOIMMUNOASSAY FOR CARCINOEMBRYONIC ANTIGEN OF THE HUMAN DIGESTIVE SYSTEM AS AN ADJUNCT IN CANCER DIAGNOSIS. *(E.)* Gold, P. (Montreal Gen. Hosp., Montreal, Quebec, Canada), T. Wilson, R. Romero, J. Shuster and S. O. Freedman. *Dis Colon Rectum* 16(5):358-365, 1973.

3312 XENOTRANSPLANTATION OF ESTABLISHED HUMAN ENDOMETRIAL CELLS INTO NEWBORN HAMSTERS. *(E.)* Arata, T. (Okayama U. Med. Sch., Okayama, Japan), Y. Tanaka and T. Tanaka. *Cancer Res* 33(8):1875-1877, 1973.

3313 ISOLATION OF IMMUNOCHEMICALLY PURE IgE FROM SERUM OF E-MYELOMA PATIENT Yu. *(E.)* Stefani, D. V. (Ministry Health Russian Federation, Moscow, USSR), A. I. Gusev and R. A. Mokeeva. *Immunochemistry* 10(8):559-561, 1973.

3314 THE *IN VITRO* EFFECT OF CONCANAVALIN A ON HUMAN LYMPHOCYTES: BLASTIC TRANSFORMATION, DNA SYNTHESIS, DNA CONTENT AND DRY MASS. *(E.)* Pienkowski, M. (Med. Acad., Warsaw, Poland), J. Abramczuk, J. Rowinski and T. Wiwatowski. *Folia Biol* 19(3):161-168, 1973.

3315 PATHOGENESIS OF THE GRAFT VERSUS HOST REACTION IN CHICKEN EMBRYOS. REQUIREMENT OF YOLK SAC DERIVED STEM CELLS FOR THE DEVELOPMENT OF PROLIFERATIVE LESIONS. *(E.)* Walker, K. Z. (Australian Natl. U., Canberra), K. J. Lafferty and G. I. Schoefl. *Aust J Exp Biol Med Sci* 51(part 3):347-355, 1973.

3316 PEMPHIGUS VULGARIS INVOLVING THE CERVIX ASSOCIATED WITH ENDOMETRIAL CARCINOMA OF THE UTERUS: A CASE REPORT WITH IMMUNOFLUORESCENT FINDINGS. *(E.)* Krain, L. S. (Dept. Med. U. California, Los Angeles), L. Rosenthal and V. D. Newcomer. *Int J Dermatol* 12(4):220-228, 1973.

See also:

* (Rev): 3009, 3016, 3022, 3023, 3029, 3031
* (Chem): 3080, 3088
* (Viral): 3117, 3119, 3123, 3135, 3137, 3156, 3159, 3160, 3161, 3166, 3173, 3190, 3192, 3200, 3202, 3225, 3226
* (Path): 3318

3317 TUMORIGENESIS IN OVARIAN IMPLANTS ON THE GREATER OMENTUM IN RATS: FAILURE OF ACCELERATION BY LONG-TERM "DORMANCY". *(E.)* Ber, A. (Beilinson Hosp., Petah-Tikva, Israel). *Endokrinologie* 62(1):49-54, 1973.

Hemiovariectomy and autografting on the greater omentum of a fragment of the ovary or of the whole organ were performed in 49 three wk old and 28 twelve wk old rats. After 6-12 months the second ovary was removed and the animals were killed after an additional period of 3-6 months. All the implants started to grow after the second operation. After comparable time intervals their growth rate was higher in the younger age group than in the older one and their development was also more pronounced when a whole ovary instead of a fragment only was transplanted. In one case only, in a mature rat spayed after 7 months and killed 5 months later, an enormous granulosa tumor developed. The results obtained showed clearly that even a prolonged time of "dormancy" extending to one year did not enhance tumorigenesis in the ovarian implants. During the period of dormancy, an inhibiting factor seems to have been produced by the ovary *in situ* which represses the growth-inducing and tumorigenesis-accelerating influence of gonadotropin.

3318 MORPHOLOGICAL AND IMMUNOLOGICAL STUDIES ON THE HISTOGENESIS OF EXPERIMENTAL BRAIN TUMORS. *(Ger.)* Stavrou, D. (Vet. Fac., Univ. Munich, Germany) and K. G. Haglid. *J Neurol Sci* 20(1):39-50, 1973.

Tumors were induced in the central and peripheral nervous system of adult Sprague-Dawley rats and Chinchilla rabbits by repeated administration of phenyldimethyltriazene s.c. or N-methyl-N-nitrosourea p.o. and i.v. Histological examination of 619 brain tumors induced in this way indicates that gliomatous transformation probably begins with proliferation of the oligodendrocytes. The isomorphous oligodendroglioma which develops may undergo transformation into a pleomorphic glioma. Few primary astrocytomas were found. In some cases simultaneous localized and diffuse proliferation of oligodendrocytes and astroglia occurred in the early stages of tumor development. Glioblastoma-like tumors develop both by progressive dedifferentiation of primarily isomorphous gliomas, i.e. as secondary glioblastomas, and by primary proliferation of abnormal glial cells. Immunofluorescence studies demonstrated that most of these experimental brain tumors contain a glia-specific S_{100} protein, suggesting that the tumors originate from glia.

3319 PRIMARY PULMONARY SCAR CARCINOMAS. *(E.)* Chaudhuri, M. R. (Med. Fac. Rotterdam, Netherlands). *Indian J Med* 61(6):858-863, 1973.

Many primary bronchial carcinomas and even a few metastatic lesions are sometimes associated with the production of much necrosis, fibrosis and scarring, and as such they are often misinterpreted as primary scar carcinomas. The present work was undertaken to determine a proper parameter for defining primary pulmonary scar carcinomas. Out of a total of 632 cases of primary bronchial carcinomas, 21 were primary pulmonary scar carcinomas. Specimens of tissue were obtained at surgery or necropsy. Presence of a dense, hyaline, pigmented scar was frequently observed in conjunction with small primary peripheral pulmonary carcinomas, and it seemed to play a large part in their genesis. Pulmonary scarring might be focal and local. Focal pulmonary scars resulted from healed tuberculous lesions, infarcts and trauma. They were always located subpleurally, while the overlying pleura often showed a depressed, puckered appearance. The alveoli and the bronchioles at the edge of these scars remained intact and showed hyperplastic changes. Neoplastic changes were rather uncommon, and when they did occur, the so-called bronchiolo-alveolar type of adenocarcinomas were formed. Local pulmonary scarring following chronic interstitial pneumonias of any aetiology led often to pulmonary honeycombing and atypical epithelial proliferation. It was speculated that these already proliferating bronchiolo-alveolar cells were further subjected to anoxia, chronic inflammation, constant physical irritation through motion, and, possibly, concentrated chemical carcinogens isolated by the scarring process. The resultant carcinoma may appear anywhere in the spectrum from well-differentiated bronchioloalveolar adenocarcinomas to undifferentiated anaplastic carcinomas, depending upon its ultimate differentiation.

3320 BURKITT'S LYMPHOMA. *(E.)* Whittaker, L. R. (Kenyatta Natl Hosp., Nairobi). *Clin Radiol* 24(3):339-346, 1973.

3321 *IN VITRO* STUDIES OF THE HUMAN MAMMARY GLAND: EFFECT OF HORMONES ON PROLIFERATION IN PRIMARY CELL CULTURES. *(E.)* Flaxman, B. A. (Temple U. Health Sci. Ctr., Philadelphia, Pa.). *J Invest Dermatol* 61(2):67-71, 1973.

3322 IMMUNODEFICIENCY IN SICKLE-CELL ANEMIA. *(E.)* Ringelhann, B. (Semmelweiss U., Budapest, Hungary). *N Engl J Med* 289(6):326-327, 1973.

3323 "PRELEUKEMIA." A MYELODYSPLASTIC SYNDROME OFTEN TERMINATING IN ACUTE LEUKEMIA. *(E.)* Fisher, W. B. (Case Western Reserve U., Cleveland, Ohio), S. A. Armentrout, R. Weisman and R. C. Graham, Jr. *Arch Intern Med* 132(2):226-232, 1973.

3324 THE INVASIVE GROWTH OF MALIGNANT TUMOURS AS A PURELY MECHANICAL PROCESS. *(E.)* Eaves, G. (Sch. Med., Leeds, England). *J Pathol* 109(3):233-237, 1973.

3325 BENIGN PAPILLOMA OR PAPILLARY CARCINOMA OF THE BLADDER? *(E.)* Greene, L. F. (Mayo Clin., Rochester, Minn.), K. A. Hanash and G. M. Farrow. *J Urol* 110(2):205-207, 1973.

3326 INVESTIGATIONS OF THE STRUCTURAL AND FUNCTIONAL CONDITIONS OF THE POSTMENOPAUSE OVARIES IN PATIENTS WITH CARCINOMAS OF THE CORPUS AND NECK. *(E.)* Mestwerdt, W. (Med. Coll, Hannover, Fed. Repub. Germany), H. Brandau, H. K. Kley and O. Müller. *Munch Med Wochenschr* 115(41):1796-1804, 1973.

3327 OESTRADIOL RECEPTORS IN THE CYTOPLASM OF PRECANCEROUS HUMAN ENDOMETRIUM. *(E.)* Milton, P. J. D. (St. Thomas' Hosp., London, England), R. W. Taylor and S. G. Crocker. *Acta Endocrinol* 74(2):245, 1973.

3328 STUDIES OF THE CELL POPULATION KINETICS OF INDUCED AND TRANSPLANTED MAMMARY ADENOCARCINOMA IN RATS. *(E.)* Simpson-Herren, L. (Southern Res. Inst., Birmingham, Alabama) and D. P. Griswold, Jr. *Cancer Res* 33(10):2415-2424, 1973.

The length of the cell cycle and the thymidine index (less than 1 day and 6.5%, resp.) for growing, 7,12-dimethylbenz(a)anthracene-induced mammary adenocarcinoma in female Sprague-Dawley rats vary only slightly over a 15-fold change in tumor mass. The observed decreasing growth rate with increasing tumor mass is attributed to a change in the balance of cell production to cell loss. The induced tumor, regressed by ovariectomy and stimulated by estradiol and progesterone, has a shorter doubling time, a 1-day cell cycle time, and a 11% thymidine index. The proliferative fraction of hormone-responsive tumors is modified by hormonal intervention. A transplanted mammary adenocarcinoma, derived from a 7,12-dimethylbenz(a)-anthracene-induced tumor in Fischer rats, has the shortest doubling time, a slightly shorter cell cycle time, and the highest thymidine index (15%). Computer analysis by two mathematical models for the percentage labeled mitoses data is presented for comparison.

3329 THE ONTARIO CANCER INCIDENCE SURVEY, 1964-1966: A NEW APPROACH TO CANCER DATA ACQUISITION. *(E.)* MacKay, E. N. (Ontario Cancer Treatment Res. Fdn., Toronto, Canada) and A. H. Sellers. *Can Med Assoc J* 109:489-494, 1973.

The Ontario Cancer Incidence Survey of 1964-1966 proved that records already collected for other purposes can be linked to provide satisfactory incidence data with minimal duplication of effort. Data from the available records were abstracted on small index cards, manually linked by patient's name, sex, age, residence, site of cancer, Ontario Hospital Services Commission contract number when available, and any other clues; these were then coded, and processed by computer to provide basic tables and a profile listing of the coded material. The project covered some 20,000,000 person-yr at risk and 147,810 reports were linked to provide information on 81,155 malignant neoplasms: the 1966 incidence rate was 316.7 per 100,000 Ontario residents. Advantages of this system over orthodox registries are respectable geographic coverage and follow-up without reporting by physicians, and the full use of accumulated material. Disadvantages are the time-lag inherent in using service records submitted for other purposes, and initial difficulty in distinguishing between new and old cases. The problem of inadequate reporting of pathology on hospital separation forms is being solved. Survey tabulations are frequently used. The survey methods have been adapted for computer processing as an on-going provincial cancer registry.

3330 SELF-REGULATION OF GROWTH IN THREE DIMENSIONS. *(E.)* Folkman, J. (Children's Hosp. Med. Ctr., Boston, Mass.) and M. Hochberg. *J Exp Med* 138(4):745-753, 1973.

Multi-cell spheroids of B-16 mouse melanoma, V-79 chinese hamster lung and L-5178Y murine leukemia cells were grown in soft agar. When each spheroid was cultured in a large volume of medium, frequently renewed, all spheroids eventually reached a dormant phase at a diameter of approximately 3-4 mm and a population of approximately 10^6 cells. In the dormant spheroid, newly generated cells at the periphery balanced those lost by necrosis in the center. It is proposed that this dormant phase is due to a gradual reduction in the ratio of surface area to volume: a size is achieved beyond which there is insufficient surface area for the spheroid to eliminate catabolites and absorb nutrients. Thus, in the face of unlimited space and of new medium, three-dimensional cell populations become self-regulating. This phenomenon contrasts with standard tissue culture in which cell populations, living on a flat plane in two dimensions, will not stop growing in the face of unlimited space and new medium because the ratio of surface area to volume remains constant. These experiments provide a mechanism for observations *in vivo*: before vascularization, solid tumors live by simple diffusion as three-dimensional spheroids or ellipsoids. They become dormant at a diameter of only a few millimeters; once vascularized, they are released from this dormant phase and begin exponential growth. Thus, tumor dormancy resulting from absence of angiogenesis *in vivo*, may operate by the same mechanism responsible for dormancy of spheroids *in vitro*. "Antiangiogenesis", by whatever means, might force a tumor to remain in the dormant phase.

3331 PREVALENCE OF MALIGNANT TUMORS AT AUTOPSY IN CHILEAN CHILDREN FROM SANTIAGO, WITH PARTICULAR REFERENCE TO LEUKAEMIA. *(E.)* Zaldivar, R. (U. Tennessee Coll. Med., Memphis). *Z Krebsforsch* 80(1):3-4, 1973.

An investigation on the prevalence of cancer at autopsy in children from the city of Santiago was made. The records of autopsies performed from 1945 through 1966 were reviewed. Of the 6431 autopsy cases, 142 (2.21%) showed cancer. Leukemia (50.70%), intracraneal and other neural tumors (25.35%), and malignant lymphomas (14.08%) were the most frequent groups of childhood cancer. Leukemia accounted for 1.12% of all autopsies, and showed a corrected male:female ratio of 1:0.61. Acute leukemia accounted for 42.95% of the tumor cases.

3332 PROLIFERATION KINETICS OF DENSITY-INHIBITED CULTURES OF HUMAN CELLS, A COMPLEX *IN VITRO* CELL SYSTEM. *(E.)* Zinninger, G. F. (Harvard U. Sch. Pub. Hlth., Boston, Mass.) and J. B. Little. *Cancer Res* 33(10):2343-2348, 1973.

The proliferation kinetics and cell cycle parameters of exponentially growing and density-inhibited cultures of Chang human liver cells (LICH) were determined. The mean duration of all phases of the cell cycle, as determined by the method of labeled mitoses, was approximately double in cultures density-inhibited at about 4 x 10^5 cells/cm^2 and refed daily with fresh medium when compared with exponentially growing cultures originally seeded at 8 x 10^3 cells/cm^2 and refed daily. About 50% of this density-inhibited population was noncycling but viable, with a 20% daily cell turnover rate. Most the non-S phase cells in these cultures were in G_1. Microscopic inspection of density-inhibited cultures revealed three cell populations: a firmly attached dense monolayer with a high plating efficiency and high initial labeling index with ^{3}H-thymidine; a loosely attached layer of rounded-up cells overlying the first layer with an intermediate plating efficiency and intermediate initial labeling index; and floating cells with a low plating efficiency and very low initial labeling index. Density-inhibited LICH cell cultures might provide a useful *in vitro* system for studying the effects of chemotherapeutic agents, protracted schedules of x-irradiation, and cell-cycle dependent interactions between the two.

3333 MELANOMA IN THE NORTH OF THAILAND IN THE LAST TEN YEARS. *(E.)* Pour, P. (Hannover Med. Sch., Germany), T. Tantachamrum, J. Althoff and U. Mohr. *Z Krebsforsch* 80(1):31-35, 1973.

The cases of melanoma diagnosed at the Chiang Mai University Medical School in the last ten years were histologically re-examined and the case histories, when available, were evaluated. The different anatomical locations of melanoma related to age and sex of the patients were analyzed. More than 50% of the melanomas were found on the lower extremities. The frequency of the tumors was higher in males than in females. During the period covered, the percentage of skin tumors as well as the incidence and sex distribution of malignant melanomas were also constant. Any additional relationships could not be calculated because the number of cases (68 with malignant melanomas) was too small.

3334 LUNG CANCER AMONG URANIUM MINERS IN THE UNITED STATES. *(E.)* Archer, V. E. (Natl. Inst. Occupational Safety Hlth., Salt Lake City, Utah), J. K. Wagoner and F. E. Lundin. *Health Phys* 25(4):351-371, 1973.

Data collected during the period July 1950 through September 1968 demonstrate excess respiratory cancer among all groups of uranium miners who have had more than 120 Working Level Months of radon daughter exposure. Lung cancer incidence rose with increasing exposure. Factors which might distort the exposure-response relationship were reviewed. Exposure to other agents such as cigarettes, diesel smoke and exposure to miscellaneous dusts probably contributed to the excess, but these factors should not be considered in setting permissible levels. Respiratory cancers are continuing to appear at a high rate among the Study Group even though radon daughter levels have been markedly reduced and most of the Study Group have stopped mining.

3335 PRELIMINARY REPORT ON THE PREVALENCE OF PALATAL PRECANCEROUS LESIONS IN THE RURAL POPULATION OF VISAKHAPATNAM DISTRICT. *(E.)* Reddy, C. R. R. M. (Andhra Med. Coll., Visakhapatnam, India), C. Ramulu, M. S. V. Raju, B. Sundareshwar, B. Ram Murthy, P. Sastry and G. V. Narasimham. *Indian J Med Res* 61(6):838-847, 1973.

Carcinoma of the hard palate is very common in the district of Visakhapatnam as also the habit of reverse smoking of chuttas (home-made cigars). Information regarding the number and sex of the people who smoke like this and any precancerous lesion which might occur in the palate is lacking. In twenty villages, in the district of Visakhapatnam, a total of 9,400 people were surveyed. The purpose of the survey was to know the prevalence of the habit of reverse smoking, stomatitis nicotina and carcinoma of hard palate. It was seen that about 50% of the women and 25% of men above 20 yr of age smoke chuttas in the reverse way. Seventy to 80% of these develop stomatitis nicotina, compared to 30-40% of those who smoke chuttas in the ordinary way. Somatitis nicotina is probably precancerous as was seen in our earlier studies. Two percent of the people with stomatitis nicotina have ulcers in the palate which could be malignant. This study shows the wide prevalence of the habit and its association with changes in palate which are probably precancerous and also explains the high prevalence of hard palate carcinoma in this area.

3336 FECUNDITY AND INFANTILE CANCER. *(E.)* Lyster, W. R. (London, England). *Lancet* (7823):267-268, 1973.

See also:

* (Rev): 3003, 3007, 3030
* (Chem): 3045
* (Immun): 3277

3337 CELLS, INTRACELLS AND SCHIZOGENY III. FORMATION OF INTRACELLS IN HUMAN AMNION CELLS AND SV40 VIRUS-TRANSFORMED MOUSE EMBRYO CELLS CULTURED *IN VITRO*.

(E.) Valladares, Y. (Natl. Inst. Oncology, City U., Madrid, Spain), Y. Alvarez, J. Alvarez and E. Tabares. *Oncology* 27(4):336-355, 1973.

Cell growth and the formation of intracells (small cells within other well-constituted cells) were studied in cultured human amnion and SV40-transformed mouse embryo cells by phase-contrast microscopy. The duration of the cell cycle, as determined from a curve of labeled mitoses following a pulse label with ^{3}H-thymidine, was about 25.5 hr with G_1 phase lasting approximately 13 hr, S phase about 9.25 hr, and G_2 about 2 hr. Both the normal and transformed cell cultures showed abundant numbers of cells which contained intracells within vacuoles. The intracells could be divided into two classes: 1) spurious, originating as a phagocytic engulfing of one cell by another 2) genuine, arising spontaneously within a cell termed the "mother cell". Genuine intracells were generally formed by a budding process, although they could also be formed by condensation of cell material within the cytoplasm of the mother cell. Intracells contained RNA, DNA, a nucleus and nucleolus, but the exact nature and mechanism of their origin could only be speculated.

3338 CARCINOMA OF THE PROSTATE PRESENTING AS OBSTRUCTIVE CARCINOMA OF THE RECTUM.

(E.) Mir, M. (Veterans Administration Hosp., Dayton, Ohio), H. Dikranian and C. L. Cogbill. *Am Surg* 39(10):582-586, 1973.

Two patients with adenocarcinoma of the prostate presenting as rectal tumors with intestinal obstruction are discussed. Although both had a preoperative tissue diagnosis of carcinoma, it was not clear that the primary site was prostate gland until abdominal perineal resection had been carried out in each case. Prostatic tumors may infiltrate locally, or venous and lymphatic spread may occur to the posterior wall of the rectum. Where the disease appears to be localized, prolonged palliation or cure may be obtained by radical operation with removal of both prostate and rectum. If the correct preoperative diagnosis is made in cases of advanced disease, colostomy may be necessary to relieve the bowel obstruction.

3339 A SYNTHETIC TRIPEPTIDE WHICH INCREASES SURVIVAL OF NORMAL LIVER CELLS, AND STIMULATES GROWTH IN HEPATOMA CELLS.

(E.) Pickart, L. (Dept. Pediatrics, U. Calif., San Francisco) L. Thayer and M. M. Thaler. *Biochem Biophys Res Commun* 54(2):562-566, 1973.

A synthetic glycyl-histidyl-lysine tripeptide was synthesized which possessed the same growth-promoting properties as a native factor previously isolated from human serum. The synthetic factor was equal to the native factor in its ability to stimulate isotopically labeled precursor incorporation into DNA and RNA of cultured rat hepatocyte and hepatoma cells. The synthetic factor, which was stable under storage for prolonged periods, also enhanced growth of rat hepatoma cells. Maximal activity of both the synthetic and native factors was seen at 20 ng/ml. Higher concentrations were inhibitory. Both the synthetic and native factors significantly increased survival of normal rat liver cells maintained in culture for 48 hr compared to untreated controls.

3340 PHOSPHOLIPIDS AND CALCIUM UPTAKE IN EXPERIMENTAL TUMORS.

(E.) Anghileri, L. J. (Tumor Res. Polyclin., Essen, W. Germany). *Oncology* 28(1):35-51, 1973.

Intracellular phospholipid levels and ^{45}Ca uptake were studied in Ehrlich ascites cells 6 days following their i.p. inoculation into mice. The highest total P, phospholipid P and ^{45}Ca uptake values were found in the mitochondrial fraction. The highest ^{45}Ca uptake, total P, and protein concentration in liver and kidney of tumor-bearing mice were in the cell-fluid fraction, with the highest phospholipid P level in the microsomal fraction. Phospholipid P levels in microsomal fractions of several solid tumors (melanoma, sarcoma, lymphoma) tended to increase as a function of growth time. An inverse relationship between malignancy of the tumor and phospholipid P concentration in the microsomal fraction was likely. Comparison of the ^{45}Ca uptake and intracellular phospholipid P of several of these tumors indicated a relationship bewteen ^{45}Ca incorporation and phospholipid concentration.

3341 ROTATIONAL DIFFUSION OF LECTINS BOUND TO THE SURFACE MEMBRANE OF NORMAL LYMPHOCYTES.

(E.) Shinitzky, M. (Weizmann Inst. Sci., Rehovot, Israel), M. Inbar and L. Sachs. *FEBS Letters* 34(2):247-250, 1973.

Studies were undertaken to determine by direct physical measurement the degree of mobility of the lectins, concanavalin A (con A), wheat germ agglutinin (WGA) and soybean agglutinin (SBA), bound to the surface membrane of normal rat lymph node lymphocytes using fluorescent polarization of fluorescein isothiocyanate conjugates of the three lectins (F-con A, F-WGA, F-SBA). Con A, unlike WGA and SBA, induced polar fluorescence cap formation on treated cells. Estimates of the relaxation times of agglutinin-treated cells showed that F-con A sites had the greatest relative degree of mobility (0.83) compared to F-WGA (0.37) and F-SBA (0.43). These results support the assumption that lectin activation of lymphocytes is associated with a dynamic redistribution of membrane components upon binding of the lectin. It is suggested that a high mobility of binding sites is also required for activation of lymphocytes by antigens.

3342 LIGHT AND ELECTRON MICROSCOPY OF PARATHYROID CARCINOMA. REPORT OF THREE CASES.

(E.) Altenähr, E. (Inst. Path., U. Hamburg, Germany) and W. Saeger. *Virchows Arch (Pathol Anat)* 360:107-122, 1973.

The morphology of three cases of parathyroid gland (PTG) carcinoma is described, including the electron microscope findings in two of these cases. The clinical and morphologic points for the tentative diagnosis of PTG carcinoma are discussed. A hard consistency and a greyish or white color are typical for PTG carcinomas and give contrast to the mostly soft consistency and yellow or tan color of PTG adenomas. The malignancy of a PTG tumor is proved by lymphogenic or hematogenic metastases, by histological evidence of tumor infiltration into the surrounding tissues (including macroscopic adherence and sometimes vocal cord paralysis), and by cytologic evidence of mitoses. Metastases of PTG carcinomas are found predominantly in the regional lymph nodes of the neck and mediastinum. Nuclear atypism is often present but is neither a necessary nor an adequate proof of malignancy, because it is also seen in benign adenomas and in hyperplastic parathyroids. The ultrastructure of the carcinoma cells was also characterized by nuclear atypism and mitoses. In one carcinoma, the contents of cytoplasmic organelles varied in different cells, indicating various endocrine activity of the tumor cells. In another parathyroid carcinoma with low endocrine activity, copious cytoplasmic organelles and many secretory granules were found. There seem to be three possible causes of nonfunctioning parathyroid carcinomas: 1. lack of hormone synthesis, 2. impairment of cellular hormone secretion, 3. synthesis of a pathologic protein with defective endocrine activity.

3343 ULTRASTRUCTURAL STUDIES ON HUMAN MYELOMA PLASMACYTES. *(E.)* Smetana, K. (Baylor Coll. Med., Houston, Texas), F. Gyorkey and H. Busch. *Cancer Res* 33(10):2300-2309, 1973.

Myeloma plasmacytes were studied in six untreated patients to obtain more information on the nuclear ultrastructural abnormalities in these cells. Asynchrony of nucleolar, nuclear, and cytoplasmic maturation was frequently observed in pleomorphic myeloma plasmacytes. Immature and mature myeloma plasmacytes of two patients contained intranuclear rodlets which consisted of fine filaments arranged in parallel. Myeloma plasmacytes in mitosis often contained persistent nucleoli, nucleolar fragments, and small nucleolus-like bodies. The mitotic division of the nucleus was also noted in differentiated cytoplasm. In some cells in mitosis, the cytoplasm was differentiated and apparently functional.

3344 CHROMOSOMAL INSTABILITY ASSOCIATED WITH SUSCEPTIBILITY TO MALIGNANT DISEASE IN PATIENTS WITH POROKERATOSIS OF MIBELLI. *(E.)* Taylor, A. M. R. (Dept. Cancer Studies, Med. Sch., U. Birmingham, England), D. G. Harnden and E. A. Fairburn. *J Natl Cancer Inst* 51(2):371-378, 1973.

Although the predisposition to porokeratosis of Mibelli is clearly inherited and the disorder commonly leads to development of epitheliomas, the type of lesion may vary considerably in different members of the same family, or even in the same person. Since, however, no relevant cytogenetic studies had previously been reported, the present investigation was carried out on skin biopsies from four patients with the disease. Two patients had squamous cell carcinomas on some of the lesions. Fibroblasts from affected and unaffected areas of the patients' skin were cultured, and clones of cytogenetically abnormal cells were found in 8 of 16 lines of fibroblasts from affected areas of skin of two patients. A third patient had no chromosomal abnormalities, and a further patient showed one line of cells from an apparently normal area of skin with two abnormal clones. No specific chromosomal abnormalities were common to all cell lines, and no unstable rearrangements were found. Ten lines of cells from seven normal healthy individuals, age-matched to the porokeratosis patients, had no abnormal clones of cells. The evidence thus indicates a chromosomal instability in cultured cells from patients with this syndrome. The association of a tendency to malignancy with chromosomal instability, both in irradiated skin and porokeratotic skin, suggests that the instability itself may play a part in the genesis of neoplasia. With regard to the observation that the cells examined were fibroblasts, while the reported tumors were carcinomas, it is noted that interaction between cells of the dermis and the epidermis is well documented. Nevertheless, the demonstration of chromosomal instability in dermal cells does not necessarily indicate that this abnormality is related to the induction of the epithelial abnormalities of porokeratosis of Mibelli. Similarly, since epithelial cells were not examined, it could not be concluded that the observed abnormalities were themselves premalignant changes.

3345 MORPHOLOGICALLY "DIFFERENTIATED" MOUSE NEUROBLASTOMA CELLS INDUCED BY NONCYCLIC AMP AGENTS: LEVELS OF CYCLIC AMP, NUCLEIC ACID AND PROTEIN. *(E.)* Prasad, K. N. (Dept. Radiol., U. Colorado Med. Ctr., Denver), K. Gilmer and S. Kumar. *Proc Soc Exp Biol Med* 143(4):1168-1171, 1973.

Noncyclic AMP agents such as X-ray, serum-free medium (SFM), 5-bromo-deoxyuridine (5-BrdU) and 6-thioguanine cause morphological "differentiation" of mouse neuroblastoma cells in culture, similar to that produced by cyclic AMP. The present study was carried out to determine whether the noncyclic AMP agents acted by increasing the intracellular level of cyclic AMP and whether like cyclic AMP, they would initiate increases in total RNA and protein contents of the affected cells. The cyclic AMP levels in 5-BrdU- and SFM-treated cells increased about twofold, suggesting that these agents induced "differentiation" via cyclic AMP. The levels remained unchanged, however, in X-irradiated- and 6-thioguanine-treated cells, suggesting that these agents bypassed the step involving elevation of cyclic AMP. Sodium butyrate increased the level of cyclic AMP without causing morphological differentiation, indicating that an elevation of cyclic AMP is not sufficient for expression of differentiated phenotype. The RNA and protein contents of "differentiated" cells markedly increased. The DNA content of SFM- and 6-thioguanine-treated cells did not change significantly when compared to controls, indicating similar distributions of cells throughout the cell cycle. The DNA content of 5-BrdU-treated cells decreased markedly, indicating accumulation of differentiated cells in the G_1 phase, as was the case with AMP-treated cells. The DNA content of X-irradiated cells, however, increased markedly, indicating expression of differentiated phenotype in the G_2 phase.

3346 TUMOR GROWTH AND DISULFIDE REDUCTION: POSSIBLE DEPENDENCE ON PROTEIN-DISULFIDE REDUCTASE. *(E.)* Apffel, C. A. (Massachusetts Dept. Public Hlth., Walpole) and J. E. Walker. *J Natl Cancer Inst* 51(2):575-583, 1973.

Tumor cells, washed and resuspended in balanced salt solution with 2-5 mM disodium ethylenediaminetetraacetate (EDTA), lost transplantability after incubation with 1 mM iodoacetate or N-ethylmaleimide. All 7 ascites and 2 solid murine tumors investigated were inactivated i.e., lost transplantability by these sulfhydryl (SH)-blocking procedures though their cells were still metabolizing and excluding supravital stains. Protection against inactivation was conferred by the SH compounds dithiothreitol (DTT), L-cysteine, and reduced glutathione (1 mM). Inactivated cells could be reactivated by further incubation with an SH compound or with native cell-free ascitic fluid. Therefore, loss of transplantability was not attributable to a lethal effect of the blocking agents. Protection with DTT or incubation with it alone resulted in more malignant tumors. Both ascitic fluid and intercellular material of solid tumors also protected against inactivation. This particular protection was overcome when a) the disulfides were converted into reactive SH's before SH blocking, or b) the nucleotide (NADPH)-dependent protein-disulfide reductase (PDR) of the fluid was inhibited by heating 30 minutes at 70 C or by 0.1 mM arsenite. Activity of the NADPH-dependent PDR was much higher in tumor cells than in normal cells. No NADPH-dependent PDR activity was found in normal serum, but activity was detected in cell-free ascitic fluid and in serum of tumor-bearing mice. Pretreatment of the ascitic fluid with DTT, EDTA, arsenite, or heat increased the immunogenicity of SH-blocked ascites tumor. Thus, both proliferation and immunogenicity of the tumors appeared to depend on reactive SH's, protein-disulfide reduction, and implicated enzyme(s) of the tumor cells.

3347 *IN VIVO* DISCONTINUOUS DNA SYNTHESIS IN EHRLICH ASCITES TUMOURS. *(E.)* Goldstein, N. O. (Sch. Veterinary Med., U. Pennsylvania, Philadelphia) and R. J. Rutman. *Nature [New Biol]* 244(139):267-269, 1973.

A discontinuous mechanism of DNA replication, involving initiation and synthesis of small 5-10S single stranded chains at numerous starting points along the template, followed by integration of the small chains into larger molecules by tandem ligation, seems to fit events observed in mammalian cells. In the present work, Ehrlich ascites tumors, grown intraperitoneally in mice, were used to study early sequences in DNA biosynthesis. The course of DNA synthesis was followed by analyzing alkali-lysed cells for DNA on alkaline sucrose gradients after intraperitoneal pulses with labeled deoxythymidine (^{3}H-dThd or ^{14}C-dThd) for 2 to 120 min. Bio-gel filtration was used to estimate ^{3}H-DNA from gradient fractions for 2-20 min pulses. Direct assay of gradient fractions for 30-120 min pulses gave ^{3}H-DNA values equivalent to trichloroacetic acid precipitation. Gradient patterns showed that at 2 and 5 min all newly synthesized ^{3}H-DNA was present as small chains, 9S, molecular weight 0.4×10^6, with little tendency of the 5 min pattern to show any increase in molecular weight or heterogeneity. By 10 min a second DNA component, ~30S, molecular weight $7\text{-}9 \times 10^6$, appeared as a major peak which predominated 20 min after ^{3}H-dThd injection. At 30 min the major component was "parental" size, 44S, molecular weight ~20×10^6; some 9S chains persisted, but 30S chains were not observed, presumably because they were concealed in the main 44S band. The observations seem to clarify the requirements for a model of DNA synthesis in Ehrlich cells. The central consideration is that newly formed DNAs in the form of fragments of molecular weight 0.4×10^6 undergo little or no direct chain elongation by addition of new deoxynucleotides; instead, 9S chains are conserved and subjected to a series of tandem joining steps (ligations) which led to discrete DNA intermediates. A model emerges from the data in which the replicon is viewed as an 8 μm length of DNA attached to the replication fork(s) along which new short chains are synthesized and within which they are then joined to form a 30S single strand unit of newly synthesized DNA. In such a model the number of DNA polymerase complexes would control the rate of initiation, whereas the replication fork(s) would control the overall rate of synthesis of the chromosome by regulating the rate of sequential integration of subunits.

3348 FACTORS AFFECTING GLYCOGEN LEVELS IN HeLa CELLS: PREDNISOLONE AND ADENINE NUCLEOTIDES. *(E.)* Melnykovych, G. (VA Hosp., Kansas City, Mo.) and C. F. Bishop. *J Natl Cancer Inst* 51(2):353-360, 1973.

Conversion of several hexoses into glycogen was previously studied in two strains of HeLa cells. The present work extends this study to effects of prednisolone on the accumulation and breakdown of glycogen in one of the strains (HeLa S3G). Incorporation of glucose into glycogen was followed by exposure of the HeLa cells to ^{14}C-labeled glucose. The radioactivity was determined either in the whole cells or in the glycogen fraction isolated by alkali solubilization of cells and precipitation with ethanol. Amounts of glycogen obtained were determined by colorimetric analysis with anthrone. The HeLa cells studied contained an increased amount of glycogen when grown from 3 to 4 days in the presence of 0.5 μg/ml prednisolone. This effect was due to a decreased degradation of glycogen, not to an increased glycogen synthesis. Glycogen levels were elevated only if prednisolone was present in the culture from the time of inoculation, which indicates a requirement for steroid during the period of initial cell attachment. Glycogen levels could also be increased in the same HeLa cell strain by adenosine or adenine nucleotides, including 3',5',-cyclic AMP. In contrast, the level of cellular glycogen was markedly decreased by dibutyryl 3',5'-cyclic AMP.

3349 ISOLATION OF RAPIDLY LABELLED NUCLEAR RNA OF HIGH SPECIFIC ACTIVITY FROM HUMAN LEUKAEMIC CELLS.

(E.) Seeber, S. (Essen Clin., Germany) and C. G. Schmidt. *Klin Wochenschr* 51(13):677-679, 1973.

Methods were developed for the preparation of highly labeled nuclear RNA from cells of different forms of human leukemia. The incubation medium was based on Hepes buffer and on the use of exhaustively dialyzed fetal calf serum offering suitable conditions for the uptake of ^{32}P-orthophosphate into the RNA of leukemic nuclei. The specific activities reached may allow more detailed nucleotide and oligonucleotide analyses of the various RNA species present in human leukemic cells.

3350 MALE BREAST CANCER. 4. GYNECOMASTIA IN PATIENTS WITH BREAST CANCER.

(E.) Scheike, O. (Frederiksberg Hosp., Copenhagen, Denmark) and J. Visfeldt. *Acta Pathol Microbiol Scand* [*A*] 81(3): 359-365, 1973.

With the aid of the Danish Tumor Registry, 265 cases of male breast cancer were reviewed with regard to previous history of gynecomastia and the finding of clinical gynecomastia on admission. In 10 patients there was a history of gynecomastia, and one patient presented clinical gynecomastia on admission. Preparations from 187 cases of the same series were assessed with a view to the presence of histologically verified gynecomastia. The 79 cases with sufficient breast tissue for a histological study revealed gynecomastia in 21 cases, 6 of which were of florid type (type I) and 15 of quiescent fibrous type (type II). In two cases ducts with severe atypia of the epithelium were found, but in spite of investigations of serial sections no transition into invasive growth was revealed. The following features seem to support the theory that gynecomastia may be a premalignant state: the finding of severe atypia of the epithelium in the ducts in concurrent gynecomastia and breast cancer; lower mean age in cases of breast cancer with concurrent gynecomastia; the higher ratio of male to female breast cancer and the low mean age of the male patients in an area with a high frequency of gynecomastia; high frequency of breast cancer in patients with the Klinefelter syndrome.

3351 LINDAU'S DISEASE. A STUDY OF ONE FAMILY THROUGH SIX GENERATIONS.

(E.) Lauritsen, J. G. (Dept. Surg., Univ. Aarhus, Denmark). *Acta Chir Scand* 139(5):482-486, 1973.

Lindau's disease (cerebellar hemangioblastoma associated with one or more of the following: other CNS hemangioblastomas, angiomatosis retinae, or multiple tumors and/or cysts in visceral organs) was diagnosed in a 34-year-old female who presented with malignant hypernephromas of both kidneys 11 yr after removal of a cerebellar hemangioblastoma. A study of six generations of her family (68 individuals) by personal interview or review of medical records revealed three verified and two probable cases of the disease. The verified cases involved a father and daughter with bilateral malignant hypernephromas plus multiple bilateral renal cysts. The third verified case involved a papilloma of the choroid plexus of the fourth ventricle, a finding not previously described in association with the disease. Chromosome studies of the affected family members and the children of the propositus revealed some chromatid breaks and hypoploids, but their frequency was not great. Ten persons were judged to have a genetic predisposition for the disease and will be followed in the future.

3352 GROWTH OF THE P388 LEUKEMIA AS AN ASCITES TUMOR IN ZINC-DEFICIENT MICE.

(E.) Barr, D. H. (Lab. Radiobiol., U. California, San Francisco) and J. W. Harris. *Proc Soc Exp Biol Med* 144(1):284-287, 1973.

Studies were conducted on the effect of dietary zinc deficiency on growth and ^{3}H-thymidine incorporation into DNA of the P388 murine lymphocytic leukemia growing as an ascites tumor in weanling male DBA/2 mice. All animals were begun on a zinc-deficient diet 17-24 days before inoculation of 10^{6} tumor cells. Whereas control animals lived an average of seven days after inoculation, zinc-deficient inoculated animals lived an average of one day longer. Successive daily cell counts showed that growth of tumor cells in zinc-deficient animals was significantly retarded after the third post-inoculation day. Autoradiographic studies of ^{3}H-thymidine labeled tumor cells indicated that zinc deficiency produced a sharp decrease in the number of cells undergoing DNA synthesis between three and four days after inoculation, coinciding with the onset of depression of the growth curve. Attempts to reverse the growth retardation by adding 670 ppm zinc to the drinking water on day five resulted in a rise in the ^{3}H-thymidine labeling index, but the extent and the time course of the increase were extremely variable.

3353 5'-NUCLEOTIDE PHOSPHODIESTERASE ISOENZYME PATTERN IN THE SERUM OF HUMAN HEPATOMA.

(E.) Tsou, K. C. (Sch. Med., U. Pennsylvania, Philadelphia), S. Ledis and M. G. McCoy. *Cancer Res* 33(10):2215-2217, 1973.

The 5'-nucleotide phosphodiesterase isoenzyme patterns of sera from normal controls and six patients with histologically confirmed hepatoma were studied by polyacrylamide gel electrophoresis followed by an assay using a 5'-(5-iodoindoxyl)-nucleoside phosphodiester as a histochemical substrate. Normal sera showed two major, quickly migrating bands and two minor, slower-moving bands. All hepatoma sera contained these four bands plus an additional fast-moving isoenzyme band. Neither serum from a patient with a benign liver adenoma nor neonatal serum contained this additional band. Double immunodiffusion tests showed that none of the hepatoma sera was positive for α-fetoprotein. The 5'-nucleotide phosphodiesterase isoenzyme pattern may thus be useful in the diagnosis of hepatoma.

3354 PRIMARY INTRALUMINAL AORTIC MYXOMA WITH INVOLVEMENT OF SEVERAL VERTEBRAE. *(E.)* Kimbrell, O. C., Jr. (Mem. Hosp. Wake Cty., Raleigh, N.C.) and L. J. Kaasa. *JAMA* 226(4):459-460, 1973.

A case of primary intraluminal aortic myxoma with vertebral involvement is reported in a 58-year-old white female. The patient presented with nausea and vomiting, acute epigastric pain, chest and right flank pain which radiated to the mid-thoracic vertebrae, hypertension refractory to drug therapy, and a grade 2/6 pansystolic murmur heard along the left sternal border. She later developed radiologic evidence of cardiac enlargement and thoracic vertebral deterioration as well as a loud abdominal bruit. Autopsy revealed subtotal aortic occlusion by a mass extending from the arch to the renal arteries. There was no evidence of involvement of the aortic wall or adventitia. Histochemical study showed that many of the tumor cells contained acid mucopolysaccharide and glycogen. Sections of the involved vertebrae showed a similar myxomatous tumor replacing the marrow. Thie case represents only the 17th reported primary aortic neoplasm, five of which have been benign myxoma.

3355 ESTROGEN RECEPTOR IN A HUMAN CELL LINE (MCF-7) FROM BREAST CARCINOMA. *(E.)* Brooks, S. C. (Michigan Cancer Fdn., Detroit), E. R. Locke and H. Soule. *J Biol Chem* 248(17):6251-6253, 1973.

A stable cell line (MCF-7) derived by pleural effusion from a breast cancer patient has been demonstrated to contain significant amounts of 17β-estradiol receptor. This binding protein is specific for 17β-estradiol and has a K_D equal to 2.5 nM, a sedimentation constant of 4.0S (and 9.2S), and provides a mechanism capable of transporting the 17β-estradiol into the nucleus. Migration of cytoplasmic 17β-estradiol receptor into the nuclei was clearly evident during 30 min incubation of MCF-7 cells at 37 C. However, appreciable nuclear uptake also occurred after 1 hr incubation at 0 C.

3356 HORMONE ASSAYS ON BREAST-TUMOUR CULTURES. *(E.)* Riley, P. A. (U. Coll. Hosp. Med. Sch., London, England), T. Latter and P. M. Sutton. *Lancet* (7833):818-820, 1973.

Cells cultured from 14 human breast-tumor biopsy specimens were examined morphologically and were tested with respect to cellular macromolecular synthesis for their response to estrogen, progesterone, and testosterone. Most of the lesions were scirrhous carcinomas, one was well-differentiated adenoid cystic carcinoma, and the remainder were fibroadenomas and chronic cystic disease. The initial growth rates were greatest for fibroadenoma cells which reached confluence in 10-15 days. Cancer cells and cells from chronic cystic disease rarely reached confluence and usually died out after about 25 days of culture. Incubation in medium containing 10^{-5} M hormone had little effect on the rate of ^{3}H-thymidine incorporation into DNA of fibroadenoma cells compared with untreated controls. All three hormones, however, decreased ^{3}H-thymidine incorporation in scirrhous carcinoma cells by about 20%. Estrogen-treated cells from the adenoid cystic carcinoma showed a 17-fold stimulation of ^{3}H-thymidine incorporation. Parallel autoradiographic studies indicated that repeated doses of estrogen over a four day period had greater stimulatory effects than a single dose.

3357 GUANOSINE ANABOLISM FOR BIOSYNTHESIS OF NUCLEIC ACIDS IN NOVIKOFF ASCITES RAT TUMOR CELLS IN CULTURE. *(E.)* Schaffer, M. (U. Texas M. D. Anderson Hosp. Tumor Inst., Houston), R. B. Hurlbert and A. Orengo. *Cancer Res* 33(10):2265-2272, 1973.

The pattern of utilization of ^{14}C-labeled guanosine and adenosine for the biosynthesis of RNA and DNA was studied in suspension cultures of rat Novikoff ascites tumor cells. Guanosine-8-^{14}C was readily incorporated, predominantly into guanine moieties of RNA and DNA as determined by chromatographic analysis of the nucleic acid hydrolysates. Adenosine-8-^{14}C, however, contributed substantially to both guanine and adenine residues. Experiments using guanosine labeled uniformly in the ribose and base indicated that guanosine did not contribute significantly to a common pool of ribose 1-phosphate or 5-phosphoribosyl-pyrophosphate for synthesis of other nucleotides. The enzymes responsible for guanosine metabolism were partially purified from cell homogenates by the use of DEAE-cellulose and Sephadex G-150 chromatography. Guanosine kinase, purine nucleoside phosphorylase, nucleoside monophosphate kinase, and nucleoside triphosphatase showed specificity for guanosine moieties with the last two enzymes also showing some activity toward adenosine compounds. Nucleoside diphosphate kinase showed little or no substrate specificity.

3358 PURINE NUCLEOTIDE METABOLISM AND NUCLEOTIDE POOL SIZES IN SYNCHRONIZED LYMPHOMA L5178Y CELLS. *(E.)* Snyder, F. F. (U. Alberta Cancer Res. Unit, Edmonton, Canada), J. F. Henderson, S. C. Kim, A. R. P. Paterson and L. W. Brox. *Cancer Res* 33(10):2425-2430, 1973.

Purine nucleotide metabolism was studied and nucleotide pool sizes were determined in mouse lymphoma L5178Y suspension cultures at 2, 4, 6, and 8 hr following synchronization by successive thymidine block and Colcemid treatment. Nucleotide metabolism was determined by the rate of incorporation of isotopically labeled purine base precursor. Nucleotide pool sizes were determined directly by high-pressure liquid chromatography. Most of the acid-soluble radioactivity rapidly accumulated in ATP and guanosine triphosphate; only small amounts were found in the mono- and diphosphates, indicating that uptake and conversion of the purine base to the nucleoside monophosphate were the rate-limiting steps in nucleoside triphosphate synthesis. ATP synthesis from adenine increased to a maximum (635 nM/30 min/10^9 cells) during late S phase and then declined during G_2. The lowest ATP levels (286 nM/30 min/10^9 cells) were found in G_1 cells. Variations in the rate of nucleotide synthesis from hypoxanthine and guanine during the cell cycle were similar to, although not so great in magnitude, those seen with adenine. After correction for variations in cell volume, however, ATP, guanosine triphosphate, and nicotinamide adenine dinucleotide con centrations were found to be relatively constant throughout the cell cycle. Uridine diphosphate-glucose, cytidine triphosphate, and uridine triphosphate levels appeared to decrease in the S and G_2 phases. Cell-cycle dependent variations in AMP deaminase and kinase, guanidylate reductase, guanosine monophosphate kinase, adenylsuccinate synthetase plus lyase, inosine monophosphate dehydrogenase, and hypoxanthine phosphoribosyltransferase were also observed.

3359 SERUM ALKALINE PHOSPHATASE IN MALIGNANT BONE TUMOURS (OSTEOSARCOMA, CHONDROSARCOMA, FIBROSARCOMA, EWING'S SARCOMA). *(E.)* Brozmanová, E. (Res. Inst. Rheumatic Dis., Piestany, Czechoslovakia) and B. Skrovina. *Neoplasma* 20(4):419-425, 1973.

Serum alkaline phosphatase levels were followed in 48 healthy subjects and in 27 patients with osteosarcoma, 31 with chondrosarcoma, 16 with fibrosarcoma, and 21 with Ewing's sarcoma. Data were collected from the time of diagnosis until surgical intervention (amputation). Mean values ranged from 8.78 ± 0.41 King-Armstrong (KA) U for fibrosarcoma to 16.8 ± 0.85 KA U for osteosarcoma, with values of 11.08 ± 0.64 and 12.9 ± 0.85 for Ewing's sarcoma and chondrosarcoma, resp. The norm value was 7.1 ± 0.44 KA U. Alkaline phosphatase levels in osteosarcoma patients varied with the stage of the disease, being 16.8 KA U at the onset, dropping to 10.1 KA U following surgical intervention and radiation therapy, and again rising to 18.5 KA U with recurrence and metastasis. Serum alkaline phosphatase levels may thus have some diagnostic and prognostic value, along with the histologic and radiologic picture, in malignant bone disease.

3360 INHERITED PREDISPOSITION TO CANCER? A DERMATOGLYPHIC STUDY. *(E.)* Fuller, I. C. (Connor Lodge, Sedgefield, Stockton-on-Tees, England). *Br J Cancer* 28(2):186-189, 1973.

Data are presented on the dermatoglyphics of a group of 70 cancer patients from Teesside and central County Durham, England, showing that they differ from those of groups suffering from diabetes, asthma, duodenal ulcers and schizophrenia and from those of reported mixed English samples. The differences are much more marked in males than in females. The mean ridge counts of the cancer patients on the right index and ring, and left middle and ring, fingers are significantly lower than those of all the other groups studied, and the mean angle on the right hand was significantly larger in the cancer group than in the others. Furthermore, the mean total ridge count and seven of the digital mean ridge counts are significantly lower in cancer patients than in the mixed sample, although this comparison is not strictly valid, for average dermatoglyphic values are known to vary from one district to another. Dermatoglyphic differences between cancer patients and controls have been reported by other researchers as well. Is is suggested that genes which produce these differences distinguish cancer patients from the general population and may predispose to the development of malignancy.

3361 A SPECIFIC PHOTOREACTION IN POLYDEOXYADENYLIC ACID. *(E.)* Pörschke, D. (Max Planck Inst. Biophys. Chem, Göttingen, W. Germany). *Proc Natl Acad Sci USA* 70(9):2683-2686, 1973.

Ultraviolet photosensitivity of the single-stranded forms of poly(A) and poly(dA) is compared. In contrast to poly(A), poly(dA) is rather sensitive to irradiation. Relatively low doses (e.g., 218 nm) of UV light lead to characteristic changes of the UV and circular dichroism spectra of poly(dA), suggesting formation of a specific photoproduct in poly(dA) that cannot be formed in poly(A). The quantum yields in poly(dA) ($\phi = 2.5 \times 10^{-3}$) are about a factor of 10 higher than in poly(A), indicating a high structure specificity. The base-pairing capacity of irradiated probes of poly(dA) to poly(dT) is inhibited. These results demonstrate a relatively high probability of DNA photolesions that cannot be repaired by simple repair enzyme systems.

3362 CHANGE OF LIPID ANTIOXIDATIVE ACTIVITY (AOA) WHILE CURING INJECTED LEUCOSIS WITH PREPARATIONS OF THE CLASS OF ALKYLATING AGENTS. *(Rus.)* Pal'mina, N. P. (Inst. Chem. Phys., Acad. Sci. USSR, Moscow) and E. B. Burlakova. *Biofizika* 18(1):166-169, 1973.

3363 THE FRACTIONAL CONTENT OF CELL PROTEINS OF SEIDEL ASCITIC HEPATOMA AND RAT LIVER. *(Rus.)* Mamontova, L. N. (P. A. Herzen Res. Inst. Onocology, Moscow, U.S.S.R.) and B. M. Zeljvin. *Vopr Onkol* 19(2):57-60, 1973.

3364 IMMUNOMORPHOLOGICAL CHARACTERISTICS OF TRANSPLANTABLE THYROID TUMORS. *(Rus.)* Okulov, V. B. (USSR Min. Public Hlth., Moscow). *Vopr Onkol* 19(2):50-56, 1973.

3365 AUER BODIES IN ACUTE PROMYELOCYTIC LEUKEMIA. DEMONSTRATION OF THEIR FINE STRUCTURE AND PEROXIDASE LOCALIZATION. *(E.)* Gorius, J. B. (Hosp. Henri Mondor, Créteil, France) and D. Houssay. *Lab Invest* 28(2):135-141, 1973.

3366 INTRACRANIAL MESENCHYMAL CHONDROSARCOMA. *(E.)* Seth, H. N. (Neurosurg. Ctr. Army Hosp., Delhi Cantt, India) and M. Singh. *Acta Neuropathol (Berl)* 24(1):86-89, 1973.

3367 TUMOURS OF THE CENTRAL NERVOUS SYSTEM IN ZAMBIA. *(E.)* Chuke, P. O. (U. Teaching Hosp., Lusaka, Zambia), T. M. Matthew and G. G. Naik. *Afr J Med Sci* 4(2):107-110, 1973.

3368 HISTOPATHOLOGY OF BRAIN TUMOURS IN THE AFRICAN IN KENYA. *(E.)* Kasili, E. G. (Dept. Path., U. Nairobi, Kenya), H. M. Cameron, R. F. Ruberti and S. A. Chopra. *Afr J Med Sci* 4(2): 99-106, 1973.

3369 ORIGIN OF THE PHILADELPHIA CHROMOSOME. *(E.)* TRACING OF CHROMOSOME 22 TO PARENTS OF PATIENTS WITH CHRONIC MYELOCYTIC LEUKEMIA. *(E.)* Gahrton, G. (Karolinska Inst., Stockholm, Sweden), J. Lindsten and L. Zech. *Exp Cell Res* 79(1):246-247, 1973.

3370 ELECTRICAL EXCITABILITY AND CHEMOSENSITIVITY OF MOUSE NEUROBLASTOMA X MOUSE OR HUMAN FIBROBLAST HYBRIDS. *(E.)* Peacock, J. H. (Natl. Inst. Hlth., Bethesda, Md.), F. A. McMorris and P. G. Nelson. *Exp Cell Res* 79(1):199-212, 1973.

3371 CAPILLARY HEMATOCRIT METHOD COMBINED WITH MICROSCOPICAL OBSERVATION AND ITS APPLICATION TO A QUICK SCREENING OF RAT LEUKEMIA. *(E.)* Sugiyama, T. (Kobe U. Sch. Med., Japan). *Acta Pathol Jap* 23(1):1-9, 1973.

3372 MAST CELLS IN TUMOUR-BEARING PATIENTS. *(E.)* Simu, G. (Oncological Inst. Cluj, Romania) and G. Csaba. *Acta Morphol Acad Sci Hung* 20(3-4): 327-338, 1972.

3373 MALIGNANT BRENNER TUMOR - A CASE REPORT. *(E.)* Toriumi, J. (Jikei U. Sch. Med., Tokyo, Japan) and Y. Ijima. *Acta Pathol Jap* 23(1): 121-128, 1973.

3374 ISOLATION AND PARTIAL CHARACTERIZATION OF MULTIPLE DNA POLYMERASES OF THE MURINE MYELOMA, MOPC-21. *(E.)* Persico, F. J. (Inst. Microbiol., Rutgers U., New Brunswick, N.J.), D. E. Nicholson and A. A. Gottlieb. *Cancer Res* 33(6):1210-1216, 1973.

3375 PHAGOCYTIC ACTIVITY OF THE LEUKEMIC CELL AND ITS RESPONSE TO THE PHAGOCYTOSIS-STIMULATING TETRAPEPTIDE, TUFTSIN. *(E.)* Constantopoulos, A. (Tufts U. Sch. Med., Boston, Mass.), V. Likhite, W. H. Crosby and V. A. Najjar. *Cancer Res* 33(6):1230-1234, 1973.

3376 PARALLELS, CONVERGENCES, AND DEPARTURES IN CASE-CONTROL STUDIES AND CLINICAL TRIALS. *(E.)* Schneiderman, M. A. (Natl. Inst. Hlth., Bethesda, Md.) and D. L. Levin. *Cancer Res* 33(6):1498-1503, 1973.

3377 PRIMARY SQUAMOUS CELL CARCINOMA OF THE ENDOMETRIUM. *(E.)* White, A. J. (Dept. Obstetrics, Gynecology, U. Iowa, Iowa City), H. J. Buchsbaum and M. A. Macasaet. *Obstet Gynecol* 41 (6):912-919, 1973.

3378 MYELOPROLIFERATIVE DISORDERS AND PLATELET FUNCTIONS. *(E.)* Kubisz, P. (Dept. Hosp., Cadca, Czechoslovakia). *Folia Haematol (Leipz)* 99 (2-3):202-211, 1973.

3379 CARCINOGENESIS IN TISSUE CULTURE: CHANGES IN BACK-TRANSPLANTABILITY OF THE POPULATION OF RAT ASCITES HEPATOMA JTC-15 CELLS DURING A LONG-TERM OF CULTIVATION. *(E.)* Tsuboi, K. (Inst. Med. Sci., U. Tokyo, Japan), T. Takaoka and H. Katsuta. *Jap J Exp Med* 43(2):107-119, 1973.

3380 FREE NUCLEOTIDES IN LEUKOCYTES IN NORMAL CONDITIONS AND IN CHRONIC LEUKAEMIAS. *(E.)* Maj, S. (Med. Clin., Warsaw, Poland), E. Zdebska, J. Daszyński and J. Radziszewski. *Folia Haematol (Leipz)* 99(2-3):157-169, 1973.

3381 TUMORS OF THE NERVOUS SYSTEM ON THE IVORY COAST. *(Fr.)* Giordano, C. (Abidjan U. Hosp., Ivory Coast). *Afr J Med Sci* 4(2):197-207, 1973.

3382 PITUITARY TUMOURS IN KAMPALA, UGANDA. *(E.)* Bailey, I. C. (Mulago Hosp., Kampala, Uganda). *Afr J Med Sci* 4(2):171-178, 1973.

3383 NEOPLASMS OF THE SELLA TURCICA REGION IN IBADAN, NIGERIA. *(E.)* Odeku, E. L. (Dept. Surg., U. Ibadan, Nigeria), B. O. Osunkoya, A. Adeloye and A. O. Williams. *Afr J Med Sci* 4(2): 127-136, 1973.

3384 SOME CHARACTERISTICS OF A FACTOR STIMULATING SKIN EPITHELIAL CELL GROWTH *IN VITRO*. *(E.)* Melbye, S. W. (Stanford U. Sch. Med., Calif.) and M. A. Karasek. *Exp Cell Res* 79(2):279-286, 1973.

3385 FIBROSARCOMA OF THE PARANASAL AIR SINUSES. *(E.)* Cronin, J. (Royal Infirmary, Manchester, England). *J Laryngol Otol* 87(8):667-674, 1973.

3386 GASTRIC CARCINOMA - A TEN YEARS SURVEY OF 189 PATIENTS. *(E.)* Bose, P. K. (Cty. Hosp., Letterkenny, Ireland) and J. A. Hanley. *J Ir Med Assoc* 66(9):238-240, 1973.

3387 ANNULATE LAMELLAE IN HUMAN LYMPHOSARCOMA AND CHRONIC LYMPHOCYTIC LEUKAEMIA. *(E.)* Hagon, E. E. (Sydney Hosp., New South Wales, Australia) and F. W. Gunz. *Cytobios* 25(7):7-14, 1973.

3388 REDUCTION OF LEUKEMIA INCIDENCE FOLLOWING SPLENECTOMY IN THE RAT. *(E.)* Moloney, W. C. (Peter Bent Brigham Hosp., Boston, Mass.) and V. P. King. *Cancer Res* 33(3):573-574, 1973.

3389 AN AUTOPSY STUDY OF SOME ROUTES OF DISSEMINATION OF CANCER OF THE BREAST. *(E.)* Viadana, E. (Roswell Park Mem. Inst., Buffalo, N.Y.), I. D. J. Bross and J. W. Pickren. *Br J Cancer* 27(4): 336-340, 1973.

3390 THE EFFECTS OF AFLATOXIN B_1 *IN VIVO* ON MEMBRANE-RIBOSOME ASSOCIATION. *(E.)* Williams, D. J. (Dept. Biochem., U. Coll. London, England), R. P. Clark and B. R. Rabin. *Br J Cancer* 27(4):283-290, 1973.

3391 BACTERIOLYTIC AND BACTERICIDAL ACTIVITY IN MONOCYTIC AND MYELOMONOCYTIC LEUKEMIA WITH HYPERLYSOZYMEMIA. *(E.)* Pruzanski, W. (Wellesley Hosp., Toronto, Canada), W.-D. Leers and A. C. Wardlaw. *Cancer Res* 867-873, 1973.

3392 ISOLATION OF ARGININE ACCEPTOR-PROTEINS FROM NORMAL RAT LIVER AND NOVIKOFF HEPATOMA SUPERNATANT. *(E.)* Pinard, J. (Notre-Dame Hosp., Montreal, Canada) and G. de Lamirande. *Br J Cancer* 27(1):18-26, 1973.

3393 SPECIFICITY AND SENSITIVITY OF CORTISOL-INDUCED CHANGES IN ALPHA AMINOISOBUTYRIC ACID TRANSPORT IN HUMAN LEUKEMIC SMALL LYMPHOCYTES AND LEUKEMIC MYELOBLASTS. *(E.)* Frengley, P. A. (U. Rochester Sch. Med., Dentistry, N.Y.), M. A. Lichtman and W. A. Peck. *J Clin Invest* 52:1518-1525, 1973.

3394 ENDOCRINE DISORDERS WITH DISTURBANCES OF WATER AND ELECTROLYTE INDUCED BY ECTOPIC HORMONE PRODUCTION IN TUMORS. *(Ger.)* Hayduk, K. (Med. U. Tübingen, West Germany) and W. Kaufmann. *Klin Wochenschr* 51(8):361-376, 1973.

3395 INDEPENDENCE OF PITUITARY TUMOR-MEDIATED SUPPRESSION OF GROWTH HORMONE SECRETION AND CYCLIC 3',5'-ADENOSINE MONOPHOSPHATE PRODUCTION. *(E.)* MacLeod, R. M. (U. Virginia Sch. Med., Charlottesville) and J. E. Lehmeyer. *Cancer Res* 33(4):843-848, 1973.

3396 METASTASIS AND TRITON WR 1339. *(E.)* Hellmann, K. (Imperial Cancer Res. Fund, London, England). *Eur J Cancer* 9(2):153-154, 1973.

3397 MORPHOLOGICAL CHANGES OF BENIGN PROSTATIC HYPERPLASIA IN CULTURE. *(E.)* McMahon, M. J. (Dept. Anatomy, U. Birmingham, England) and G. H. Thomas. *Br J Cancer* 27(4):323-335, 1973.

3398 INHIBITION BY ACETYLCHOLINE OF PROTEIN SYNTHESIS IN CULTURED LIMB REGENERATES. *(E.)* Foret, J. E. (Dept. Zoology, U. New Hampshire, Durham) and G. L. Babich. *Oncology* 27(3):281-285, 1973.

3399 FURTHER STUDIES ON THE SELF-LIMITATION OF GROWTH OF JB-1 ASCITES TUMOURS. *(E.)* Bichel, P. (Cancer Res. Inst., Aarhus, Denmark). *Eur J Cancer* 9(2):133-138, 1973.

3400 FERRITINAEMIA IN LEUKAEMIA AND HODGKIN'S DISEASE. *(E.)* Jones, P. A. E. (Welsh Natl. Sch. Med., Cardiff, Wales), F. M. Miller, M. Worwood and A. Jacobs. *Br J Cancer* 27(3):212-217, 1973.

3401 PLASMA PROLACTIN IN BREAST CANCER. *(E.)* Boyns, A. R. (Welsh Natl. Sch. Med., Cardiff, Wales), E. N. Cole, K. Griffiths, M. M. Roberts, R. Buchan, R. G. Wilson and A. P. M. Forrest. *Eur J Cancer* 9(2):99-102, 1973.

3402 THE BIOCHEMICAL MECHANISM OF SELECTIVE HEAT SENSITIVITY OF CANCER CELLS. IV. INHIBITION OF RNA SYNTHESIS. *(E.)* Strom, R. (Inst. Biol. Chem., Applied Biochem., U. Rome, Italy), A. S. Santoro, C. Crifo', A. Bozzi, B. Mondovi' and A. R. Fanelli. *Eur J Cancer* 9(2):103-112, 1973.

3403 SULFATED MUCOPOLYSACCHARIDE PRODUCTION BY SYNOVIAL SARCOMA CELLS *IN VIVO* AND IN TISSUE CULTURE. *(E.)* Buonassisi, V. (Michael Reese Hosp., Med. Ctr., Chicago, Ill.) and L. Ozzello. *Cancer Res* 33(4):874-881, 1973.

3404 THE CLINICAL SIGNIFICANCE OF GASTRIC CANCER SPREAD FEATURES. *(Rus.)* Budlyanskiy, A. Y. (Urban Oncol. Dispensary, Sverdlovsk, USSR). *Vopr Onkol* 19(4):64-68, 1973.

3405 PROTEIN METABOLISM INDICES DETERMINED IN THE UTERINE TISSUE FROM PATIENTS WITH FIBROMYOMA. *(Rus.)* Vishnevskaya, T. M. (A. M. Gor'kiy Med. Inst., Donetsk, USSR), I. M. Ishchenkd and Ye. V. Molzhaninoy. *Vopr Onkol* 19(4):27-30, 1973.

3406 SOMATOTROPIC ACTIVITY OF BLOOD SERUM IN LEUKEMIC PATIENTS. *(Rus.)* Shurybin, D. Ya. (S. M. Kiroy Military Acad., Leningrad, USSR), N. S. Petrov and V. I. Mazurov. *Vopr Onkol* 19(4):13-17, 1973.

3407 ALTERATIONS OCCURRING IN THE BLOOD CLOTTING POST-PHASE (CLOT RETRACTION) IN MALIGNANT TUMOR PATIENTS. *(Rus.)* Zhardetskiy, A. S. (Kursk, USSR), G. A. Komardina, M. L. Tatarskiy and L. N. Trusov. *Vopr Onkol* 19(4):77, 1973.

3408 SARCOMAS AND CARCINOSARCOMAS OF THE UTERINE CERVIX. *(E.)* Abell, M. R. (U. Michigan Med. Ctr., Ann Arbor) and J. A. Ramirez. *Cancer* 31(5): 1176-1192, 1973.

3409 FREEZING, MAINTAINING AND THAWING EHRLICH-LETTRE ASCITES CARCINOMA CELLS. *(E.)* McKee, R. W. (UCLA Sch. Med., Ca.) and B. McCarty. *Proc Soc Exp Biol Med* 143(1):50-54, 1973.

3410 HEMOCOAGULATION IN MALIGNANT TUMORS IN CHILDREN. *(Rus.)* Kirdan, G. V. (USSR Acad. Med. Sci., Moscow), Ju. I. Lorie and L. A. Durnov. *Vopr Onkol* 19(3):54-58, 1973.

3411 HEAD AND NECK CANCER IN ALCOHOLICS. THE RELATIONSHIP TO DRINKING, SMOKING, AND DIETARY PATTERNS. *(E.)* Kissin, B. (Downstate Med. Ctr., St. U. New York, N.Y.), M. M. Kaley, W. H. Su and R. Lerner. *JAMA* 224(8):1174-1175, 1973.

3412 BORDERLINE AND MALIGNANT MUCINOUS TUMORS OF THE OVARY. HISTOLOGIC CRITERIA AND CLINICAL BEHAVIOR. *(E.)* Hart, W. R. (Armed Forces Inst. Path., Washington, D.C.) and H. J. Norris. *Cancer* 31(5):1031-1045, 1973.

3413 RETENTION OF LYMPHOCYTE CHARACTERISTICS BY MYELOMAS AND θ^+-LYMPHOMAS: SENSITIVITY TO CORTISOL AND PHYTOHEMAGGLUTININ. *(E.)* Ralph, P. (Salk Inst. Biol. Studies, San Diego, Calif.). *J Immunol* 110(6):1470-1475, 1973.

3414 PSEUDOSARCOMA OF THE ESOPHAGUS (POLYPOID CARCINOMA OF THE ESOPHAGUS WITH PSEUDOSARCOMATOUS FEATURES). *(E.)* Enrile, F. T. (Jewish Hosp., Med Ctr., Brooklyn, New York, N.Y.), P. O. De Jesus, A. A. Bakst and R. Baluyot. *Cancer* 31(5):1197-1202, 1973.

3415 ELECTRON MICROSCOPY IN THE DIAGNOSIS OF PLEURAL MESOTHELIOMAS. *(E.)* Wang, N.-S. (Dept. Path., McGill U., Montreal, Quebec, Canada). *Cancer* 31(5):1046-1054, 1973.

3416 TRYPTOPHAN METABOLISM IN BREAST CANCER. CORRELATION WITH URINARY STEROID EXCRETION. *(E.)* Davis, H. L., Jr. (U. Wisconsin Med. Sch., Madison), R. R. Brown, J. Leklem and I. H. Carlson. *Cancer* 31(5):1061-1064, 1973.

3417 *IN VITRO* CULTURE OF HUMAN NORMAL AND LYMPHOMA CELLS. CYTOCHEMICAL STUDY AND UPTAKE OF LABELED NUCLEIC PRECURSORS. *(E.)* Bachmann, A. E. (Natl. Acad. Med., Buenos Aires, Argentina), L. C. G. Conesa, L. Sen, J. C. Scornik, S. B. de Salum and A. Pavlovsky. *Acta Cytol* 17(3):209-213, 1973.

3418 DETERMINATION OF γ GLUTAMYL TRANSPEPTIDASE ACTIVITY IN HUMAN SERUM. ITS USE IN ONCOLOGY. *(E.)* Delarue, J. C. (Inst. Gustave-Roussy, Villejuif, France), H. Sancho, J. Rouesse and C. Bohuon. *Biomedicine* 18(2):152-158, 1973.

3419 TRANSPORT AND METABOLISM OF GLUCOSAMINE BY CULTURED NOVIKOFF RAT HEPATOMA CELLS AND EFFECTS ON NUCLEOTIDE POOLS. *(E.)* Plagemann, P. G. W. (Med. Sch., U. Minnesota, Minneapolis) and J. Erbe. *Cancer Res* 33(3):482-492, 1973.

3420 ANDROGENS IN BREAST CANCER. I. ATYPICAL ENDOMETRIAL HYPERPLASIA AND BREAST CANCER IN MARRIED PREMENOPAUSAL WOMEN. *(E.)* Grattarola, R. (Natl. Inst. Study Cure Tumors, Milan, Italy). *Am J Obstet Gynecol* 116(3):423-428, 1973.

3421 ARRHENOBLASTOMA: *IN VIVO* AND *IN VITRO* STUDIES. *(E.)* Younglai, E. V. (Dept. Obstetrics, Gynecology, McMaster U., Ontario, Canada), H. Richmond, R. Atyeo and F. L. Johnson. *Am J Obstet Gynecol* 116 (3):401-407, 1973.

3422 THE DEGRADATION OF NEWLY SYNTHESIZED RNA IN EHRLICH ASCITES TUMOR CELLS. *(E.)* Tigerstrom, R. G. von (Dept. Microbiol., U. Alberta, Edmonton, Canada). *Can J Biochem* 51(5):495-505, 1973.

3423 PERTURBATION OF CELL CYCLE OF HUMAN LEUKAEMIC MYELOBLASTS *IN VIVO* BY CYTOSINE ARABINOSIDE. *(E.)* Ernst, P. (U. Hosp. Copenhagen, Denmark), A. Faille and S.-A. Killmann. *Scand J Haematol* 10(3):209-218, 1973.

3424 ISOZYMES OF EXPERIMENTALLY INDUCED PRIMARY SARCOMAS IN THE RAT. *(E.)* Levan, G. (Inst. Genetics, Lund, Sweden), F. Mitelman and J. Mark. *Hereditas* 73(2):318-321, 1973.

3425 IDENTIFICATION OF NORMAL AND ABNORMAL CHROMOSOMES IN TUMOR CELLS. *(E.)* Tiepolo, L. (Euratom Unit, Human Radiation, Cytogenetics, U. Pavia, Italy) and O. Zuffardi. *Cytogenet Cell Genet* 12(1):8-16, 1973.

3426 CYCLIC AMP LEVELS AND THE MORPHOLOGICAL DIFFERENTIATION OF MOUSE NEUROBLASTOMA CELLS. *(E.)* Sheppard, J. R. (U. Colorado Med. Sch., Denver, Colo.) and K. N. Prasad. *Life Sci* 12(2):431-439, 1973.

3427 STUDY OF COLD + RESTRAINT STRESS GASTRIC LESIONS IN SPONTANEOUSLY HYPERTENSIVE, WISTAR AND SPRAGUE-DAWLEY RATS. *(E.)* Goldenberg, M. M. (Morton-Norwich Products, Inc., Norwich, New York). *Life Sci* 12(10):519-527, 1973.

3428 ATYPICAL FIBROXANTHOMA OF THE SKIN. A CLINICOPATHOLOGIC STUDY OF 140 CASES. *(E.)* Fretzin, D. F. (Armed Forces Inst. Path., Washington, D.C.) and E. B. Helwig. *Cancer* 31(6): 1541-1552, 1973.

3429 EFFECT OF 5-BROMO-2-DEOXYURIDINE ON TRANSPORT OF DEOXYGLUCOSE AND CYCLOLEUCINE IN 3T6 FIBROBLASTS. *(E.)* Tsuboi, A. (Temple U. Sch. Med., Philadelphia, Pa.) and R. Baserga. *Cancer Res* 33(6):1326-1330, 1973.

3430 MALIGNANT HISTIOCYTOSIS OF THE ROBB-SMITH TYPE. HISTIOCYTIC MEDULLARY RETICULOSIS. *(E.)* Macken, M. F. (Henry Ford Hosp., Detroit, Mich.), Van Slyck, E. J. and S. M. Saeed. *Henry Ford Hosp. Med J* 21(1):3-10, 1973.

3431 SECONDARY BRONCHOGENIC CARCINOMA OF THE PENIS. *(E.)* Pikula, B. (Sch. Med., Sarajevo, Yugoslavia), A. Nikulin, P. Plamenac and E. Ljubović. *Lijec Vjesn* 94(7):345-348, 1972.

3432 EFFECT OF FAST-GROWING TRANSPLANTABLE HEPATOMA ON CELL PROLIFERATION IN HOST TISSUE OF THE MOUSE. *(E.)* Morgan, W. W. (U. Texas Med. Sch., San Antonio) and I. L. Cameron. *Cancer Res* 33(3):441-448, 1973.

3433 RENAL CARCINOMA IN PREGNANCY. *(E.)* Anderson, M. F. (U. Hosp. West Indies, Mona, Kingston, Jamaica) and D. W. Atkinson. *Br J Urol* 45:270-272, 1973.

3434 AN EVALUATION OF THE TNM CLASSIFICATION IN CANCER OF THE BREAST. *(E.)* Phillips, A. J. (Natl. Cancer Inst., Canada, Toronto). *Int J Cancer* 12(1):301-308, 1973.

3435 AGGREGATION OF FELINE LYMPHOMA CELLS BY HYALURONIC ACID. *(E.)* Wasteson, A. (Inst. Med. Chem., U. Uppsala, Sweden), B. Westermark and J. Pontén. *Int J Cancer* 12(1):169-178, 1973.

3436 *IN VITRO* STUDIES OF HUMAN TUMOR AND EMBRYONIC TISSUE. *(E.)* Allen, A. (Cedars-Sinai Med. Ctr., U. California, Los Angeles), T. Ramon, A. Jacobson and M. E. Wade. *Am J Obstet Gynecol* 116(4):463-469, 1973.

3437 CYTOLOGIC DETECTION OF MINUTE OVARIAN, ENDOMETRIAL AND BREAST CARCINOMAS, WITH EMPHASIS ON CLINICAL-PATHOLOGICAL APPROACHES. *(E.)* Masukawa, T. (Med. Coll. Wisconsin, Milwaukee), Y. Wada, R. F. Mattingly and J. F. Kuzma. *Acta Cytol* 17(4):316-319, 1973.

3438 MALIGNANT BONE TUMORS OCCURRING IN CHILDHOOD. *(Ger.)* Staufer, U. G. (Zurich U. Pediat. Clin., Switzerland) and H. J. Pluess. *Helv Chir Acta* 40(1-2):171-176, 1973.

3439 EXPERIMENTS ON THE TUMOUR CELLS USING TIME-LAPSE MICROCINEMATOGRAPHY. *(E.)* Bolognari, A. (U. Messina Ctr. Tumor Res., Italy) and V. Ciano. *Experientia* 29(2):255, 1973.

3440 CYTOLOGIC DIAGNOSIS OF MALIGNANT MELANOMA. *(E.)* Hajdu, S. I. (Mem. Hosp. Cancer, Allied Dis., New York, N.Y.) and A. Savino. *Acta Cytol* 17(4):320-327, 1973.

3441 GENETIC CUTANEOUS DISORDERS WITH GYNECOLOGIC TUMORS. *(E.)* Walker, R. H. (Dept. Obstetrics, Gynecology, Loma Linda U.) and W. B. Reed. *Am J Obstet Gynecol* 116(4):485-492, 1973.

3442 THE ULTRASTRUCTURE OF HORMONE-SECRETING PARATHYROID GLAND CARCINOMAS. *(Ger.)* Thiele, J. (Hannover Med. Sch., W. Germany), E. Reale and A. Georgii. *Virchows Arch [Zellpathol]* 13(2):153-168, 1973.

3443 MICRO-INVASIVE AND *IN SITU* CARCINOMAS OF THE ORAL CAVITY. A MORPHOLOGICAL AND STATISTICAL STUDY. *(Fr.)* Brocheriou, C. (Pitie-Salpetriere Med. Sch., Paris, France), G. Chomette, P. Cernea, M. Auriol and R. Helenon. *Pathol Microbiol (Basel)* 39(3/4):214-221, 1973.

3444 ELECTROPHORETIC ANALYSIS OF NORMAL AND CANCER PATIENT SERA AFTER INCUBATION WITH AN RNA-RICH FRACTION. *(Fr.)* Guarini, G. (Inst. Exp. Zooprophylaxis, Piemonte, Italy), V. Poppi, V. Biarese, G. Molfese, P. Lovisetto and Ch. Deckers. *Nouv Presse Med* 2(23):1579-1580, 1973.

3445 THE ULTRASTRUCTURE OF A CRANIOPHARYNGIOMA. AN *IN VIVO* AND *IN VITRO* STUDY. *(Fr.)* Choux, R. (Marseille Med. Sch., France), J. Hassoun, M. Berard, J.-F. Pellissier, M. Choux and M. Toga. *Arch Anat Pathol (Paris)* 21(2):179-188, 1973.

3446 BONE MARROW ALTERATIONS OCCURRING IN LYMPHORETICULAR SARCOMA. *(Fr.)* Duhamel, G. (No affiliation), J. Debray, M. Krulick, A. Najman, J. Deloux, T. Bonal and R. André. *Nouv Rev Fr Hematol* 13(3):401-410, 1973.

3447 ELECTRON-HISTOCHEMICAL INVESTIGATION OF LYSOSOMES IN EXPERIMENTAL HEPATOMAS WITH DIFFERENT DEGREES OF DIFFERENTIATION. *(E.)* Astashova, E. A. (Acad. Med. Sci. USSR, Moscow). *Bull Exp Biol Med (USSR)* 74(10):1302-1305, 1972.

3448 CLEAR CELL CARCINOMA OF THE HUMAN OVARY TRANSPLANTED TO THE PERITONEAL CAVITY OF THE HAMSTER. *(E.)* Cobb, L. M. (Chester Beatty Res. Inst., London, England), E. A. M. Boesen and A. M. Neville. *Transplantation* 16(1):76-78, 1973.

3449 EXPERIENCE WITH PRIMARY PULMONARY CARCINOMA AT GEISINGER MEDICAL CENTER. *(E.)* Hendricks, G. L. (Geisinger Med. Ctr., Danville, Pa.) and W. T. Barnes. *Bull Geisinger Med Ctr* 25(2):41-51, 1972.

3450 ADENOMATOID ODONTOGENIC TUMOR. *(E.)* Khin, U. (Cook County Hosp., Chicago, Ill.), E. Kasper and H. Adilman. *J Oral Surg* 31(8):607-612, 1973.

3451 RELIABILITY OF HISTOPATHOLOGIC DIAGNOSIS OF MALIGNANT LYMPHOMA. *(E.)* Hakama, M. (Finnish Cancer Registry, Helsinki, Finland), K. Franssila and E. Saxen. *Ann Clin Res* 5(2):104-108, 1973.

3452 ORAL MUCOSAL NEUROMA-MEDULLARY THYROID CARCINOMA SYNDROME. *(E.)* Walker, D. M. (Welsh Natl. Sch. Med., Cardiff, Wales). *Br J Dermatol* 88(6):599-603, 1973.

3453 LEUKOCYTE MEAN CORPUSCULAR VOLUME IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE LEUKEMIAS. *(E.)* Gauthier, J. (Sacré-Coeur Hosp., Montreal, Canada), P. Harel and C. Brosseau. *Can Med Assoc J* 109(3):187-189, 1973.

3454 SYNOVIAL SARCOMA OF THE HEAD AND NECK. *(E.)* Krugman, M. E. (Mt. Sinai Sch. Med., New York, N.Y.), H. D. Rosin and C. Toker. *Arch Otolaryngol* 98:53-54, 1973.

3455 OAT-CELL CARCINOMA OF THE LUNG SIMULTANEOUSLY PRODUCING ACTH AND SEROTONIN. *(E.)* Horai, T. (Ctr. Adult Dis., Osaka, Japan), H. Nishihara, R. Tateishi, M. Matsuda and S. Hattori. *J Clin Endocrinol Metab* 37(2):212-214, 1973.

3456 CRANIUM BIFIDUM OCCULTA WITH JUVENILE FIBROMATOSIS OF FACE. *(E.)* Bhatia, P. L. (Udaipur, India), H. K. Jain, R. L. Shrimali and M. K. Dube. *J Laryngol Otol* 87(6):587-592, 1973.

3457 OXYPHIL ADENOMA OF THE LARYNGEAL VENTRICLE. *(E.)* Mendonca, D. R. (Sheffield, England) and A. M. Ward. *J Laryngol Otol* 87(6):583-585, 1973.

3458 GLYCOLYTIC MECHANISMS IN EHRLICH ASCITES TUMOR CELLS - EFFECTS OF DISULFIDE DERIVATIVES OF PYRIDINE. *(E.)* Poole, D. T. (U. North Carolina Sch. Med., Chapel Hill), T. C. Butler and M. E. Williams. *Biochem Pharmacol* 22(15):1893-1904, 1973.

3459 LOW-RESISTANCE JUNCTIONS IN EPITHELIAL OUTGROWTHS FROM NORMAL AND CANCEROUS EPIDERMIS *IN VITRO*. *(E.)* Flaxman, B. A. (Skin Cancer Hosp., Philadelphia, Pa.) and F. V. Cavoto. *J Cell Biol* 58(1):219-223, 1973.

3460 OSHA ISSUES PROPOSAL FOR PERMANENT STANDARD ON 14 CARCINOGENS. *(E.)* Anonymous. *Am Paint J* 58(3):50, 1973.

3461 THYMUS-DERIVED PROLIFERATING LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKEMIA. *(E.)* Anonymous. *Lancet* (7823):263, 1973.

3462 NORMAL FAECAL BLOOD LOSS AFTER INJECTION OF ^{51}Cr-LABELLED RED CELLS. *(E.)* O'Neill, B. J. (Repatriation General Hosp., Concord, New South Wales), H.-Y. Shum and A. M. Streeter. *Lancet* (7823):262, 1973.

3463 MENINGITIS AND BRAIN TUMOUR. *(E.)* Arnstein, E. J. (U. Washington Sch. Med., Seattle) and R. J. Wedgwood. *Lancet* (7823):262-263, 1973.

3464 CAVERNOUS HEMANGIOMA OF THE ORAL CAVITY: REVIEW OF THE LITERATURE AND REPORT OF CASE. *(E.)* Friedman, J. M. (Brooklyn, N.Y.), M. B. Gormley, S. Kupfer and W. Jarrett. *J Oral Surg* 31(8):617-619, 1973.

3465 CENTRAL HEMANGIOMA OF THE MANDIBLE: REPORT OF CASE. *(E.)* Martis, C. (Thessaloniki, Greece) and D. Karakasis. *J Oral Surg* 31(8):613-616, 1973.

3466 INFLUENCE OF PROTEIN UNDER- AND OVERNUTRITION OF SPONTANEOUS TUMOR PREVALENCE IN THE RAT. *(E.)* Ross, M. H. (Inst. Cancer Res., Fox Chase, Philadelphia, Pa.) and G. Bras. *J Nutr* 103(7):944-963, 1973.

3467 PRIMARY SARCOMAS OF THE TEMPORAL BONE. *(E.)* Naufal, P. M. (Harvard Med. Sch., Boston, Mass.). *Arch Otolaryngol* 98(1):44-50, 1973.

3468 FIBROUS HISTIOCYTOMA OF THE PARANASAL SINUSES. REPORT OF A CASE. *(E.)* Townsend, G. L. (Mayo Clin., Rochester, Minn.), H. B. Neel, L. H. Weiland, K. D. Devine and J. B. McBean. *Arch Otolaryngol* 98:51-52, 1973.

3469 SUBCELLULAR DEMONSTRATION OF PEROXIDATIC OXIDATION OF TYROSINE TO MELANIN USING DIHYDROXYFUMARATE AS COFACTOR IN MOUSE MELANOMA CELLS. *(E.)* Okun, M. R. (Tufts U. Sch. Med., Boston, Mass.), B. Donnellan, R. P. Patel and L. M. Edelstein. *J Invest Dermatol* 61(2):60-66, 1973.

3470 STIMULATION BY PROSTAGLANDIN E_1 OF ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE FORMATION IN NEUROBLASTOMA CELLS IN THE PRESENCE OF PHOSPHODIESTERASE INHIBITORS. *(E.)* Hamprecht, B. (Max-Planck-Inst. Biochem., Munich, Germany) and J. Schultz. *FEBS Letters* 34(1):85-89, 1973.

3471 SERUM LACTIC DEHYDROGENASE AND ISOENZYME CHANGES IN CLINICAL CANCER. *(E.)* Wood, D. C. (U. Oregon Med. Sch., Portland), V. Varela, M. Palmquist and F. Weber. *J Surg Oncol* 5(3):251-257, 1973.

3472 MODIFIED HORMONAL CONTROL OF ENZYMATIC ACTIVITY IN TUMORS. *(E.)* Criss, W. E. (U. Florida Coll. Med., Gainesville). *Am J Obstet Gynecol* 116(6):753-759, 1973.

3473 THE BIOCHEMICAL DIAGNOSIS OF OVARIAN CANCER. *(E.)* McGowan, L. (George Washington U. Med. Ctr., Washington, D.C.), R. H. Davis and B. Bunnag. *Am J Obstet Gynecol* 116(6):760-768, 1973.

3474 BILATERAL GLOMUS TUMORS OF THE TEMPORAL BONE. REPORT OF A CASE. *(E.)* House, W. F. (Los Angeles Fdn. Otology, Ca.) and M. D. Graham. *Arch Otolaryngol* 98(1):58-59, 1973.

3475 CARCINOMA OF PROSTATIC ORIGIN. METASTATIC TO CERVICAL LYMPH NODES. *(E.)* Nussbaum, M. (Beth Israel Med. Ctr., New York, N.Y.). *NY State J Med* 73(16):2050-2054, 1973.

3476 RHABDOMYOSARCOMA - A TEMPORAL BONE REPORT. *(E.)* El-Gothamy, B. (Henry Ford Hosp., Detroit, Mich.), S. Fujita and R. C. Hayden, Jr. *Arch Otolaryngol* 98(2):106-110, 1973.

3477 A CASE OF JUVENILE ANGIOFIBROMA. *(E.)* Parker, R. (Guy's Hosp., London, England). *Arch Otolaryngol* 98(2):129-130, 1973.

3478 MIDDLE EAR EXTENSION OF NASOPHARYNGEAL CARCINOMA VIA EUSTACHIAN TUBE. A TEMPORAL BONE REPORT. *(E.)* Cundy, R. L. (U. Colorado Med. Ctr., Denver), I. Sando and W. G. Hemenway. *Arch Otolaryngol* 98(2):131-133, 1973.

3479 CARCINOMA OF MÜLLERIAN VESTIGIAL ORIGIN: REPORT OF A CASE. *(E.)* Gupta, I. M. (Banaras Hindu U., Varanasi, India) and B. N. Prasad. *Aust NZ J Surg* 42(4):398-401, 1973.

3480 EMBRYONAL RHABDOMYOSARCOMA OF THE HEAD AND NECK IN CHILDREN. *(E.)* Sessions, D. G. (Washington U. Med. Sch., St. Louis, Missouri), A. H. Ragab, T. J. Vietti, H. F. Biller and J. H. Ogura. *Laryngoscope* 83(6):890-897, 1973.

3481 ANDROBLASTOMA OF THE OVARY WITH HETEROTOPIC ELEMENTS. *(E.)* Hayes, D. M. (Belfast City Hosp., Ireland) and R. J. Hunter. *J Pathol* 109(3):267-270, 1973.

3482 TRIPLE CARCINOMA OF THE BREAST, ONE ARISING WITHIN A FIBRO-ADENOMA. *(E.)* More, I. A. R. (Western Infirmary, Glasgow, Scotland) and A. T. Sandison. *J Pathol* 109(3):263-265, 1973.

3483 MALIGNANT INTRASPINAL EXTRADURAL TUMORS. *(E.)* Storen, E. J. (Ulleval Hosp., Oslo, Norway) and I. O. Skalpe. *J Oslo City Hosp* 23(5):85-95, 1973.

3484 CHROMOSOME STUDY OF MALIGNANT MELANOMAS. *(Fr.)* Berger, R. (Gutsave-Roussy Inst., Villejuif, France) and J. Lacour. *Biomedicine* 19(1):22-27, 1973.

3485 CAROTID BODY TUMORS - IDIOSYNCRACIES. *(E.)* Pollack, R. S. (San Francisco, Calif.) *Onocology* 27(1):81-91, 1973.

3486 EFFECT OF ANTICANCER DRUGS ON UTERINE CARCINOGENESIS. *(E.)* Takamizawa, H. (Chiba U. Sch. Med., Japan) and K. Wong. *Obstet Gynecol* 41(5):701-706, 1973.

3487 ELECTRONMICROSCOPY OF LEUKAEMIC MYELOBLASTS WITH NUMEROUS LIPID-CONTAINING VACUOLES. *(E.)* Polliack, A. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel) and R. Timberg. *Scand J Haematol* 9(5):437-441, 1972.

3488 CLEAR CELL ADENOCARCINOMA IN YOUNG FEMALES. *(E.)* Gilson, M. D. (Natl. Naval Med. Ctr., Bethesda, Md.), D. D. Dibona and D. R. Knab. *Obstet Gynecol* 41(4):494-500, 1973.

3489 NASOPHARYNGEAL FIBROMA. *(E.)* Tapia Acuña, R. (Mexico City, Mexico). *Acta Otolaryngol (Stockh)* 75(2/3):119-126, 1973.

3490 THE MALATE AND OXALOACETATE LEVELS DETERMINED IN WHOLE BLOOD SAMPLES TAKEN FROM PATIENTS WITH AND WITHOUT MALIGNANT TUMOR DISEASE. *(Ger.)* Schauenstein, E. (Graz U. Inst. Biochem., Austria), L. Kronberger, R. F. Schaur, E. Fink and E. Georgiopulos. *Wien Klin Wochenschr* 85(26):478-482, 1973.

3491 CELL PROLIFERATION IN THE CASTRATE MOUSE SEMINAL VESICLE IN RESPONSE TO TESTOSTERONE PROPIONATE. II. THEORETICAL CONSIDERATIONS. *(E.)* Appleton, D. (Dept. Med. Statistics, Path., U. Newcastle upon Tyne, England), A. R. Morley and N. A. Wright. *Cell Tissue Kinet* 6(3):247-258, 1973.

3492 GLUCOCORTICOID-BINDING PROTEINS IN HUMAN ACUTE LYMPHOBLASTIC LEUKEMIC BLAST CELLS. *(E.)* Lippman, M. E. (Natl. Inst. Hlth., Bethesda, Md.), R. H. Halterman, B. G. Leventhal, S. Perry and E. B. Thompson. *J Clin Invest* 52(7):1715-1725, 1973.

3493 CRITICAL EVALUATION OF LAPAROTOMY AND SPLENECTOMY IN HODGKIN'S DISEASE. *(E.)* Meeker, W. R., Jr. (U. Kentucky Med. Ctr., Lexington), J. D. Richardson, W. O. West and J. C. Parker. *Arch Surg* 105(2):222-229, 1972.

3494 USEFULNESS OF BONE MARROW SERUM ACID PHOSPHATASE IN STAGING CARCINOMA OF THE PROSTATE. *(E.)* Reynolds, R. D. (Med. Ctr., Travis Air Force Base, Calif.), B. R. Greenberg, N. D. Martin, R. N. Lucas, C. N. Gaffney and L. Hawn. *Cancer* 32(1):181-184, 1973.

3495 SINUS HISTIOCYTOSIS IN NON-DIAGNOSTIC SCALENE LYMPH NODE BIOPSIES. *(E.)* Silverberg, S. G. (U. Colorado Sch. Med., Denver), W. J. Frable and J. W. Brooks. *Cancer* 32(1):177-180, 1973.

3496 HISTOLOGY OF GASTRIC CARCINOMA OCCURRING AFTER GASTRIC SURGERY FOR BENIGN CONDITIONS. *(E.)* Taksdal, S. (Ulleval Hosp., Oslo, Norway) and H. Stalsberg. *Cancer* 32(1):162-166, 1973.

3497 HODGKIN'S DISEASE. A CLINICAL AND PATHOLOGIC STUDY OF 235 CASES. *(E.)* Patchefsky, A. S. (Thomas Jefferson U. Hosp., Philadelphia, Pa.), H. Brodovsky, M. Southard, H. Menduke, S. Gray and W. S. Hoch. *Cancer* 32(1):150-161, 1973.

3498 ACUTE MYELOMONOCYTIC LEUKEMIA WITH MACROGLOBULINEMIA, BENCE JONES PROTEINURIA, AND HYPERCALCEMIA. *(E.)* Allen, E. L. (Dept. Med., Ohio St. U., Columbus), E. N. Metz and S. P. Balcerzak. *Cancer* 32(1):121-124, 1973.

3499 ANERYTHRAEMIC DI GUGLIELMO SYNDROME. REPORT OF A CASE. *(E.)* Petersen, H. S. (U. Dept. Internal Med. II (Haematology), Arhus, Denmark. *Acta Med Scand* 193(4):363-364, 1973.

3500 ANGIOGRAPHIC DIFFERENTIATION OF NEUROGENIC TUMORS AT THE CAROTID ARTERY BIFURCATION. *(E.)* Skucas, J. (St. Vincent's Hosp., Med. Ctr., New York, N.Y.), P. Rossi, R. A. Brinker and R. C. Eberle. *Surg Gynecol Obstet* 137(1):35-39, 1973.

3501 INTERFERON AND CELL DIVISION. VIII. EFFECT OF INTERFERON ON MACROMOLECULAR SYNTHESIS IN L1210 CELLS *IN VITRO*. *(E.)* Brouty-Boyé, D. (Cancer, Immunogenetics Inst., Villejuif, France), A. Macieira-Coelho, M. Fiszman and I. Gresser. *Int J Cancer* 12(1):250-258, 1973.

3502 LEUKOCYTE PURINE PHOSPHORIBOSYLTRANSFERASES IN HUMAN LEUKEMIAS SENSITIVE AND RESISTANT TO 6-THIOPURINES. *(E.)* Rosman, M. (San Francisco Gen. Hosp., Calif.) and H. E. Williams. *Cancer Res* 33(6):1202-1209, 1973.

3503 HYPERCALCEMIA IN EPIDERMOID TUMORS OF THE HEAD AND NECK AND ESOPHAGUS. *(E.)* Stephans, R. L. (U. Kansas Med. Ctr., Kansas City), H. H. Hansen and F. M. Muggia. *Cancer* 31(6):1487-1491, 1973.

3504 THE EFFECT OF GLUCOCORTICOSTEROIDS ON THE PROLIFERATION AND KINETICS OF PROMONOCYTES AND MONOCYTES OF THE BONE MARROW. *(E.)* Thompson, J. (U. Hosp., Leiden, Netherlands) and R. van Furth. *J Exp Med* 137(1):10-21, 1973.

3505 BASOPHILIC LEUKAEMIA IN THE ALBINO RAT AND A DEMONSTRATION OF THE BASOPROTEIN. *(E.)* Eccleston, E. (ICI Ltd., Pharmaceuticals Division, Cheshire, England), B. J. Leonard, J. S. Lowe and H. J. Welford. *Nature [New Biol]* 244(133):73-76, 1973.

3506 THE SURFACE ULTRASTRUCTURE OF NORMAL AND METAPLASTIC CERVICAL EPITHELIA AND OF CARCINOMA *IN SITU*. *(E.)* Williams, A. E. (Dept. Microbiol., U. Birmingham, England), J. A. Jordan, J. M. Allen and J. F. Murphy. *Cancer Res* 33(3):504-513, 1973.

3507 ACTIVE TRANSPORT OF GLUCOSE ANOMERS BY ASCITES TUMOUR CELLS. *(E.)* Fishman, P. H. (George Washington U. Sch. Med., Washington, D.C.) and J. M. Bailey. *Nature [New Biol]* 243(123):59-60, 1973.

3508 DETECTION OF SINGLE-STRANDED DNA IN THE NUCLEOLUS. *(E.)* Amalric, F. (Fac. Med., U. Sherbrooke, Canada), S. Bernard and R. Simard. *Nature [New Biol]* 243(123):38-41, 1973.

3509 A GENETIC DIATHESIS TO SKIN CANCER. *(E.)* Lane-Brown, M. M. (Harvard Med. Sch., Boston, Mass.) and D. F. Melia. *J Invest Dermatol* 61(1):39-41, 1973.

3510 STUDIES OF THE GROWTH AND CYTOGENETIC PROPERTIES OF HUMAN ENDOMETRIAL ADENOCARCINOMA IN CULTURE AND ITS DEVELOPMENT INTO AN ESTABLISHED LINE. *(E.)* Kuramoto, H. (Sch. Med., Keio U., Tokyo, Japan). *Acta Obstet Gynaecol Jap* 19(1):47-58, 1972.

3511 TERATOCARCINOMA WITH THE CAPACITY FOR DIFFERENTIATION RESTRICTED TO NEURO-ECTODERMAL TISSUE. *(E.)* Damjanov, I. (U. Zagreb, Sch. Med., Yugoslavia), D. Solter and D. Serman. *Virchows Arch [Zellpathol]* 13(3):179-195, 1973.

3512 CALCIFIED LYMPH NODE METASTASES FROM CARCINOMA OF THE CERVIX. *(E.)* Hutcheson, J. (Vanderbilt U. Hosp., Nashville, Tenn.), D. L. Page and R. R. Oldham. *Cancer* 32(1):266-269, 1973.

3513 THE MESONEPHROID (HYPERNEPHROID) CARCINOMATOUS PATTERN - A RARE VARIANT OF PRIMARY ENDOCERVICAL ADENOCARCINOMA. *(E.)* Parker, J. C., Jr. (U. Kentucky Med. Ctr., Lexington), J. R. van Nagell, Jr., and J. D. Harralson. *Cancer* 32(1):259-265, 1973.

3514 MYELOID SARCOMA. REVIEW OF THE LITERATURE AND REPORT OF A CASE. *(E.)* Fayemi, A. O. (Mount Sinai Sch. Med., City U. New York, N.Y.), M. A. Gerber, I. Cohen, S. Davis and A. D. Rubin. *Cancer* 32(1):253-258, 1973.

3515 ULTRASTRUCTURE AND *IN VITRO* GROWTH CHARACTERISTICS OF A TRANSPLANTABLE RAT PHEOCHROMOCYTOMA. *(E.)* DeLellis, R. A. (U. Hosp., Boston, Mass.), F. B. Merk, P. Deckers, S. Warren and K. Balogh. *Cancer* 32(1):227-235, 1973.

3516 ECTOPIC PRODUCTION OF HUMAN CHORIONIC GONADOTROPIN IN UGANDAN PATIENTS WITH HEPATOCELLULAR CARCINOMA. *(E.)* Braunstein, G. D. (Natl. Inst. Hlth., Bethesda, Md.), C. L. Vogel, J. L. Vaitukaitis and G. T. Ross. *Cancer* 32(1):223-226, 1973.

3517 NEOPLASTIC DIATHESIS OF PATIENTS WITH CARCINOID. REPORT OF A CASE WITH FOUR OTHER NEOPLASMS. *(E.)* Brown, N. K. (Swedish Hosp. Med. Ctr., Seattle, Wash.) and M. P. Smith. *Cancer* 32(1):216-222, 1973.

3518 Y BODIES AND SIMILAR FLUORESCENT CHROMOCENTRES IN HUMAN TUMOURS INCLUDING TERATOMATA. *(E.)* Atkin, N. B. (Mount Vernon Hosp., Northwood, Middlesex, England). *Br J Cancer* 27(2):183-189, 1973.

3519 HUMAN PERIPHERAL LUNG TUMOURS: LIGHT AND ELECTRON MICROSCOPIC CORRELATION. *(E.)* Mollo, F. (Ctr. Electron Microscopy, Fac. Med., Turin, Italy), M. G. Canese and O. Campobasso. *Br J Cancer* 27(2):173-182, 1973.

3520 ALPHA-FETOPROTEIN AND ENDODERMAL SINUS TUMOR OF THE OVARY. *(E.)* Wilkinson, E. J. (Med. Coll. Wisconsin, Milwaukee), E. G. Friedrich and T. A. Hosty. *Am J Obstet Gynecol* 116(5):711-714, 1973.

3521 MORPHOLOGICAL ANOMALIES OF THE ERYTHROBLASTS AND ERYTHROCYTES OCCURRING IN MYELOPROLIFERATIVE DISEASES. *(Fr.)* Muratore, R. (No affiliation), R. Foa, R. Follana, J. Bayle and J. Olmer. *Nouv Rev Fr Hematol* 13(3):339-344, 1973.

3522 LEUKOKININ-FORMING SYSTEM IN THE ASCITIC FLUID OF A MURINE MASTOCYTOMA. *(E.)* Anonymous. *Biochem Pharmacol* 22(11):1386-1389, 1973.

3523 ANITSCHKOW NUCLEAR STRUCTURE IN CARDIAC METASTASES. *(E.)* Ragsdale, B. D. (Massachusetts Gen. Hosp., Boston). *Am J Clin Pathol* 59(6):798-802, 1973.

3524 CLEAR CELL CARCINOMA OF THE CERVIX AND VAGINA IN YOUNG WOMEN. A REPORT OF SIX CASES WITH ASSOCIATION OF MATERNAL STILBESTROL THERAPY AND ADENOSIS OF THE VAGINA. *(E.)* Hill, E. C. (U. California Hosp., San Francisco). *Am J Obstet Gynecol* 116(4):470-484, 1973.

3525 MIXED ADENOSQUAMOUS CARCINOMA OF THE ENDOMETRIUM. *(E.)* Ng, A. B. P. (U. Hosp. Cleveland, Ohio), J. W. Reagan, J. P. Storaasli and W. B. Wentz. *Am J Clin Pathol* 59(6):765-781, 1973.

3526 THE PHOSPHORYLATION OF 3'-DEOXY-3'-FLUOROTHYMIDINE AND ITS INCORPORATION INTO DNA IN A CELLFREE SYSTEM FROM TUMOR CELLS. *(E.)* Langen, P. (Central Inst. Molecular Biol., German Acad. Sci., Berlin), G. Kowollik, G. Etzold, H. Venner and H. Reinert. *Acta Biol Med Ger* 29:483-494, 1972.

3527 CARCINOMA OF THE PITUITARY GLAND. *(E.)* D'Abrera, V. St. E. (Lewisham Hosp., Sydney, Australia), W. J. Burke, K. F. Bleasel and L. Bader. *J Pathol* 109(4):335-343, 1973.

3528 ULTRASTRUCTURAL AND LIPID BIOCHEMICAL COMPARISONS OF HUMAN ERUPTIVE, TUBEROUS AND PLAMAR XANTHOMAS. *(E.)* Parker, F. (U. Washington Sch. Med., Seattle) and G. F. Odland. *Isr J Med Sci* 9(4):395-423, 1973.

3529 FURTHER OBSERVATIONS ON THE EFFECTS OF TRYPSIN ON THE VOLUME AND FUNCTIONS OF MITOCHONDRIA ISOLATED FROM NORMAL LIVER AND AH-130 YOSHIDA ASCITES HEPATOMA. *(E.)* Feo, F. (Gen. Path. Inst., U. Cso, Turin, Italy), G. Bonelli, R. A. Canuto and R. Garcea. *Cancer Res* 33(7):1804-1812, 1973.

3530 FINE STRUCTURE OF PRIMARY LIVER TUMORS AND TUMOR-BEARING LIVERS IN MAN. *(E.)* Ma, M. H. (Roy. Prince Alfred Hosp., Sydney, Australia) and C. R. B. Blackburn. *Cancer Res* 33(7):1766-1774, 1973.

3531 PREPARATIVE ELECTROPHORETIC SEPARATION OF NORMAL AND NEOPLASTIC HUMAN BONE MARROW CELLS. *(E.)* Schubert, J. C. F. (Frankfurt/M. U. Int. Med. Ctr., W. Germany), F. Walther, E. Holzberg, G. Pascher and K. Zeiller. *Klin Wschr* 51(7):327-332, 1973.

3532 PRODUCTION OF A NERVOUS-SYSTEM-SPECIFIC PROTEIN (14-3-2) BY HUMAN NEUROBLASTOMA CELLS IN CULTURE. *(E.)* Herschman, H. R. (Sch. Med., U. California, Los Angeles) and M. P. Lerner. *Nature* [*New Biol*] 241(112):242-244, 1973.

3533 CONTROL OF PHENOTYPIC EXPRESSION OF CULTURED MELANOMA CELLS BY MELANOCYTE STIMULATING HORMONES. *(E.)* Wong, G. (Yale U. Sch. Med., New Haven, Conn.) and J. Pawelek. *Nature* [*New Biol*] 241(111):213-215, 1973.

3534 GLUCOCORTICOID BINDING PROTEINS IN HUMAN LEUKAEMIC LYMPHOBLASTS. *(E.)* Lippman, M. (Natl. Cancer Inst., Natl. Inst. Hlth., Bethesda, Md.), R. Halterman, S. Perry, B. Leventhal and E. B. Thompson. *Nature* [*New Biol*] 242(118):157-158, 1973.

3535 ESTABLISHMENT OF HUMAN MALIGANANT MELANOMA CLONAL CELL LINES THAT SECRETE ECTOPIC ADRENOCORTICOTROPIN. *(E.)* Orth, D. N. (Dept. Med., Vanderbilt U., Nashville, Tenn.). *Nature* [*New Biol*] 242(114):26-28, 1973.

3536 BINDING OF [^{3}H]CONCANAVALIN A TO NORMAL AND TRANSFORMED CELLS. *(E.)* Noonan, K. D. (Dept. Biochem. Sci., Princeton U., N.J.) and M. M. Burger. *J Biol Chem* 248(12):4286-4292, 1973.

3537 HISTOLOGIC CLASSIFICATION AND GRADING OF MALIGNANCY IN CARCINOMA OF THE LARYNX. *(E.)* Jakobsson, P. A. (Karolinska Inst., Stockholm, Sweden), C.-M. Eneroth, D. Killander, G. Moberger and B. Martensson. *Acta Radiol* 12(1):1-8, 1973.

3538 PLATELET METABOLISM IN ACUTE LEUKEMIA. *(E.)* Cowan, D. H. (Case Western Reserve U. Sch. Med., Cleveland, Ohio). *J Lab Clin Med* 82(1):54-66, 1973.

3539 ADENOLYMPHOMA OF THE TONSILLAR FOSSA. *(E.)* Fahmy, S. (North Staffordshire Roy. Infirm., Stoke-on-Trent, England). *J Laryngol Otol* 87(7):675-679, 1973.

3540 OAT CELL CARCINOMA OF THE TRACHEA. *(E.)* Jash, D. K. (Tindal Gen. Hosp., Aylesbury, England). *J Laryngol Otol* 87(7):681-684, 1973.

3541 GASTRIC CARCINOMA FOLLOWING LONG-TERM GASTROENTEROSTOMY. REPORT OF TWO CASES. *(E.)* Carter, T. L. (Madison Gen. Hosp., Wisconsin) and W. Martel. *JAMA* 225(1):52-53, 1973.

3542 THOROTRAST-INDUCED HEPATOMA PRESENTING AS HYPERPARATHYROIDISM. *(E.)* Kiely, J. M. (Mayo Clin., Fdn., Rochester, Minn.), J. L. Titus and A. L. Orvis. *Cancer* 31(6):1312-1314, 1973.

3543 SEPARATION OF VIABLE FROM NONVIABLE TUMOR CELLS BY FLOTATION ON A FICOLL TRIOSIL MIXTURE. *(E.)* Vries, J. E. De (Netherlands Cancer Inst., Amsterdam), M. Van Benthem and P. Rümke. *Transplantation* 15(4):409-410, 1973.

3544 EFFECT OF LIVER REGENERATION ON THE NUMBER OF HAEMATOPOIETIC STEM CELLS. *(E.)* Osipova, T. V. (Acad. Med. Sci., USSR, Moscow) and G. J. Svet-Moldavsky. *Folia Biol* 19(2):157-161, 1973.

3545 GENETIC DISORDERS ASSOCIATED WITH CHROMOSOMAL INSTABILITY AND CANCER. *(E.)* German, J. (New York Blood Ctr., N.Y.). *J Invest Dermatol* 60(6):427-434, 1973.

3546 DEMONSTRATION OF IMPAIRED AUTOREGULATION IN TUMOURS AND CEREBRAL INFARCTION BY ANGIOGRAPHY. *(E.)* Huber, P. (Dept. Radiology, U. Berne, Switzerland). *Acta Radiol* [*Diagn*] *(Stockh)* 13(Part 1):115-122, 1972.

3547 COLONY FORMATION IN AGAR BY MURINE PLASMACYTOMA CELLS: POTENTIATION BY HEMOPOIETIC CELLS AND SERUM. *(E.)* Metcalf, D. (Roy. Melbourne Hosp., Victoria, Australia). *J Cell Physiol* 81(3):397-410, 1973.

3548 ERYTHROPOIETIC PROGENITORS CAPABLE OF COLONY FORMATION IN CULTURE: STATE OF DIFFERENTIATION. *(E.)* Gregory, C. J. (Ontario Cancer Inst., Toronto, Canada), E. A. McCulloch and J. E. Till. *J Cell Physiol* 81(3):411-420, 1973.

3549 FIBROSARCOMA ASSOCIATED WITH *CYSTICERCUS FASCIOLARIS*. *(E.)* Tucek, P. C. (Coll. Med., U. Florida, Gainesville), J. C. Woodard and A. F. Moreland. *Lab Anim Sci* 23(3):401-407, 1973.

3550 SIMILARITIES BETWEEN EXPERIMENTAL MOUSE LYMPHOMA CUTIS AND HUMAN DISEASE. *(E.)* Maruyama, Y. (No affiliation), T. Mariani, E. P. Engels and R. A. Good. *Work Prog* 107(3):687-688, 1973.

3551 CATECHOLAMINE STORAGE IN LIVER METASTASES OF A MALIGNANT CAROTID BODY TUMOUR. A BIOCHEMICAL AND MORPHOLOGICAL STUDY. *(E.)* Hörtnagl, H. (Dept. Pharmacology, U. Innsbruck, Austria), H. Hörtnagl, A. Propst, H. Schwingshackl, G. Weiser and H. Winkler. *Virchows Arch* [*Zellpathol*] 12(4):330-337, 1973.

3552 DNA-DEPENDENT RNA POLYMERASE FROM EHRLICH ASCITES TUMOR CELLS. I. PARTIAL PURIFICATION AND CHARACTERIZATION OF RNA POLYMERASE II. *(E.)* Natori, S. (Fac. Pharmaceutical Sci., U. Tokyo, Japan), K. Takeuchi and D. Mizuno. *J Biochem* 73(2):345-351, 1973.

3553 ULTRASTRUCTURE OF A BENIGN POLYPOID RHABDOMYOMA OF THE VAGINA. *(E.)* Leone, P. G. (St. Louis U. Hosp., Mo.) and H. B. Taylor. *Cancer* 31(6):1414-1417, 1973.

3554 BIOSYNTHESIS AND STORAGE OF CATECHOLAMINES IN PHEOCHROMOCYTOMA AND NEUROBLASTOMA CELLS. *(E.)* Itoh, T. (Ctr. Adult Dis., Osaka, Japan) and K. Ohmori. *J Lab Clin Med* 81(6):887-896, 1973.

3555 OSTEOGENIC POTENTIAL OF LEUKEMIC MARROW. *(E.)* Ratzan, R. J. (New England Med. Ctr. Hosp., Boston, Mass.), M. Tavassoli and W. H. Crosby. *Proc Soc Exp Biol Med* 143(2):391-394, 1973.

3556 SIGNIFICANCE OF SERUM COPPER LEVELS IN ADULT PATIENTS WITH HODGKIN'S DISEASE. *(E.)* Hrgovcic, M. (Texas Med. Ctr., Houston), C. F. Tessmer, F. B. Thomas, L. M. Fuller, J. F. Gamble and C. C. Shullenberger. *Cancer* 31(6):1337-1345, 1973.

3557 S-ADENOSYL-L-METHIONINE DECARBOXYLASE DURING LYMPHOCYTE TRANS FORMATION: DECREASED DEGRADATION IN THE PRESENCE OF A SPECIFIC INHIBITOR. *(E.)* Fillingame, R. H. (Dept. Biochem., U. Washington, Seattle) and D. R. Morris. *Biochem Biophys Res Commun* 52(3):1020-1025, 1973.

3558 IATROGENIC CUSHING'S DISEASE IN A BOY AFTER MISDIAGNOSIS OF SALT-LOSING VIRILIZING ADRENAL HYPERPLASIA: IMPAIRED METYRAPONE RESPONSE WITH FAILURE OF CATCH-UP GROWTH. *(E.)* Mendilaharzu, H. (St. U. New York, Upstate Med. Ctr., Syracuse), T. Gregory and L. I. Gasrdner. *J Pediatr* 83(1): 69-76, 1973.

3559 SEPARATION OF HUMAN LYMPHOID CELLS INTO G_1, S, AND G_2 CELL CYCLE POPULATIONS BY USE OF A VELOCITY SEDIMENTAION TECHNIQUE. *(E.)* Everson, L. K. (Natl. Cancer Inst., Bethesda, Md.), D. N. Buell and G. N. Rogentine, Jr. *J Exp Med* 137(2):343-358, 1973.

3560 PIGMENTED SKIN TUMORS. *(E.)* Davis, N. C. (Princess Alexandra Hosp., Brisbane, Australia), R. McLeod, J. Little and R. Quinn. *CA* 23(3): 160-170, 1973.

3561 THE FLUORESCENCE KARYOTYPES OF THREE HUMAN MENINGIOMAS WITH HYPERDIPLOID-HYPOTRIPLOID STEMLINES. *(E.)* Mark, J. (Huddinge Hosp., Sweden). *Acta Neuropathol (Berl)* 25:46-53, 1973.

3562 TUMOR-LIKE LESION OF THE BRAIN CAUSED BY *TRYPANOSOMA CRUZI*. *(E.)* De Queiroz, A. C. (Fac. Med., Bahia Federal U., Salvador, Brazil). *Am J Trop Med Hyg* 22(4):473-476, 1973.

3563 HISTOENZYMOLOGIC STUDY OF SECRETORY TUMOURS OF THE OVARY. *(Fr.)* Laffargue, P. (Fac. Med., Marseille, France), A. Chamlian, F. Laffargue and L. Adechy-Benkoel. *Arch Anat Pathol (Paris)* 21 (2):113-122, 1973.

3564 STUDIES OF ENDONEOPLASTIC TRANSFORMATION OF GLIAL TUMORS *IN VITRO* IN LONG-TERM TISSUE CULTURES AND SUBCULTURES. *(E.)* Kaluza, J. (Dept. Pharmacology, Polish Acad. Sci., Cracow) and Z. Kowalska. *Pol Med J* 11(6):1675-1686, 1972.

3565 COMPARISON BETWEEN ANATOMICAL AND SCANNING EVIDENCE OF SPLENIC INVOLVEMENT IN 62 LAPAROTOMIES FOR HODGKIN'S DISEASE. *(Fr.)* Robillard, J. (Francois-Baclesse Ctr., Caen, France), J. Cheguillaume, J. P. Nouel, L. V. Hoa, J. E. Couette and H. Piguet. *Nouv Rev Franc Hematol* 13(4):453-458, 1973.

3566 CARCINOMA OF THE COLON. SOME CONTROVERSIAL CONCEPTS. *(E.)* Barrett, F. A. (Wyoming St. Med. Soc.) and R. D. Yajko. *Rocky Mt Med J* 70 (7):38-41, 1973.

3567 KERATOACANTHOMA DEVELOPING IN A TATTOO. *(E.)* Cipollaro, V. A. (New York Hosp.-Cornell Med. Ctr., N.Y.). *Cutis* 11(6):809-810, 1973.

3568 PITUITARY CYTOLOGY IN A CASE OF PARANEOPLASTIC HYPERCORTICISM. *(Fr.)* Ketelbant-Balasse, P. (Fac. Med., Brussels Free U., Belgium), M. Herlant and J. L. Pasteels. *Ann Endocrinol (Paris)* 34(3):289-292, 1973.

3569 THE ULTRASTRUCTURE OF THE CELLS OF SQUAMOUS CELL CARCINOMA OF THE HUMAN UTERINE CERVIX. *(E.)* Chinese Acad. Med. Sci. *Chin Med J* (5):63-64, 1973.

3570 FACTORS INFLUENCING SPLENIC POLLING OF ERYTHROCYTES IN THE MYELO- AND LYMPHOPROLIFERATIVE SYNDROMES. *(E.)* Toghill, P. J. (Gen. Hosp., Nottingham, England) and S. Green. *Acta Haematol* 49(4):215-222, 1973.

3571 RAPID ARREST OF DNA SYNTHESIS BY $N^6,O^{2'}$-DIBUTYRYL CYCLIC ADENOSINE 3',5'-MONOPHOSPHATE IN CULTURED HEPATOMA CELLS. *(E.)* Van Wijk, R. (Natl. Jewish Nosp., Res. Ctr., Denver, Colo.), W. D. Wicks, M. M. Bevers and J. Van Rijn. *Cancer Res* 33(6):1331-1338, 1973.

3572 CARCINOEMBRYONIC ANTIGEN (CEA) PLASMA LEVELS IN UNTREATED CANCER PATIENTS AND PATIENTS WITH METASTATIC DISEASE. *(E.)* Concannon, J. P. (No affiliation), M. H. Dalbow and J. C. Frich, Jr. *Work Prog* 108(1):191-193, 1973.

3573 TRANSPORT OF α-OXOGLUTARATE IN MITOCHONDRIA OF TUMOUR CELLS AND ITS FUNCTIONAL IMPLICATIONS. *(E.)* Papa, S. (Dept. Biochem., U. Bari, Italy), G. Paradies, T. Galeotti, O. Dionisi and M. L. Eboli. *Nature* [*New Biol*] 242(116):86-87, 1973.

3574 B-LYMPHOCYTE MALIGNANCY. *(E.)* Alper, C. A. (Ctr. Blood Res., Boston, Mass.). *N Engl J Med* 289(3):154-155, 1973.

3575 URINARY ESTROGEN EXCRETION IN MEN WITH BREAST CANCER. *(E.)* Dao, T. L. (Roswell Park Mem. Inst., Buffalo, N.Y.), C. Morreal and T. Nemoto. *N Engl J Med* 289(3):138-140, 1973.

3576 STUDIES ON TUMOR VARIABILITY. IV. VARIATIONS *IN VIVO* OF $H_{10}Z$ TUMOR CELL POPULATION. *(E.)* Fadei, L. (Oncological Inst., Bucharest, Romania). *Oncology* 27(4):305-314, 1973.

3577 CHRONIC GRANULOCYTIC LEUKEMIA. PROLONGED SURVIVAL, MUSCLE INFILTRATION AND SEA-BLUE HISTIOCYTOSIS. *(E.)* Steinberg, W. H. (Jackson VA Ctr., Miss.) and B. J. Dreiling. *Am J Med* 55(1):93-98, 1973.

3578 PRIMARY RETICULUM CELL SARCOMA OF THE BREAST. *(E.)* Bushkin, F. L. (Coll. Med., U. Florida, Gainesville), S. D. E. Demian and K. K. Pierson. *Am J Surg* 126(1):63-66, 1973.

3579 MELKERSSON-ROSENTHAL SYNDROME, HODGKIN DISEASE, AND CORNEAL KERATOPATHY. *(E.)* Mulvihill, J. J. (Natl. Cancer Inst., Bethesda, Md.), W. W. Eckman, J. F. Fraumeni, Jr., R. M. Dryden and R. C. Young. *Arch Intern Med* 132(1):116-117, 1973.

3580 CYTOGENETIC STUDIES IN HAEMATOLOGICAL DISORDERS WHICH MAY TERMINATE IN ACUTE LEUKAEMIA. *(E.)* Jensen, M. K. (Rigs Hosp., U. Copenhagen, Denmark) and P. Philip. *Acta Med Scand* 193(4): 353-357, 1973.

3581 RECENT EXPERIENCE WITH BACTEREMIA IN PATIENTS PRESENTING CANCER. *(E.)* Klastersky, J. (Tumor Ctr., Brussels Free U., Belgium) and D. Weerts. *Eur J Cancer* 9(1):69-76, 1973.

3582 SEPARABLE DNA POLYMERASE ACTIVITIES IN HOST LIVER AND MORRIS HEPATOMAS. *(E.)* Ove, P. (U. Pittsburgh Sch. Med., Pa.), M. L. Coetzee and H. P. Morris. *Cancer Res* 33(6):1272-1283, 1973.

3583 PROTEIN TURNOVER IN MICROSOMAL SUBFRACTIONS OF LIVER AND MORRIS HEPATOMAS 7800 and 9618A. *(E.)* Moyer, G. H. (U. Wisconsin Med. Sch., Madison) and H. C. Pitot. *Cancer Res* 33(6):1316-1325, 1973.

3584 CHOLESTEROL AND PHOSPHOLIPID COMPOSITION OF MITOCHONDRIA AND MICROSOMES ISOLATED FROM MORRIS HEPATOMA 5123 AND RAT LIVER. *(E.)* Feo, F. (Gen. Path. Inst., Turin U., Italy), R. A. Canuto, G. Bertone, R. Garcea and P. Pani. *FEBS Lett* 33(2):229-232, 1973.

3585 ACTIN-LIKE FILAMENTS IN EHRLICH ASCITES TUMOR CELLS AND THEIR REACTION WITH HEAVY MEROMYOSIN. *(E.)* Kristensen, B. I. (Zoophysiological Lab. B, U. Copenhagen, Denmark), L. O. Simonsen and L. Pape. *Virchows Arch* [*Zellpathol*] 13(2):103-112, 1973.

3586 THE ROLE OF ACROCENTRIC CHROMOSOMES IN NUCLEOLAR ORGANIZATION. II. CONSTANCY OF THE NUCLEOLAR AREA INDEPENDENT OF THE NUMBER OF ACROCENTRIC CHROMOSOMES IN MENINGIOMA CELL CULTURES. *(E.)* Zankl, H. (Max Planck Inst. Psychiatry, Munich, Germany), S. Stengel-Rutkowski and K. D. Zang. *Virchows Arch* [*Zellpathol*] 13(2):113-118, 1973.

3587 ESTROGENIC EFFECT IN VAGINAL SMEARS IN CASES OF CARCINOMA *IN SITU* AND MICROINVASIVE CARCINOMA OF THE UTERINE CERVIX. *(E.)* Rubio, C. A. (Karolinska Inst., Stockholm, Sweden). *Acta Cytol* 17(4):361-365, 1973.

3588 ULTRASTRUCTURE OF A SACROCOCCYGEAL CHORDOMA. *(E.)* Gessaga, E. C. (Natl. Hosp., London, England), W. G. P. Mair and D. N. Grant. *Acta Neuropathol (Berl)* 25(1):27-35, 1973.

3589 THE FINE STRUCTURE OF A MYXOPAPILLARY EPENDYMOMA OF THE FILUM TERMINALE. *(E.)* Rawlinson, D. G. (Stanford U. Sch. Med., Calif.), M. M. Herman and L. J. Rubinstein. *Acta Neuropathol (Berl)* 25(1):1-13, 1973.

3590 STUDIES ON ENDOTOXIN-LIKE SUBSTANCES IN YOSHIDA SARCOMA CELLS. *(E.)* Kodama, M. (Kyoto Prefectural U. Med., Japan), H. Yajima, Y. Kubo, I. Hashimoto and Y. Katayama. *Jap J Med Sci Biol* 26(1):3-6, 1973.

3591 THE EFFECT OF INTERFERON ON THE SYNTHESIS OF RNA, PROTEINS, AND POLYRIBOSOMES IN L1210 MURINE LEUKEMIA CELLS. *(Fr.)* Brouty-Boye, D. (Cancer Res. Inst. Villejuif, France), M. Fiszman, A. Macieira-Coelho and I. Gresser. *C R Acad Sci* [*D*] *(Paris)* 276(11):1793-1795, 1973.

3592 METABOLIC AND THERAPEUTIC STUDIES OF A PATIENT WITH ACUTE LEUKEMIA AND SEVERE LACTIC ACIDOSIS OF PROLONGED DURATION. *(E.)* Wainer, R. A. (Natl. Cancer Inst., Baltimore, Md.), P. H. Wiernik and W. L. Thompson. *Am J Med* 55(2):255-260, 1973.

3593 ATYPICAL PATTERN OF UTILIZATION OF AMINO ACIDS FOR MITOCHONDRIAL PROTEIN SYNTHESIS IN HeLa CELLS. *(E.)* Costantino, P. (Division Biol., California Inst. Technology, Pasadena) and G. Attardi. *Proc Nat Acad Sci USA* 70(5):1490-1494, 1973.

3594 PANCREATIC CARCINOMA IN CHILDREN IN JAPAN. *(E.)* Tsukimoto, I. (Toho U. Sch. Med., Tokyo, Japan), K. Watanabe, J. -B. Lin and T. Nakajima. *Cancer* 31(5):1203-1207, 1973.

3595 DIFFERENTIAL DIAGNOSIS OF PRIMARY MALIGNANT HEPATOMA IN 569 BANTU MINEWORKERS. *(E.)* Geddes, E. W. (Crown Mines Hosp., Johannesburg, South Africa) and G. Falkson. *Cancer* 31(5): 1216-1221, 1973.

3596 DIARRHEA AND MEDULLARY CARCINOMA OF THE THYROID. *(E.)* Steinfeld, C. M. (Mayo Clin., Mayo Fdn., Rochester, Minn.), C. G. Moertel and L. B. Woolner. *Cancer* 31(5):1237-1239, 1973.

3597 CLEAR CELL CARCINOMA OF THE ENDOMETRIUM. CLINICAL, PATHOLOGIC, AND ULTRASTRUCTURAL FINDINGS. *(E.)* Silverberg, S. G. (Med. Coll. Virginia, Richmond) and L. S. de Giorgi. *Cancer* 31(5):1127-1140, 1973.

3598 CANCER OF THE PANCREAS. AVAILABLE EPIDEMIOLOGIC INFORMATION AND ITS IMPLICATIONS. *(E.)* Levin, D. L. (Natl. Cancer Inst., Bethesda, Md.) and R. R. Connelly. *Cancer* 31(5):1231-1236, 1973.

3599 SPONTANEOUS EPIDERMAL TUMOR IN AN ADULT NEWT, *CYNOPS PYRRHOGASTER*. *(E.)* Bryant, S. V. (Dept. Developmental, Cell Biol., U. California, Irvine). *Cancer Res* 33(3):623-625, 1973.

3600 ESTIMATION OF THE ASSOCIATION CONSTANT OF THE ESTROGEN-RECEPTOR COMPLEX IN HUMAN BREAST CANCER. *(E.)* Hahnel, R. (U. Western Australia, Dept. Obstetrics, Gynecology, Perth) and E. Twaddle. *Cancer Res* 33(3):559-566, 1973.

LIBRARY U. OF I. URBANA

ABE, S.
3121
ABELL, M.R.
3408*
ABRAHAMS, S.
3255
ABRAMCZUK, J.
3314*
ACHARJYO, L.N.
3217*
ACHUNG, B.G.
3130
ADAMS, A.
3128, 3166
ADECHY-BENKOEL, L.
3563*
ADELOYE, A.
3383*
ADILMAN, H.
3450*
AEIKENS, B.
3046
AGHAI, E.
3299*
AISENBERG, A.C.
3228
AKIMOV, A.A.
3116*
AKIYAMA, Y.
3257
ALBERT, A.E.
3139
ALBRECHT, C.F.
3037, 3038
ALEKSANDROVA, M.A.
3158
ALFORD, C.
3261
ALLEN, A.
3436*
ALLEN, E.L.
3498*
ALLEN, J.M.
3506*
ALONI, Y.
3126
ALPER, C.A.
3574*
ALTANAR, C.
3171*
ALTENAHR, E.
3342
ALTHOFF, J.
3078, 3333
ALVAREZ, J.
3337
ALVAREZ, Y.
3337
AMALRIC, F.
3508*
ANDERSEN, A.
3053
ANDERSON, J.R.
3240
ANDERSON, M.F.
3433*
ANDRE, R.
3446*
ANGHILERI, L.J.
3340
ANTOINE, J.C.
3302*
APFFEL, C.A.
3346
APPLETON, D.
3491*
ARAI, M.
3183*
ARATA, T.
3312*
ARCHER, V.E.
3334
ARGYRIS, T.S.
3075
ARMENTROUT, S.A.
3323*
ARNSTEIN, E.J.
3463*
ARTHAUD, J.B.
3082*
ASTASHOVA, E.A.
3447*
ATHANASIU, P.
3120, 3223*, 3224*
ATKIN, N.B.
3518*
ATKINSON, D.W.
3433*
ATTARDI, G.
3593*
ATYEO, R.
3421*
AUBERY, M.
3288*
AUCKER, J.
3138
AULT, K.A.
3229
AURIOL, M.
3443*
AVRAMEAS, S.
3302*
BABICH, G.L.
3398*
BACHMANN, A.E.
3417*
BADER, L.
3527*
BAILEY, I.C.
3382*
BAILEY, J.M.
3507*
BAILEY, R.E.
3030*
BAILLY, C.
3080*
BAIRD, W.M.
3042, 3044
BAKST, A.A.
3414*
BALCERZAK, S.P.
3498*
BALDA, B.R.
3194*
BALINSKY, D.
3038
BALL, G.
3153
BALUGH, K.
3515*
BALUYOT, R.
3414*
BARBAN, S.
3181*
BARBARESE, E.
3202*
BARBASON, H.
3083*, 3097*
BARBIERI, D.
3141
BARNES, W.T.
3449*
BARON, S.
3155
BARONI, C.D.
3088*
BARR, D.H.
3352
BARRETT, F.A.
3566*
BARSKI, G.
3141
BASERGA, R.
3429*
BASU, S.
3197*
BATAILLON, G.
3156, 3281*
BAYLE, J.
3521*
BECKER, V.
3160
BECKER, Y.
3186*
BEDIGIAN, H.G.
3213*
BEJU, D.
3197*
BELLHORN, R.W.
3211*
BENDA, R.
3175*
BER, A.
3317
BERARD, M.
3445*
BERGER, R.
3484*
BERNARD, S.
3508*
BERNHEIM, J.L.
3087*
BERNSTEIN, D.
3264
BERRY, K.W.
3143
BERSOHN, D.I.
3038
BERTONE, G.
3584*
BETZ, E.H.
3083*, 3097*
BEVERS, M.M.
3571*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

BHATIA, P.L.
3456*
BIARESE, V.
3444*
BICHEL, P.
3399*
BIGNER, D.D.
3252
BILLER, H.F.
3480*
BILLS, D.D.
3062
BINIAMINOV, M.
3299*
BIRKMAYER, G.D.
3194*
BISHOP, C.F.
3348
BLACK, L.M.
3209*
BLACKBURN, C.R.B.
3530*
BLANC, M.
3284*
BLEASEL, K.F.
3527*
BLOCH, K.J.
3228
BLOUGH, H.A.
3177*
BOESEN, E.A.M.
3448*
BOHUON, C.
3418*
BOIRON, M.
3017*, 3204*
BOKISCH, V.A.
3264
BOLOGNARI, A.
3439*
BOLTON, P.M.
3270
BONAL, T.
3446*
BONELLI, G.
3529*
BOONE, C.W.
3267
BOOTH, J.
3105*
BORDJUSHOV, J.N.
3103*
BOSE, P.K.
3386*
BOUCHER, J.
3119
BOUDREAULT, A.
3119
BOURGOIN, J.J.
3080*
BOURRILLON, R.
3288*
BOUTWELL, R.K.
3056
BOYLE, W.F.
3142
BOYNS, A.R.
3401*
BOZZI, A.
3402*
BRANDAU, H.
3326*
BRAS, G.
3466*
BRATSSLAVSKAIA, O.I.
3158
BRAUNSTEIN, G.D.
3516*
BREBOROWICZ, J.
3104*
BRENNAN, P.J.
3193*
BRIGGS, R.T.
3201*
BRINKER, R.A.
3500*
BROCHERIOU, C.
3443*
BROCKMAN, W.W.
3122
BRODOVSKY, H.
3497*
BRODZKA, W.
3280*
BRONNIKOVA, I.A.
3095*
BROOKES, P.
3042, 3044, 3071
BROOKS, J.W.
3495*
BROOKS, S.C.
3355
BROSS, I.D.J.
3389*
BROSSEAU, C.
3453*
BROUTY-BOYE, D.
3501*, 3591*
BROWN, N.K.
3517*
BROWN, R.R.
3416*
BROX, L.W.
3356
BROZMANOVA, E.
3359
BRUNNER, K.T.
3031*
BRYAN, G.T.
3066
BRYANT, S.V.
3599*
BRYSON, W.R.
3214*
BUCHAN, R.
3401*
BUCHNER, C.D.
3292*
BUCHSBAUM, H.J.
3377*
BUDA, J.
3247
BUDLYANSKIY, A.Y.
3404*
BUELL, D.N.
3559*
BUNNAG, B.
3473*
BUONASSISI, V.
3403*
BURDICK, J.F.
3233
BURDZY, K.
3076
BURGER, M.M.
3265, 3536*
BURKE, W.J.
3527*
BURLAKOVA, E.B.
3362*
BURNY, A.
3169
BURTON, P.R.
3201*
BUSBEE, D.L.
3107*
BUSCH, H.
3343
BUSELMAIER, W.
3092*
BUSHKIN, F.L.
3578*
BUTLER, T.C.
3458*
BYKOVSKY, A.F.
3131
CALDWELL, G.G.
3030*
CALLAWAY, E.
3222*
CALOTHY, G.
3196*
CAMERON, H.M.
3368*
CAMERON, I.L.
3432*
CAMPBELL, A.C.
3289*
CAMPOBASSO, O.
3519*
CANESE, M.G.
3519*
CANEVARI, S.
3254
CANTRELL, E.T.
3107*
CANUTO, R.A.
3529*, 3554*
CARDESA, A.
3078
CARLSON, I.H.
3416*
CARLSSON, C.A.
3274
CARTER, T.L.
3541*
CASAZZA, A.M.
3173*
CASSAI, E.
3190*
CATOVSKY, D.
3236*
CAVALLERO, A.
3086*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

CAVOTO, F.V.
3459*
CAYANIS, E.
3038
CERNEA, P.
3443*
CHAMLIAN, A.
3563*
CHAPENKO, S.V.
3158
CHARD, R.L.
3292*
CHARNEY, J.
3205*
CHASHINA, S.S.
3158
CHAUDHURI, M.R.
3319
CHAUVET, C.
3182*
CHAYOTH, R.
3055
CHEGUILLAUME, J.
3565*
CHIAO, J.W.
3264
CHIN, A.H.
3290*
CHIN, T.D.Y.
3030*
CHIRIGOS, M.A.
3208*
CHO, B.K.
3214*
CHO, H.Y.
3187*
CHOMETTE, G.
3443*
CHOPRA, S.A.
3368*
CHOUX, M.
3445*
CHOUX, R.
3445*
CHUKE, P.O.
3367*
CHURCH, E.C.
3267
CIANO, V.
3439*
CIAPPARELLI, L.
3054
CINATL, J.
3175*
CIPOLLARO, V.A.
3567*
CLARK, H.F.
3146
CLARK, R.P.
3390*
CLIFT, R.A.
3292*
COBB, L.M.
3448*
COCHRAN, A.J.
3240
COETZEE, M.L.
3582*
COGBILL, C.L.
3338
COHEN, I.
3514*
COHEN, M.M.
3146
COHEN, S.M.
3066
COLE, E.N.
3401*
COLE, J.W.
3006
COLIGAN, J.E.
3275
COLLARD, W.
3124
CONCANNON, J.P.
3572*
CONE, R.E.
3269
CONESA, L.C.G.
3417*
CONNELLY, R.R.
3598*
CONSTANTOPOULOS, A.
3375*
COSTANTINO, P.
3593*
COUETTE, J.E.
3565*
COURTNEY, R.J.
3206*
COWAN, D.H.
3538*
COX, K.O.
3226*
CREPIN, Y.
3282*
CRIFO, C.
3402*
CRISS, W.E.
3472*
CROCKER, S.G.
3327*
CRONIN, J.
3385*
CROSBY, W.H.
3375*, 3555*
CROVETTI, A.J.
3066
CSABA, G.
3372*
CUEFF, J.
3080*
CUNDY, R.L.
3478*
D'ABRERA, V.S.E.
3527*
D'HALLUIN, J.C.
3182*
DABASAKI, H.
3121
DALBOW, M.H.
3572*
DALENS, M.
3207*
DALES, S.
3189*
DAMJANOV, I.
3511*
DANIEL, R.W.
3238
DAO, T.L.
3575*
DARGENT, M.
3080*
DASZYNSKI, J.
3380*
DAVIS, H.L., JR.
3416*
DAVIS, N.C.
3560*
DAVIS, R.H.
3473*
DAVIS, S.
3514*
DAY, E.D.
3252
DE CLERCQ, E.
3190*
DE GIORGI, L.S.
3597*
DE HARVEN, E.
3197*
DE JESUS, P.O.
3414*
DE LAMIRANDE, G.
3392*
DE MOL, A.W.
3198*
DE QUEIROZ, A.C.
3562*
DE SALUM, S.B.
3417*
DE-THE, G.
3230
DEBRAY, J.
3446*
DECKERS, C.
3444*
DECKERS, P.
3515*
DEFENDI, V.
3164, 3196*
DEINHARDT, F.
3191*
DELARUE, J.C.
3418*
DELELLIS, R.A.
3515*
DELLA PORTA, G.
3254
DELOUX, J.
3446*
DEMIAN, S.D.E.
3578*
DEVINE, K.D.
3468*
DEVRIES, J.E.
3543*
DI MARCO, A.
3173*
DIBONA, D.D.
3498*
DIDIER, J.
3207*

DIKRANIAN, H.
3338
DIL'MAN, V.M.
3028*
DIONISI, O.
3573*
DIPPLE, A.
3042
DOMBOS, L.
3166
DONALDSON, B.
3064
DONNELLAN, B.
3469*
DRAKE, W.P.
3291*
DREILING, B.J.
3577*
DRUCKREY, H.
3047, 3084*
DRYDEN, R.M.
3579*
DUBE, M.K.
3456*
DUCOS, J.
3284*
DUH, F.G.
3187*
DUHAMEL, G.
3446*
DUNAWAY, G.A., JR.
3172*
DUNGWORTH, D.L.
3222*
DURNOV, L.A.
3410*
EAVES, G.
3324*
EEERLE, R.C.
3500*
EBOLI, M.L.
3573*
ECCLESTON, E.
3505*
ECKHART, W.
3129
ECKMAN, W.W.
3579*
EDELSTEIN, L.M.
3469*
EGUCHI, G.
3098*
EISENBRAND, G.
3046
EL-GOTHAMY, B.
3476*
ELIASSON, R.
3165
EMANOIL-RAVICOVITCH, R.
3204*
EMMETT, E.A.
3010
ENEROTH, C.M.
3537*
ENGELS, E.P.
3550*
ENJALBERT, L.
3207*
ENRILE, F.T.
3414*
EPSTEIN, M.A.
3130, 3150, 3153
EPSTEIN, S.M.
3055
ERBE, J.
3419*
ERICKSON, J.S.
3176*
ERNST, P.
3423*
ERTURK, E.
3066
ETZOLD, G.
3526*
EVANS, D.I.K.
3237
EVENSON, D.P.
3197*
EVERSON, L.K.
3559*
FADEI, L.
3576*
FAHMY, M.J.
3074
FAHMY, O.G.
3074
FAHMY, S.
3539*
FAILLE, A.
3423*
FAIRBURN, E.A.
3344
FALK, L.A.
3191*
FALKSON, G.
3595*
FANELLI, A.R.
3402*
FAREED, G.C.
3184*
FARROW, G.M.
3325*
FATTI, L.P.
3054
FAYEMI, A.O.
3514*
FEFER, A.
3292*
FEO, F.
3529*, 3584*
FERRER, J.F.
3234
FERRERO, A.
3077
FIALA, E.S.
3040
FIALA, S.
3040
FIELD, J.B.
3055
FILLINGAME, R.H.
3557*
FINK, E.
3490*
FISCHER, R.G.
3178*, 3179*, 3188*
FISCHER, W.
3024*
FISHER, W.B.
3323*
FISHMAN, P.H.
3507*
FISZMAN, M.
3591*
FISZMANAAND, M.
3501*
FLAXMAN, B.A.
3321*, 3459*
FOA, R.
3521*
FOCAN, C.
3083*, 3097*
FOLKMAN, J.
3330
FOLLANA, R.
3521*
FOMENKO, V.N.
3094*
FORET, J.E.
3398*
FORNI, L.
3271
FORREST, A.P.M.
3401*
FOSSATI, G.
3254
FOULIS, A.K.
3240
FRABLE, W.J.
3495*
FRANCKE, B.
3129
FRANSSILA, K.
3451*
FRAUMENI, J.F., JR.
3579*
FRAY, A.
3282*
FREEDMAN, S.O.
3311*
FREEMAN, C.B.
3289*
FREIDMAN, J.M.
3464*
FRENGLEY, P.A.
3393*
FRENKEL, N.
3180*
FRETZIN, D.F.
3428*
FREUND, R.
3230
FRICH, J.C., JR.
3572*
FRIEDMAN, A.H.
3211*
FRIEDMAN, L.
3067
FRIEDRICH, E.G.
3520*
FROSCH, Y.
3151
FUJIKURA, T.
3019*

FUJITA. S.
3476*
FUJIWARA. S.
3121
FUKUDA. Y.
3183*
FULLER. I.C.
3360
FULLER. L.M.
3556*
FUNK. D.D.
3292*
GAFFNEY. C.N.
3494*
GAHRTON. G.
3369*
GALEOTTI. T.
3573*
GALETTO. J.
3286*
GALLAGHER. R.E.
3203*
GALLAHER. W.R.
3177*
GALLIEN-LARTIGUE. O.
3212*
GALLO. R.C.
3203*. 3246
GALTON. D.A.
3286*
GAMBLE. J.F.
3556*
GARAPIN. A.C.
3015*
GARCEA. R.
3529*. 3584*
GARDNER. M.B.
3136
GARDNER. W.U.
3167
GARON. C.F.
3143
GASHNIKOVA. L.I.
3103*
GASRDNER. L.I.
3558*
GAUDIN. D.
3008
GAUTHIER. J.
3453*
GAZDAR. A.F.
3155
GEBHARDT. B.M.
3293*
GEDDES. E.W.
3595*
GELB. L.D.
3184*
GELBOIN. H.V.
3069
GEORGADZE. G.E.
3081*
GEORGII. A.
3442*
GEORGIOPULOS. E.
3490*
GERBER. M.A.
3514*
GERMAN. J.
3545*
GESSAGA. E.C.
3588*
GEVERS. W.
3073
GIBSON. J.P.
3102*
GILDEN. R.
3157
GILDEN. R.V.
3136
GILMER. K.
3345
GILSON. M.D.
3488*
GIMMY. J.
3084*
GIORDANO. C.
3381*
GIULIANI. F.
3173*
GLASGOW. L.A.
3221*
GLEW. R.H.
3258
GOGEBASHVILI. N.K.
3112
GOLD. P.
3311*
GOLDENBERG. M.M.
3427*
GOLDSTEIN. N.O.
3347
GOLDSTONE. A.H.
3294*
GOOD. R.A.
3277. 3550*
GORDIN. M.
3186*
GORIUS. J.B.
3365*
GORMLEY. M.B.
3464*
GOTTLIEB. A.A.
3374*
GRAHAM. F.L.
3147
GRAHAM. M.D.
3474*
GRAHAM. R.C.. JR.
3323*
GRANGER-JULIEN. M.
3119
GRANT. D.N.
3588*
GRASSER. A.
3301*
GRATTAROLA. R.
3420*
GRAY. S.
3497*
GREEN. I.
3306*
GREEN. M.
3124
GREEN. S.
3570*
GREENBERG. B.R.
3494*
GREENE. L.F.
3325*
GREGORY. C.J.
3546*
GREGORY. T.
3558*
GRESSER. I.
3501*. 3591*
GRIFFITHS. K.
3401*
GRISWOLD. D.P.
3328
GROVER. P. L.
3042
GRUNDMANN. E.
3295*
GRUNICKE. H.
3049
GUARINI. G.
3444*
GUBERGRITS. M.
3058. 3106*
GUERRIN. J.
3298*
GUNZ. F.W.
3387*
GUPTA. I.M.
3479*
GUPTA. R.K.
3310*
GUSEV. A.I.
3313*
GUTMANN. H.R.
3051
GYORKEY. F.
3343
HAGERSTRAND. I.
3045
HAGLID. K.
3274
HAGLID. K.G.
3318
HAGON. E.E.
3387*
HAHNEL. R.
3600*
HAJDU. S.I.
3440*
HAKAMA. M.
3451*
HALPEREN. S.
3216*
HALPERN. B.
3282*
HALTERMAN. R.
3534*
HALTERMAN. R.H.
3492*
HAMADA. C.
3276
HAMMARSKJOLD. M.L.
3154
HAMPRECHT. B.
3470*
HAN. P.F.S.
3218*

HANASH. K.A.
3325*
HANLEY, J.A.
3386*
HANSEN. H.H.
3503*
HARDY, W.D., JR.
3136
HARE. J.D.
3118
HAREL, P.
3453*
HARLOZINSKA, A.
3280*
HARNDEN, D.G.
3344
HARRALSON. J.D.
3513*
HARRIS, C.C.
3068
HARRIS, J.W.
3352
HARRIS, R.
3289*
HART, W.R.
3412*
HARVEIT, F.
3278*
HASHIMOTO. I.
3590*
HASSOUN, J.
3445*
HATANAKA, M.
3157
HATCH, G.G.
3268
HATTORI, S.
3455*
HAWN, L.
3494*
HAWTREY, A.O.
3073
HAY, D.
3161
HAYDEN, R.C., JR.
3476*
HAYDUK. K.
3394*
HAYES, D.M.
3481*
HELENUN, R.
3443*
HELLMAN, K.
3396*
HELLSTROM, I.
3232
HELLSTROM, K.E.
3232
HELWIG, E.B.
3428*
HEMENWAY, W.G.
3478*
HENDERSON, J.F.
3358
HENDRICKS, G.L.
3449*
HENKART, P.A.
3275
HENKIND, P.
3211*
HENLE, G.
3248
HENLE, W.
3248
HENRY, M.C.
3068
HERBERMAN, R.B.
3244, 3261, 3273
HERLANT, M.
3568*
HERMAN, M.M.
3589*
HERSCHMAN, H.R.
3532*
HERSEY, P.
3289*
HERSH, E.M.
3243
HETRICK, F.M.
3149
HIERHOLZER, J.C.
3143
HILDRUM, K.I.
3062
HILL, E.C.
3524*
HILLER, A.J.
3242
HIRAI, K.
3164, 3196*
HLADKA, M.
3171*
HO, H.C.
3248
HO, J.H.C.
3230
HOA, L.V.
3565*
HOBBS, J.R.
3308*
HOBIK, H.P.
3295*
HOCH, W.S.
3497*
HOCHBERG, M.
3330
HOGETVEIT, A.
3053
HOLLATZ, R.
3085*
HOLLINSHEAD, A.C.
3261
HOLZBERG, E.
3531*
HOLZMANN, S.
3036
HONG, S.C.L.
3138
HOPKINSON, J.
3075
HORAI, T.
3455*
HORSEIDE, V.
3278*
HORTNAGL, H.
3551*
HOSTY. T.A.
3520*
HOUSE, W.F.
3474*
HOUSSAY, D.
3365*
HOZOC, M.
3223*
HRGOVCIC, M.
3556*
HUAULME, D.
3212*
HUBER, P.
3546*
HUE, G.
3141
HUEBNER, R.J.
3125, 3134
HUNG, P.P.
3127
HUNIG, S.
3047
HUNSMANN, G.
3135
HUNT, R.D.
3150
HUNTER, R.J.
3481*
HURLBERT, R.B.
3357
HUTCHESON, J.
3512*
HYDE, R.M.
3300*
IAGUBOV, A.S.
3093*
IAKIMENKO, L.V.
3272
IJIMA, Y.
3373*
ILYIN, K.V.
3131
INBAR, M.
3341
IONESCU, T.
3223*
IRINO, S.
3121
IRVINE, W.J.
3294*
ISHCHENKO, I.M.
3405*
ISHIMOTO, A.
3117
ITO, S.
3307*
ITO, Y.
3117
ITOH, T.
3554*
IVANKOVIC, S.
3084*, 3066*
IWA, N.
3174*
IYENGAR, J.R.
3064
IYPE, P.T.
3070

JACCBS, A.
3400*
JACOBSON, A.
3436*
JAIN, H.K.
3456*
JAKOBSSON, P.A.
3537*
JANISCH, W.
3032*
JARPLID, B.
3111
JARRETT, O.
3161
JARRETT, W.
3464*
JARVIS, J.
3153
JASH, D.K.
3540*
JASMIN, C.
3018*
JENSEN, M.K.
3580*
JOGANNSEN, M.I.
3297*
JOHANSSON, K.
3199*
JOHNSON, A.M.
3009
JOHNSON, F.L.
3292*, 3421*
JOHNSON, F.S.
3002
JONCAS, J.
3119
JONES, P.A.
3073
JONES, P.A.E.
3400*
JORDAN, J.A.
3506*
KAASA, L.J.
3354
KAGAN, G.Y.
3200*
KAHN, L.B.
3220*
KAKUNAGA, T.
3065
KALEY, M.M.
3411*
KALUZA, J.
3564*
KAPLAN, A.S.
3176*
KARAKASIS, D.
3465*
KARASEK, M.A.
3384*
KARNOVSKY, M.J.
3229
KARU, T.
3058
KASILI, E.G.
3368*
KASPER, E.
3450*
KASPRZAK, K.S.
3104*
KASSEL, R.L.
3266
KATAYAMA, Y.
3590*
KATO, N.
3307*
KATO, S.
3174*, 3257
KATSUTA, H.
3379*
KATZ, D.H.
3227
KAUFMAN, D.G.
3068
KAUFMANN, W.
3394*
KAWAI, S.
3019*
KAWAKAMI, T.G.
3222*
KAYMAN, S.C.
3258
KEANE, A.T.
3113
KEAST, D.
3226*
KELLEN, J.A.
3091*
KELLER, R.
3249
KELLOFF, G.J.
3125, 3134
KERN, E.R.
3221*
KERR, L.
3294*
KERSEY, J.H.
3277
KETELBANT-BALASSE, P.
3568*
KETELS, K.V.
3068
KEYSELL, G.R.
3105*
KHIN, U.
3450*
KIE, F.E.
3034*
KIELY, J.M.
3542*
KILLANDER, D.
3537*
KILLMANN, S.A.
3423*
KIM, S.C.
3358
KIMBRELL, O.C., JR.
3354
KING, V.P.
3388*
KINGSBURY, E.W.
3242
KINKEL, H.J.
3036
KINOSHITA, N.
3069
KIPDAN, G.V.
3410*
KIRSO, U.
3058, 3106*
KISSIN, B.
3411*
KLASTERSKY, J.
3581*
KLEIN, G.
3128, 3166
KLEIN, R.
3157
KLENER, V.
3114*
KLEPPING, C.
3298*
KLEY, H.K.
3326*
KNAB, D.R.
3488*
KOBAYASHI, H.
3235, 3305*
KODAMA, M.
3590*
KODAMA, T.
3235, 3305*
KOLB, J.P.
3279*
KOLEANICHENKO, T.S.
3043
KUMARDINA, G.A.
3407*
KONDA, S.
3239, 3256
KORANT, B.D.
3216*
KOS, W.L.
3149
KOSKE, R.E.
3041
KOSS, M.N.
3266
KOTLAREK-HAUS, S.
3280*
KOWALSKA, Z.
3564*
KOWOLLIK, G.
3526*
KOZLOV, A.P.
3116*
KRAIN, L.S.
3316*
KRETSCHMER, H.
3301*
KRIEG, K.
3033*
KRISTENSEN, B.I.
3585*
KRONBERGER, L.
3490*
KRSMANOVIC, K.
3182*
KRUGMAN, M.F.
3454*
KRULICK, M.
3446*
KUBISZ, P.
3378*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

KUBO, Y.
3590*
KUDELEVA, G.V.
3158
KUKAIN, R.A.
3158
KUMAR, S.
3345
KUPFER, S.
3464*
KURAMOTO, H.
3510*
KURASHIGE, S.
3259
KUWATA, T.
3219*
KUZMA, J.F.
3437*
KUZUMAKI, N.
3235
KWAN, H.C.
3248
LACOUR, J.
3484*
LAFFARGUE, F.
3563*
LAFFARGUE, P.
3563*
LAFFERTY, K.J.
3315*
LAIRD, H.M.
3161
LANE-BROWN, M.M.
3509*
LANE, I.
3151
LANGE, J.
3135
LANGEN, P.
3526*
LARSEN, C.J.
3204*
LARSON, E.J.
3102*
LATTER, T.
3356
LAU, D.T.L.
3222*
LAUFS, R.
3215*
LAURITSEN, J.G.
3351
LAWLEY, P.D.
3048, 3060
LAWRENCE, A.M.
3012*
LAWSON, T.A.
3099*
LECOU, C.
3283*
LEDIS, S.
3353
LEE, J.A.H.
3025*
LEE, T.N.H.
3122
LEERS, W.D.
3391*
LEHMEYER, J.E.
3395*
LEKLEM, J.
3416*
LEMON, H.M.
3030*
LENNETTE, E.H.
3142
LEONARD, B.J.
3505*
LEONE, P.G.
3553*
LERNER, M.P.
3532*
LERNER, R.
3411*
LESPINATS, G.
3279*
LEVALLOIS, C.
3283*
LEVAN, G.
3424*
LEVENTHAL, B.
3534*
LEVENTHAL, B.G.
3492*
LEVIN, D.L.
3376*, 3598*
LEVINE, A.J.
3152
LEVINE, A.S.
3185*
LEVINE, P.H.
3168
LEVITAN, D.B.
3177*
LEVY, J.P.
3016*
LEWIS, A.M., JR.
3185*
LIBBEY, L.M.
3062
LICHTMAN, M.A.
3393*
LIEEENEERG, N.V.D.W.
3037
LIEEER, M.M.
3145
LIJINSKY, W.
3090*
LIKHITE, V.
3375*
LILLY, F.
3195*
LIN, J.B.
3594*
LINDSTEN, J.
3369*
LINNA, T.J.
3192*
LINNIK, A.
3106*
LIPPMAN, M.
3534*
LIPPMAN, M.E.
3492*
LIPTON, A.
3140
LITTLE, J.
3560*
LITTLE, J.B.
3332
LIVINGTON, D.M.
3145
LIZONCVA, A.
3171*
LJUBOVIC, E.
3431*
LOBER, M.
3225*
LOCKE, E.R.
3355
LONG, C.W.
3157
LONG, J.C.
3228
LORIE, J.I.
3410*
LORINET, A.M.
3282*
LOVISETTO, P.
3444*
LOWE, J.S.
3505*
LOWENFELS, A.B.
3001
LU, S.T.
3109
LUBCEINSKI, H.P.
3034*
LUCAS, R.N.
3494*
LUDEEN, D.E.
3063
LUECKE, D.H.
3178*, 3179*, 3188*
LUNDIN, F.E.
3334
LUNGER, P.D.
3146
LUNGU, M.
3120, 3223*, 3224*
LYSTER, W.R.
3336*
MA, M.H.
3530*
MAC KAY, E.N.
3329
MAC KAY, I.R.
3287*
MAC LENNAN, I.C.M.
3289*
MAC LECD, R.M.
3395*
MAC SWEEN, J.M.
3287*
MACASAET, M.A.
3377*
MACIEIRA-COELHO, A.
3501*, 3591*
MACKEN, M.F.
3430*
MAGOUS, R.
3283*
MAIR, W.G.P.
3588*

M J. S.
3380*
MAK, S.
3210*
MALEJKA-GIGANTI, D.
3051
MAMONTOVA, L.N.
3363*
MANY, A.
3299*
MARCHALONIS, J.J.
3269
MARCHOW, L.
3104*
MARDINEY, M.R., JR.
3291*
MARIANI, T.
3550*
MARK, J.
3424*, 3561*
MARSH, R.J.
3139
MARTEL, N.
3541*
MARTENSSON, B.
3537*
MARTHY, Y.
3284*
MARTIN, F.
3298*
MARTIN, M.A.
3184*
MARTIN, N.D.
3494*
MARTIN, R.R.
3107*
MARTIS, C.
3465*
MARUYAMA, Y.
3550*
MASSICOT, J.G.
3208*
MASUKAWA, T.
3437*
MATSUDA, M.
3455*
MATTHEW, T.M.
3367*
MATTHIES, E.
3245
MATTINGLY, R.F.
3437*
MAVLIGIT, G.M.
3243
MAYS, C.W.
3113
MAZUROV, V.I.
3406*
MC ALLISTER, R.M.
3136, 3267
MC BEAN, J.B.
3468*
MC BRIDE, C.M.
3243
MC CARTY, B.
3409*
MC CORMICK, G.M.
3039
MC CORMICK, K.J.
3268
MC COY, M.G.
3353
MC CULLOCH, E.A.
3548*
MC GOWAN, L.
3473*
MC KEE, R.W.
3409*
MC LAUGHLIN, H.
3308*
MC LEOD, R.
3560*
MC MAHON, M.J.
3397*
MC MANUS, J.
3247
MC MORRIS, F.A.
3370*
MCDONALD, J.M.
3309*
MEEKER, W.R., JR.
3493*
MELEYE, S.W.
3384*
MELENDEZ, L.V.
3153, 3215*
MELIA, D.F.
3509*
MELNICK, J.L.
3193*, 3206*
MELNYKOVYCH, G.
3348
MENDILAHARZU, H.
3558*
MENDONCA, D.R.
3457*
MENDUKE, H.
3497*
MERK, F.B.
3515*
MERRILL, J.M.
3025*
MESTWERDT, W.
3326*
METCALF, D.
3547*
METZ, E.N.
3498*
MICHAELSON, S.M.
3109
MILES, W.F.
3064
MILLER, F.M.
3400*
MILLER, G.G.
3131
MILLER, R.G.
3255
MILSTIEN, J.B.
3184*
MILTON, P.J.D.
3327*
MIR, M.
3338
MIRAKIAN, A.
3091*
MITELMAN, F.
3424*
MITSUHASHI, S.
3259
MIYOSHI, I.
3121
MIZELL, M.
3124
MIZUNG, D.
3307*, 3552*
MIZUNO, F.
3123
MOBERGER, G.
3537*
MOERTEL, C.G.
3596*
MOHANTY, G.C.
3217*
MOHR, U.
3078, 3333
MOKEEVA, R.A.
3313*
MOLFESE, G.
3444*
MOLLO, F.
3519*
MOLONEY, W.C.
3388*
MOLZHANINOY, Y.V.
3405*
MONDOVI, B.
3402*
MONTAGNIER, L.
3014*
MOON, R.C.
3039
MOORE, D.H.
3205*
MORAN, S.
3151
MORE, I.A.R.
3482*
MORELAND, A.F.
3549*
MORGAN R.W.
3007
MORGAN, W.W.
3432*
MORGUNOVA, T.D.
3200*
MORLEY, A.R.
3491*
MORREAL, C.
3575*
MORRIS, D.R.
3557*
MORRIS, H.P.
3582*
MOSCOVICI, C.
3159
MOSKALIK, K.G.
3116*
MOUSSERON-CANET, M.
3283*
MOYER, G.H.
3583*
MUELLER, J.F.
3118

MUGGIA, F.M.
3503*
MUHLBOCK, O.
3005
MULLER, O.
3326*
MULLER, R.
3138
MULVIHILL, J.J.
3579*
MUNSON, B.R.
3132
MURATORE, R.
3521*
MURPHY, G.
3238
MURPHY, J.F.
3506*
MYCINO, N.
3144
MYERS, D.K.
3110
NAGAEVA, L.I.
3158
NAHMIAS, A.J.
3004
NAIK, G.G.
3367*
NAITO, M.
3174*, 3257
NAJJAR, V.A.
3375*
NAJMAN, A.
3446*
NAKAJIMA, S.
3276
NAKAJIMA, T.
3594*
NAKAMURA, M.
3183*
NAKAO, Y.
3256
NARASIMHAM, G.V.
3335
NASTAC, E.
3120, 3223*, 3224*
NATHANS, D.
3122
NATORI, S.
3552*
NAUFAL, P.M.
3467*
NEEL, H.B.
3468*
NEILL, V.M.
3300*
NEIMAN, P.
3292*
NELSON, P.G.
3370*
NEMOTO, T.
3575*
NETTESHEIM, P.
3090*
NEVILLE, A.M.
3448*
NEWBERNE, J.W.
3102*
NEWCOMER, V.D.
3316*
NEWELL, G.R.
3225*
NG, A.B.P.
3525*
NICHOLSON, D.E.
3374*
NICOLSON, M.
3136
NIKULIN, A.
3431*
NISHIHARA, H.
3455*
NISONOFF, A.
3241
NOONAN, K.D.
3536*
NORRIS, H.J.
3412*
NOUEL, J.P.
3565*
NUSSBAUM, M.
3475*
NYORMOI, O.
3166
O'CONOR, G.
3168
O'NEILL, B.
3462*
ODEKU, E.L.
3383*
ODLAND, G.F.
3528*
OGURA, J.H.
3480*
OHAYON, E.
3284*
OHMORI, K.
3554*
OKAZAKI, T.
3219*
OKOS, A.
3286*
OKULOV, V.B.
3303*, 3364*
OKUN, M.R.
3469*
OKUNEVA, L.A.
3095*
OLDHAM, R.R.
3512*
OLMER, J.
3521*
OLSHEVSKY, U.
3186*
ONO, K.
3174*
ORENGO, A.
3357
ORTALDO, J.R.
3273
ORTH, D.N.
3535*
ORVIS, A.L.
3542*
OSATO, T.
3123
OSHIRO, L.S.
3142
OSIPOVA, T.V.
3544*
OSSWALD, H.
3057
OSUNKOYA, B.O.
3383*
OVE, P.
3582*
OVERALL, J.C., JR.
3221*
OZZELLO, L.
3403*
PADGETT, B.L.
3139
PAGE, D.L.
3512*
PAL'MINA, N.P.
3362*
PALMQUIST, M.
3471*
PALUTKE, M.
3309*
PANALAKS, T.
3064
PANI, P.
3584*
PAPA, S.
3573*
PAPE, L.
3585*
PARADIES, G.
3573*
PARKER, F.
3528*
PARKER, J.C.
3493*
PARKER, J.C., JR.
3513*
PARKER, R.
3477*
PARKS, W.P.
3163
PASCAL, R.R.
3266
PASCHER, G.
3531*
PASQUALINI, C.D.
3061
PASTEELS, J.L.
3568*
PATCHEFSKY, A.S.
3497*
PATEL, R.P.
3469*
PATERSON, A.R.P.
3358
PAUL, P.
3022*
PAVLOVSKY, A.
3417*
PAWELEK, J.
3533*
PAWLAK, L.L.
3241
PEACOCK, J.H.
3370*

PECK, W.A.
3393*
PEDERSEN, E.
3053
PEEREBOOM, J.W.C.
3011*
PEGG, A.E.
3052
PELLISSIER, J.F.
3445*
PERNIS, B.
3271
PERUNACE, L.
3082*
PERRY, S.
3492*, 3534*
PERSICO, F.J.
3374*
PETERS, R.L.
3125, 3134
PETERSEN, H.S.
3499*
PETREA, I.
3167
PETROCIV, S.
3175*
PETROV, N.S.
3406*
PFEIFFER, R.
3304*
PHILIP, P.
3580*
PHILIPSON, L.
3199*
PHILLIPS, A.J.
3434*
PHILLIPS, R.A.
3255
PHILLIPS, S.M.
3250
PICKART, L.
3339
PICKREN, J.W.
3389*
PIENKOWSKI, M.
3314*
PIERSON, K.K.
3578*
PIGIET, V.
3165
PIGUET, H.
3565*
PIHKO, H.
3260
PIKULA, B.
3431*
PINARD, J.
3392*
PITCHER, C.
3308*
PITOT, H.C.
3583*
PLACTICA, O.
3282*
PLAGEMANN, P.G.
3419*
PLAISNER, V.
3175*
PLAMENAC, P.
3431*
PLATT, D.
3034*
PLISS, G.B.
3297*
PLUESS, H.J.
3438*
POGO, B.G.T.
3169*
POLLACK, R.S.
3485*
POLLIACK, A.
3487*
POMEROY, B.S.
3022*
PONSOLD, W.
3245
PONTEN, J.
3435*
POOLE, D.T.
3458*
POPP, F.A.
3089*
POPPI, V.
3444*
PORSCHKE, D.
3361
PORT, C.D.
3068
POSTNIKOVA, Z.A.
3200*
POTTER, C.W.
3263
POUND, A.W.
3099*
POUPON, M.F.
3279*
POUR, P.
3078, 3333
PRASAD, B.N.
3479*
PRASAD, K.N.
3345, 3426*
PREUSSMANN, R.
3086*
PRIS, J.
3207*
PROPPING, P.
3092*
PROPST, S.
3551*
PRUZANSKI, W.
3391*
PUSCHENDORF, B.
3049
QUIGLEY, M.M.
3020*
QUINLAN, W.J.
3109
QUINN, R.
3560*
RABIN, B.R.
3390*
RABIN, H.
3150, 3153
RABOURDIN, A.
3282*
RABSTEIN, L.S.
3125, 3134
RADZISZEWSKI, J.
3380*
RAGAB, A.H.
3480*
RAGSDALE, B.D.
3523*
RAICK, A.N.
3076
RAINBOW, A.J.
3210*
RAJU, M.S.V.
3335
RAJYA, B.S.
3217*
RAKOVSKAYA, I.V.
3200*
RALPH, P.
3413*
RAM MURTHY, B.
3335
RAMIREZ, J.A.
3408*
RAMON, T.
3436*
RAMUT, B.
3299*
RAMULU, C.
3335
RASHEED, S.
3136
RASKAS, H.J.
3133
RATHSACK, R.
3108*
RATZAN, R.J.
3555*
RAWLINGS, W.
3225*
RAWLINSON, D.G.
3589*
RAYDT, G.
3049
REAGAN, J.W.
3525*
REALE, E.
3442*
REDDY, C.R.R.M.
3335
REDDY, D.V.R.
3209*
REED, R.J.
3225*
REED, W.B.
3441*
REEMTSMA, K.
3247
REES, R.C.
3263
REHACEK, J.
3178*, 3179*, 3188*
REICHARD, P.
3165
REINERT, H.
3526*
RETIEF, D.H.
3054

REYNOLDS. R.D.
3494*
RHIM. J.S.
3187*
RHO. Y.C.
3133
RICE. J.M.
3035
RICHARDSON. J.D.
3493*
RICHMOND. H.
3421*
RICHTER. R.
3280*
RICKINSON. A.B.
3153
RIGGS. J.L.
3142
RIKANS. L.E.
3063
RILEY. P.A.
3356
RINGELHANN. B.
3322*
RITTERSON. A.L.
3118
RITZI. E.M.
3152
ROBBINS. P.W.
3137
ROBERTS-DORY. C.
3242
ROBERTS. M.M.
3401*
ROBILLARD. J.
3565*
ROBIN. J.
3204*
RODRIGUES. D.
3244
ROEHM. C.
3140
ROELANTS. G.
3271
ROELS. H.
3087*
ROGENTINE. G.N.. JR.
3559*
ROHOVSKY. M.W.
3102*
ROHRBORN. G.
3092*
ROHRSCHNEIDER. L.R.
3056
ROIZMAN. B.
3180*
ROLLINGHOFF. M.
3253. 3269
ROMERO. R.
3311*
RONGEY. R.W.
3136
ROSE. J.A.
3143
ROSENKRANZ. H.S.
3186*
ROSENLOF. R.C.
3030*
ROSENSTREICH. D.L.
3306*
ROSENTHAL. L.
3316*
ROSIN. H.D.
3454*
ROSMAN. M.
3502*
ROSS. G.T.
3516*
ROSS. M.H.
3466*
ROSSI. P.
3500*
ROUBAL. J.
3175*
ROUESSE. J.
3418*
ROUSE. B.T.
3253
ROWINSKI. J.
3314*
RUBERTI. R.F.
3368*
RUBIN. A.D.
3514*
RUBINSTEIN. L.J.
3589*
RUBIO. C.A.
3587*
RUDDON. R.W.
3063
RUMKE. P.
3543*
RUNTSIS. AI.
3158
RUOSLAHTI. E.
3231. 3260
RUSTIA. M.
3078
RUTERS. J.
3262
RUTMAN. R.J.
3347
RYDELL. R.E.
3051
SAAL. F.
3061
SACHS. L.
3341
SAEED. S.M.
3430*
SAEGER. W.
3342
SAIKI. J.H.
3290*
SAL'NIKOVA. L.S.
3094*
SAMSO. A.
3204*
SANCHO. H.
3418*
SANDER. J.
3046
SANDISON. A.T.
3482*
SANDO. I.
3478*
SANTORO. A.S.
3402*
SARKAR. N.H.
3205*
SASAKI. M.S.
3059
SASTRY. P.
3335
SAUERWEIN. H.
3202*
SAVINO. A.
3440*
SAVLUCHINSKAIA. L.
3106*
SAVLUCHINSKAYA. L.A.
3043
SAXEN. E.
3451*
SCANLAN. R.A.
3062
SCELSI. R.
3088*
SCHAFER. W.
3135
SCHAFFER. M.
3357
SCHAUENSTEIN. E.
3490*
SCHAUR. R.F.
3490*
SCHEIFFARTH. F.
3262
SCHEIKE. O.
3350
SCHMAHL. D.
3086*. 3101*
SCHMIDT. C.G.
3349
SCHNEIDERMAN. M.A.
3376*
SCHNIPPER. L.E.
3185*
SCHOEFL. G.I.
3315*
SCHREIBER. D.
3032*
SCHUBERT. J.C.F.
3531*
SCHULTZ. J.
3470*
SCHWARTZ. L.
3061
SCHWEINSBERG. F.
3046
SCHWINGSHACKL. H.
3551*
SCOLNICK. E.M.
3163
SCORNIK. J.C.
3417*
SCRIBNER. J.D.
3035
SEEBER. S.
3349
SEEMAYER. N.H.
3164
SEIFERT. B.
3045

SELLERS, A.H.
3329
SEN, L.
3061, 3417*
SEN, N.P.
3064
SENDO, F.
3305*
SERGEANT, A.
3182*
SERMAN, D.
3511*
SESSIONS, D.G.
3480*
SETH, H.N.
3366*
SEVOIAN, M.
3213*
SHABAD, L.
3106*
SHABAD, L.M.
3027*, 3043
SHAH, K.V.
3238
SHAH, S.A.
3060
SHEARS, B.
3069
SHEPPARD, J.R.
3426*
SHEVACH, E.M.
3306*
SHINITZKY, M.
3341
SHITOVA, T.F.
3158
SHRIMALI, R.L.
3456*
SHUBIK, P.
3050, 3077
SHULLENBERGER, C.C.
3556*
SHUM, H.Y.
3462*
SHURYBIN, D.Y.
3406*
SHUSTER, J.
3311*
SIEGLER, R.
3151
SILVA, R.F.
3159
SILVERBERG, S.G.
3495*, 3597*
SILVERSTEIN, S.
3180*
SIMARD, R.
3508*
SIMIZU, B.
3219*
SIMONSEN, L.O.
3585*
SIMPKINS, H.
3202*
SIMPSON-HERREN, L.
3328
SIMS, H.
3155
SIMS, P.
3042, 3105*
SIMU, G.
3372*
SINGH, M.
3366*
SISSONS, H.A.
3113
SKALPE, I.O.
3483*
SKROVINA, B.
3359
SKUCAS, J.
3500*
SLAGA, T.J.
3035
SMETANA, K.
3343
SMETS, L.A.
3162
SMITH, E.C.
3172*
SMITH, H.S.
3242
SMITH, M.P.
3517*
SMITH, R.T.
3239, 3256
SMYTH, J.R., JR.
3218*
SNYDER, C.
3090*
SNYDER, F.F.
3358
SNYDER, S.P.
3222*
SOBELS, F.H.
3021*
SOHIER, R.
3230
SOKOL, F.
3170*
SOLTER, D.
3511*
SOULE, H.
3355
SOUTHARD, M.
3497*
SPAHN, G.J.
3125, 3134, 3155
SPARROS, L.
3282*
SPECTOR, B.D.
3277
SPIEGELMAN, S.
3169
STALSBERG, H.
3496*
STATHOPOULOS, G.
3286*
STAUFER, U.G.
3438*
STAVROU, D.
3274, 3318
STEEVES, R.A.
3195*
STEFANI, D.V.
3313*
STEINBERG, W.H.
3577*
STEINER, S.
3193*, 3206*
STEINFELD, C.M.
3596*
STEINHOFF, D.
3101*
STEKAR, J.
3047
STEMPLE, D.
3233
STENBACK, F.
3050
STENBACK, F.G.
3077
STENGEL-RUTKOWSKI, S.
3586*
STEPHENS, R.L.
3503*
STEWART, A.M.
3115*
STICH, H.F.
3041
STILLER, D.
3096*
STILLSTROM, J.
3251
STOIAN, M.
3223*
STONE, H.O.
3216*
STORAASLI, J.P.
3525*
STORB, R.
3292*
STOREN, E.J.
3483*
STREETER, A.M.
3462*
STROM, R.
3402*
SU, S.H.
3411*
SUGIYAMA, T.
3371*
SUICU-FOCA, N.
3247
SUNDARESHWAR, B.
3335
SUTTON, P.M.
3356
SUVALOVA, T.I.
3093*
SVET-MOLDAVSKY, G.J.
3544*
SVOBODA, V.
3114*
SZAFARZ, D.
3212*
TABARES, E.
3337
TACHOVSKY, T.G.
3118
TAKAMIZAWA, H.
3486*
TAKAOKA, T.
3379*

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

TAKEICHI, N.
3235
TAKEUCHI, K.
3552*
TAKSDAL, S.
3496*
TAN, K.B.
3170*
TANAKA, T.
3312*
TANAKA, Y.
3312*
TANTACHAMRUM, T.
3333
TAPIA ACUNA, R.
3489*
TARASOV, V.A.
3112
TARMY, E.M.
3071
TATARSKIY, M.L.
3407*
TATEISHI, R.
3455*
TAVASSOLI, M.
3555*
TAVITIAN, A.
3204*
TAYLOR, A.N.R.
3344
TAYLOR, G.N.
3113
TAYLOR, H.B.
3553*
TAYLOR, H.W.
3090*
TAYLOR, R.W.
3327*
TEICHMANN, B.
3108*
TEMPEL, K.
3085*
TERRY, W.D.
3275
TESSMER, C.F.
3556*
THALER, M.M.
3339
THAYER, L.
3339
THERON, J.J.
3037
THIELE, J.
3442*
THIEM, T.
3247
THOMAS, E.D.
3292*
THOMAS, F.B.
3556*
THOMAS, G.H.
3397*
THOMPSON, E.B.
3492*, 3534*
THOMPSON, J.
3504*
THOMPSON, K.D.
3192*
THOMPSON, W.L.
3592*
THORTON, H.
3124
THUNOLD, S.
3278*
THURMAN, G.B.
3113
TIBBETTS, C.
3199*
TIEPOLO, L.
3425*
TILL, J.E.
3548*
TIMBERG, R.
3487*
TING, C.C.
3244, 3273
TING, R.C.
3203*
TITUS, J.L.
3542*
TKACZEVSKI, L.Z.
3061
TOBER, C.L.
3132
TODARO, G.J.
3145, 3246
TODD, C.W.
3275
TOGA, M.
3445*
TOGHILL, P.J.
3570*
TOKER, C.
3454*
TONOMURA, A.
3059
TORIUMI, J.
3373*
TOWNSEND, G.L.
3468*
TRENTIN, J.J.
3268
TROUILLAS, P.
3023*
TRUJILLO, J.M.
3290*
TRUSOV, L.N.
3407*
TSOU, K.C.
3353
TSUBOI, A.
3429*
TSUBOI, K.
3379*
TSUBOTA, T.
3121
TSUKIMOTO, I.
3594*
TUCEK, P.C.
3549*
TWADDLE, E.
3600*
UCCINI, S.
3088*
UETAKE, H.
3276
UMANSKII, I.A.
3272
UMEDA, M.
3070
UNANUE, E.R.
3227, 3229
UNGARC, P.C.
3291*
URSU, E.
3223*
USTINKOVA, E.M.
3158
VAHERI, A.
3231
VAITUKAITIS, J.L.
3516*
VAKIL, D.V.
3007
VALLADARES, Y.
3337
VAN BENTHEM, M.
3543*
VAN DER EB, A.J.
3147
VAN DER RIET, F.D.S.J.
3220*
VAN FURTH, R.
3504*
VAN NAGELL, J.R., JR.
3513*
VAN RIJN, J.
3571*
VAN SLYCK, E.J.
3430*
VAN WIJK, R.
3571*
VARELA, V.
3471*
VARSANYI, T.
3154
VENITT, S.
3071
VENNER, H.
3526*
VERRETT, M.J.
3067
VIADANA, E.
3389*
VIETTI, T.J.
3480*
VISFELDT, J.
3350
VISHNEVSKAYA, T.M.
3405*
VOGEL, C.L.
3516*
VOLFSON, N.J.
3297*
VON ESCH, A.M.
3065
VON TIGERSTROM, R.G.
3422*
WADA, Y.
3437*
WADE, M.E.
3436*
WADELL, G.
3154

WAGNER, H.
3269
WAGONER, J.K.
3334
WAINER, R.A.
3592*
WALDVOGEL, F.A.
3013*
WALKER, D.L.
3139
WALKER, D.M.
3452*
WALKER, J.E.
3346
WALKER, K.Z.
3315*
WALKER, R.H.
3441*
WALKER, R.I.
3003
WALTHER, F.
3531*
WANG, N.S.
3415*
WARD, A.M.
3457*
WARDLAW, A.C.
3391*
WARNAAR, S.O.
3198*
WARNATZ, H.
3262
WARNER, G.A.
3232
WARNER, N.L.
3253, 3287*
WARR, G.A.
3107*
WARREN, S.
3515*
WASTESON, A.
3435*
WATANABE, K.
3098*, 3594*
WEBB, J.H.
3208*
WEBER, F.
3471*
WEBER, J.
3148
WEDGWOOD, R.J.
3463*
WEERTS, D.
3581*
WEILAND, L.H.
3468*
WEISER, G.
3551*
WEISMAN, R.
3323*
WEISSBACH, A.
3138
WEISSMAN, I.L.
3236
WELFORD, H.J.
3505*
WELLS, S.A., JR.
3233
WENTZ, W.B.
3525*
WESSEL, H.
3032*
WEST, W.D.
3493*
WESTERHAUSEN, M.
3285*
WESTERMARK, B.
3435*
WETHERLY-MEIN, G.
3308*
WETTER, O.
3029*, 3304*
WHITE, A.J.
3377*
WHITMIRE, C.E.
3072
WHITTAKER, L.R.
3320*
WICKS, W.D.
3571*
WICKUS, G.G.
3137
WIERNIK, P.H.
3592*
WIEST, L.
3100*
WILDHACK, R.
3079*
WILKINSON, E.J.
3520*
WILLIAMS, A.E.
3506*
WILLIAMS, A.O.
3383*
WILLIAMS, D.J.
3390*
WILLIAMS, H.E.
3502*
WILLIAMS, M.E.
3458*
WILLIAMS, R.C.
3290*
WILSON, R.G.
3401*
WILSON, T.
3311*
WILTSHAW, E.
3286*
WINKLER, H.
3551*
WINNACKER, E.L.
3165
WITTER, R.
3135
WIWATOWSKI, T.
3314*
WOLF, H.
3049, 3160
WOLFE, L.G.
3191*
WONG, G.
3533*
WONG, K.
3486*
WOOD, D.C.
3471*
WOODARD, J.C.
3549*
WOODS, W.A.
3208*
WOOLNER, L.B.
3596*
WORWOOD, M.
3400*
WRIGHT, N.A.
3491*
WRIGHT, S.E.
3203*
WUNDERLICH, V.
3026*
YAJIMA, Y.
3590*
YAJKO, R.D.
3566*
YAMAGUCHI, M.
3183*
YAMAMURA, M.
3296*
YAMAZAKI, M.
3307*
YANIV, A.
3169
YERSHOV, F.I.
3131
YIN, L.
3067
YOSHIMOTO, S.
3121
YOUN, J.K.
3141
YOUNG, R.C.
3579*
YOUNGLAI, E.V.
3421*
ZAJDELA, F.
3212*
ZALDIVAR, R.
3331
ZANG, K.D.
3586*
ZANKL, H.
3586*
ZATZKIN, J.B.
3258
ZDEBSKA, E.
3380*
ZECH, L.
3369*
ZEILLER, K.
3531*
ZELJVIN, B.M.
3363*
ZELLER, J.
3086*
ZHARDETSKIY, A.S.
3407*
ZHDANOV, V.M.
3131
ZIEBARTH, D.
3108*
ZINNINGER, G.F.
3332
ZUFFARDI, O.
3425*

ZUR HAUSEN, H.
3160
ZURHEIN, G.M.
3139
ZWEIMAN, B.
3250

* INDICATES A PLAIN CITATION WITHOUT ACCOMPANYING ABSTRACT

- ACETYLCHOLINE
  - PROTEIN SYNTHESIS
    - LIMB REGENERATION, AMPHIBIAN, 3398*
- ACTINIC KERATOSIS
  - GENETIC DIATHESIS
    - CLINICAL STUDY, 3509*
- ADENOCARCINOMA
  - ENDOMETRIUM
    - CELL CULTURE, HUMAN, 3510*
  - MAMMARY GLAND
    - CELL POPULATION KINETICS, RAT, 3328
  - PAPILLARY CLEAR CELL
    - CASE REPORT, 3513*
  - VAGINA, CLEAR CELL
    - DIETHYLSTILBESTROL, CASE REPORTS, 3488*
- ADENOLYMPHOMA
  - SALIVARY GLAND
    - CASE REPORT, 3539*
- ADENYLATE CYCLASE
  - HEPATOMA, MALIGNANT
    - GLUCAGON, PROSTAGLANDIN E1 STIMULATION, ETHIONINE-TREATED RAT, 3055
- AFLATOXIN B1
  - ENDOPLASMIC RETICULUM
    - LIVER, RAT, 3390*
  - TOXIC FLUORESCENT METABOLITE
    - NON-MICROSOMAL RAT LIVER PREPARATION, 3067
- AGE
  - CANCER
    - CIRRHOSIS OF LIVER, ATHEROSCLEROSIS, CORRELATIONS, REVIEW, 3034*
- AGGLUTINATION
  - CONCANAVALIN A
    - CELL CYCLE DEPENDENCE, TRANSFORMED HUMAN LYMPHOCYTES, 3162
  - CONCANOVALIN A
    - TRANSFORMED CELL, MOUSE, 3242
- AGING
  - ELEVATION MECHANISM
    - RISK FACTOR FOR TUMOR DEVELOPMENT, REVIEW, 3028*
- ALBUMIN
  - SYNTHESIS
    - EFFECT OF FRIEND VIRUS, MOUSE LIVER, 3212*
- ALCOHOLISM
  - HEAD AND NECK CANCER
    - CLINICAL STUDY, 3411*
- ALKYL CAREAMATES
  - BINARY MIXTURE WITH COMPONENTS
    - ONCOGENIC ACTIVITY, COMPARATIVE EVALUATION, 3093*
- AMINOPYRENE
  - WITH NITRITE
    - LIVER, LUNG MALIGNANT TUMORS, RAT, 3090*
- ANDROBLASTOMA
  - OVARY
    - HETEROTOPIC ELEMENTS, CASE HISTORY, 3481*
- ANDROGEN
  - ARRHENOBLASTOMA
    - CLINICAL STUDY, 3421*
  - MAMMARY CARCINOMA
    - ATYPICAL ENDOMETRIAL PATTERN, CLINICAL STUDY, 3420*
- ANGIOFIBROMA
  - JUVENILE
    - CASE REPORT, 3477*
- ANTI-P-AZOPHENYLARSONATE
  - ANTIBODY
    - DISTRIBUTION OF A CROSS-REACTIVE IDIOTYPIC SPECIFICITY, MICE, 3241
- ANTIBODY
  - ANTI-IGG
    - ISOLATION AND IMMUNOCHARACTERIZATION, RABBIT, 3264
  - ANTI-IMMUNOGLOBULIN
    - T AND B LYMPHOCYTES, ANTIGEN RECEPTOR BLOCKING, REDISTRIBUTION, 3271
  - ANTI-P-AZOPHENYLARSONATE
    - DISTRIBUTION OF A CROSS-REACTIVE IDIOTYPIC SPECIFICITY, MICE, 3241
  - ANTI-TYROSINASE ACTIVITY
    - IMMUNE RESPONSE, RABBIT, 3302*
  - ANTIHAPTEN
    - LYMPHOCYTE INITIATING MECHANISM, MOUSE, 3227
  - COMPLEMENT-FIXING
    - NASOPHARYNGEAL CARCINOMA PATIENTS, 3230
  - CROSS-REACTING
    - SV40 TUMOR IMMUNIZATION, ISOTOPIC ANTIGLOBULIN TECHNIQUE, MOUSE, 3233
  - 125I-LABELED ANTIGLIOMA
    - SEQUENTIAL ADSORPTION ANALYSIS, CULTURED GLIOMA CELLS, 3252
  - LYMPHOCYTE KILLING OF ALLOGENEIC MYELOBLASTS
    - CYTOTOXICITY, HUMAN LEUKEMIC CELLS, 3289*
  - PRODUCING CELLS
    - CHANGES DURING CARCINOGENESIS, SPLEEN, MOUSE, 3272
  - 7S
    - IMMUNE RESPONSE INHIBITION, MECHANISM, MOUSE, 3255
  - VIRUS, SV40
    - T ANTIGEN, HUMAN SERA, 3238
- ANTIGEN

ABO SYSTEM TRANSPLANTATION
PARALYMPHOBLASTS, LEUKEMIA, HUMAN, 3283*
CARCINOEMBRYONIC
COLON CARCINOMA, RADIOIMMUNOASSAY, HUMAN, 3311*
HEPATOMA, CLINICAL STUDY, 3572*
HETEROGENEITY, 3275
MALIGNANT, NONMALIGNANT DISEASE, HUMAN, 3287*
RADIOIMMUNOLOGICAL DETERMINATION, CLINICAL STUDY, 3298*
CELL-SURFACE
MAREK'S DISEASE VIRUS, LYMPHOID CELLS, CHICKEN, 3257
RADIATION LEUKEMIA VIRUSES, RAT, 3234
RD-114 VIRUS, RHABDOMYOSARCOMA, HUMAN, 3267
CELL SURFACE-BOUND
IMMUNOGENICITY, MOUSE, 3227
CELL-SURFACED
IMMUNOLOGICAL TOLERANCE BREAKDOWN, RAT, 3235
FETAL
SV40-TRANSFORMED CELLS, MOUSE, 3244, 3273
GROUP-SPECIFIC
AVIAN TYPE-C VIRUSES, CHICKEN CELLS AND TISSUES, 3231
HISTOCOMPATIBILITY
DISTRIBUTION, T AND B CELLS, GUINEA PIG, 3306*
RECEPTOR BLOCKING, REDISTRIBUTION
T AND B LYMPHOCYTES, ANTI-IG ANTIBODY, 3271
ROUS SARCOMA VIRUS-TRANSFORMED CELL
GROWTH, HAMSTER CELLS, 3156
SPECIFIC SURFACE
DETECTION, ADENOVIRUS 12-TRANSFORMED CELLS, MOUSE, HAMSTER, 3276
TRANSPLANTATION
MURINE TUMOR, 3239
TUMOR-SPECIFIC
SARCOMA, MOUSE, 3239
SV40-TRANSFORMED CELLS, MOUSE, 3244, 3273
VIRION-ENVELOPE
RADIATION LEUKEMIA VIRUSES, RAT, 3234
VIRUS, ADENO 12
IMMUNE RESPONSE, MACROPHAGE INHIBITION TEST, MOUSE, 3263
VIRUS, C-TYPE
PASSIVE HEMAGGLUTINATION TEST, 3268
VIRUS, EPSTEIN-BARR
NASOPHARYNGEAL CARCINOMA, HUMAN, 3248
VIRUS, MAREK'S DISEASE
LYMPHOID CELLS, CHICKEN, 3257

ANTIGEN, SURFACE
VIRUS, ROUS SARCOMA
HUMORAL CYTOTOXICITY TEST, HAMSTER, 3281*

ANTIGEN, T
SV40 ANTIBODY
HUMAN SERA, 3238

ANTISERUM
ANTI-IGG
ISOLATION AND IMMUNOCHARACTERIZATION, RABBIT, 3264

ARRHENOBLASTOMA
ANDROGEN
CLINICAL STUDY, 3421*

ARYL HYDROCARBON HYDROXYLASE
INDUCTION
BENZ(A)ANTHRACENE, FIBROBLASTS, MOUSE, 3063
INDUCTION BY CIGARETTE SMOKE
PULMONARY ALVEOLAR MACROPHAGES, HUMAN, 3107*

ASBESTOS
CARCINOGEN, ATMOSPHERIC
PLEURAL PLAQUES, HUMAN, 3045
LUNG
PLEURAL PLAQUES, HUMAN, 3045

ASCITES TUMOR
SEE ALSO EHRLICH
GROWTH
SELF LIMITATION, MOUSE, 3399*

ASCORBIC ACID
PREVENTION OF HYDROCEPHALI
NITROSAMIDE-INDUCED, RAT, 3086*

AUTOIMMUNE DISORDERS
LEUKEMIA
CYTOTOXICITY, IMMUNOFLUORESCENCE, CLINICAL STUDY, 3280*

BASAL CELL CARCINOMA
EPITHELIAL OUTGROWTHS
LOW-RESISTANCE JUNCTIONS, 3459*
RADIATION, UV
SUPERSONIC TRANSPORT, REVIEW, 3002

BASAL CELL EPITHELIOMA
GENETIC DIATHESIS
CLINICAL STUDY, 3509*

BENZ(A)ANTHRACENE
ARYL HYDROCARBON HYDROXYLASE INDUCTION
FIBROBLAST CELL CULTURE, MOUSE, 3063
OXIDATIVE ACTIVATION
MUTAGENESIS, CARCINOGENESIS, DROSOPHILA, 3074

BENZIDINE
INTESTINAL TUMOR INDUCTION
RAT, 3297*

BENZO(A)PYRENE
CARCINOGENESIS
UV SPECTRA, 3089*
CARCINOGENIC ACTIVITY
PHENOL, 5-METHYLRESORCINOL, MICE, 3106*

- INTRATRACHEAL INJECTION
  - PATHOLOGICAL REACTION. RAT. 3104*
- MAXIMALLY PERMISSIBLE DOSES. CONCENTRATIONS
  - REVIEW. 3027*
- METABOLISM
  - LIVER MICROSOMES. 3-METHYLCHOLANTHRENE-TREATED RAT. 3069
- SKIN CANCER
  - NUCLEAR DNA SYNTHESIS. CELL PROLIFERATION. MOUSE. 3087*
- TRACHEOBRONCHIAL EPITHELIUM
  - SURFACE MORPHOLOGY. MOUSE. 3068
- UV-INDUCED COOXIDATION
  - PHENOLS. KINETICS. 3058

BENZO(E)PYRENE
- CARCINOGENESIS
  - UV SPECTRA. 3089*

BLASTOGENIC RESPONSE
- LYMPHOCYTE
  - AUTOCHTHONOUS TUMOR CELLS. HUMAN. 3243

BLEOMYCIN
- EFFECT ON CERVICAL CARCINOMA
  - MOUSE. 3486*

BLOOD
- CELL PURIFICATION
  - FLOTATION ON FICOLL TRIOSIL MIXTURE. 3543*
- ERYTHROPOIETIC PROGENITORS
  - COLONY FORMATION IN CULTURE. MOUSE. 3548*
- FECAL LOSS
  - RADIOACTIVELY LABELED. NORMAL HUMAN. 3462*
- MALATE. OXALOACETATE LEVEL
  - BENIGN SURGICAL MALIGNANT DISEASE. HUMAN. 3490*

BONE
- MALIGNANT TUMORS
  - CHILDREN. 3438*
  - SERUM ALKALINE PHOSPHATASE. HUMAN. 3359
- MARROW ALTERATIONS
  - LYMPHORETICULAR SARCOMA. CLINICAL STUDY. 3446*

BONE MARROW
- ALLOGENEIC MARROW GRAFTING DURING MARROW REMISSION
  - ACUTE LYMPHOID LEUKEMIA PATIENTS. 3292*
- COLONY FORMING UNITS
  - EFFECT OF 226 RADIUM. SPLENECTOMIZED MICE. 3114*
- LEUKEMIA
  - OSTEOGENIC POTENTIAL. RAT. 3555*
- LEUKEMIA. MYELOMA CELLS
  - ELECTROPHORETIC CELL SEPARATION. HUMAN. 3531*
- MONOCYTE PROLIFERATION
  - GLUCOCORTICOSTEROIDS. HUMAN. 3504*
- MYELOPROLIFERATIVE DISEASE
  - MORPHOLOGICAL ANOMALIES. HUMAN. 3521*
- RADIATION. X-RAY
  - CYTOGENETIC EFFECT. MOUSE . 3112

BRAIN
- CEREBRAL VASCULAR NEOPLASMS
  - IMPAIRED REGIONAL CIRCULATION. HUMAN. 3546*
- MYXOPAPILLARY EPENDYMOMA OF THE FILUM TERMINALE
  - ULTRASTRUCTURE. 3589*
- TUMOR-LIKE LESION
  - TRYPANOSOMA CRUZI. CASE REPORT. 3562*

BRAIN TUMOR
- HISTOGENESIS
  - MORPHOLOGICAL AND IMMUNOLOGICAL STUDIES. RAT. 3318
- HISTOPATHOLOGY
  - AFRICANS. KENYA. 3368*
- HUMAN PAPOVAVIRUS(JC)-INDUCED
  - HAMSTER. 3139
- IMMUNOLOGY
  - IMMUNOTHERAPY. REVIEW. 3023*
- MENINGITIS
  - CASE REPORT. 3463*
- PROTEIN
  - IMMUNOLOGICAL STUDY. HUMAN. 3274

BREAST
- SEE ALSO MAMMARY
- PRIMARY LYMPHOMA
  - CASE REPORTS. 3578*

BREAST CANCER
- ETIOLOGY
  - EPIDEMIOLOGICAL ASPECTS. REVIEW. 3007
- MALE
  - GYNECOMASTASIA. CLINICAL STUDY. 3350
  - URINARY ESTROGEN EXCRETION. CLINICAL STUDY. 3575*

BRENNER TUMOR
- MALIGNANT
  - CASE REPORT. 3373*

5-BROMO-2-DEOXYURIDINE
- FIBROBLAST
  - MEMBRANE FUNCTION. MOUSE. 3429*

5-BROMODEOXYURIDINE
- C-TYPE VIRUS SUPERINDUCTION
  - TRANSFORMED MOUSE CELLS. 3145
- INCORPORATION
  - SV40 DNA. MONKEY CELLS. 3196*

5'-BROMODEOXYURIDINE
- MUTAGENESIS
  - MURINE SARCOMA VIRUS-TRANSFORMED NONPRODUCER CELL LINES. 3157

7-BROMOMETHYLBENZ(A)ANTHRACENE
- DNA
  - ESCHERICHIA COLI. 3071
- MUTAGENESIS

    - ESCHERICHIA COLI, 3071
  - RNA
    - ESCHERICHIA COLI, 3071

BURKITT'S LYMPHOMA
- CELL LINE
  - SENSITIVITY TO EPSTEIN-BARR VIRUS SUPERINFECTION, 3166
- PATHOGENESIS
  - REVIEW, 3320*
- VIRUS, EPSTEIN-BARR
  - ETIOLOGY, AMERICAN AND AFRICAN, 3168
  - MODES OF INFECTION, HUMAN, 3130

CALCIUM
- UPTAKE
  - EXPERIMENTAL TUMORS, MOUSE, 3340

CANCER
- AGE
  - CIRRHOSIS OF LIVER, ATHEROSCLEROSIS, CORRELATIONS, REVIEW, 3034*
- DATA ACQUISITION
  - ONTARIO CANCER INCIDENCE SURVEY, 3329
- DETECTION
  - SINUS HISTIOCYTOSIS, CLINICAL STUDY, 3495*

CARCINOGEN, ATMOSPHERIC
- ASBESTOS
  - PLEURAL PLAQUES, HUMAN, 3045

CARCINOGEN, CHEMICAL
- ALKYL CARBAMATES AND COMPONENTS
  - COMPARATIVE EVALUATION, 3093*
- CENTRAL NERVOUS SYSTEM TUMORS
  - REVIEW, 3032*
- DETECTION
  - INTRA-ANIMAL CULTURE OF MICROORGANISMS, 3092*
  - USE OF CHLORELLA, 3108*
- EFFECT OF RADIATION LEUKEMIA VIRUS
  - MOUSE, 3072
- ENZYME INDUCTION
  - REVIEW, 3024*
- GENERAL ASPECTS
  - REVIEW, 3026*
- GLUTATHIONASE
  - LIVER, RAT, 3040
- INDUSTRIAL HEALTH STANDARDS
  - U.S. DEPARTMENT OF LABOR, 3460*
- MAXIMALLY PERMISSIBLE DOSES, CONCENTRATIONS
  - REVIEW, 3027*
- TRANSFORMATION, MALIGNANT
  - QUANTITATIVE SYSTEM FOR ASSAY, MOUSE, 3065

CARCINOLIPIN
- TRANSPLACENTAL EFFECT
  - MOUSE, 3043

CARCINOSARCOMA
- UTERINE CERVIX
  - CLINICAL STUDY, 3408*

CELL
- COLONY FORMATION
  - ERYTHROPOIETIC PROGENITORS, MOUSE, 3548*
- VIABILITY
  - EFFECT OF HYDROCORTISONE, HUMAN LYMPHOBLASTOID CELL LINES, 3119

CELL CYCLE
- AGGLUTINATION WITH CON A
  - TRANSFORMED HUMAN LYMPHOCYTES, 3162

CELL FUSION
- VIRUS, HERPES SIMPLEX
  - EFFECT OF 2-DEOXY-D-GLUCOSE, 3177*
- VIRUS, NEWCASTLE DISEASE
  - EFFECT OF 2-DEOXY-D-GLUCOSE, 3177*

CELL PROLIFERATION
- EFFECT OF TRANSPLANTABLE HEPATOMA
  - MOUSE, 3432*
- ETHYLPHENYLPROPIOLATE
  - EPIDERMAL HYPERPLASIA, MOUSE, 3076
- MAMMARY GLAND
  - HORMONAL EFFECTS, IN VITRO STUDIES, 3321*
- TESTOSTERONE PROPIONATE
  - SEMINAL VESICLE, CASTRATE MOUSE, 3491*

CERVICAL CARCINOMA
- CLEAR CELL
  - MATERNAL STILBESTROL THERAPY, VAGINAL ADENOSIS, CASE REPORTS, 3524*
- EPITHELIUM
  - ULTRASTRUCTURE, HUMAN, 3506*
- ESTROGEN
  - VAGINAL SMEARS, CLINICAL STUDY, 3537*
- METASTASIS
  - CALCIFIED LYMPH NODE, CASE REPORT, 3512*
- 20-METHYLCHOLANTHRENE
  - EFFECT OF ENDOXAN, BLEOMYCIN, MOUSE, 3486*

CERVIX
- PEMPHIGUS VULGARIS
  - ENDOMETRIAL CARCINOMA OF UTERUS, CASE REPORT, 3316*

CHEMODECTOMA
- CAROTID ARTERY BIFURCATION TUMORS
  - ANGIOGRAPHIC DIFFERENTIATION, HUMAN, 3500*
- CATECHOLAMINE STORAGE IN LIVER METASTASES
  - MORPHOLOGY, BIOCHEMISTRY, HUMAN, 3551*
- DIAGNOSIS, TREATMENT
  - REVIEW, HUMAN, 3485*

CHEMOTHERAPY
- MOLONEY SARCOMA VIRUS-INDUCED TUMORS
  - MOUSE, 3173*

CHILDREN

CANCER
IN UTERO DEATHS. RADIOGENIC LEUKEMIA. 3115*
CHILDHOOD DISEASES
EPSTEIN-BARR VIRUS. CASE REPORTS. 3183*
INFANTILE CANCER
MORTALITY. FECUNDITY. UNITED STATES. 3336*
LEUKEMIA. ACUTE LYMPHOBLASTIC
IMMUNE RESPONSE IN FAMILIES. 3237
LEUKEMIA INCIDENCE
CHILE. 3331
MALIGNANT LYMPHOMA INCIDENCE
CHILE. 3331
NERVOUS SYSTEM TUMOR INCIDENCE
CHILE. 3331

CHLOROPRENE
EMBRYOGENESIS
RAT. 3094*

CHOLESTERYL-14-METHYLHEXADECANOATE
TRANSPLACENTAL EFFECT
MOUSE. 3043

CHONDROSARCOMA
INTRACRANIAL. MESENCHYMAL
CASE REPORT. 3366*
SERUM ALKALINE PHOSPHATASE
HUMAN. 3359

CHORDOMA
SACROCOCCYGEAL
ULTRASTRUCTURE. CASE REPORT. 3588*

CHROMOSOME
ANALYSIS
FLUORESCENCE TECHNIQUE. MENINGIOMA. HUMAN. 3561*
INSTABILITY
CANCER OCCURRENCE. HUMAN. 3545*
Y BODIES
FLUORESCENT CHROMOCENTERS. HUMAN TUMORS. 3518*

CHROMOSOME ABERRATIONS
DNA CROSS-LINKING AGENTS
FANCONI'S ANEMIA PATIENTS. 3059
MELANOMA. MALIGNANT
CLINICAL STUDY. 3484*
MENINGIOMA
ACROCENTRICITY. NUCLEOLAR AREA. HUMAN. 3586*
MITOMYCIN C
FANCONI'S ANEMIA PATIENTS. 3059
NITROGEN MUSTARD
FANCONI'S ANEMIA PATIENTS. 3059
OVARIAN CARCINOMA
HUMAN. 3425*
PRE-LEUKEMIC HEMATOLOGICAL DISORDERS
CLINICAL STUDY. 3580*
RADIATION. UV
FANCONI'S ANEMIA PATIENTS. 3059
SUSCEPTIBILITY TO MALIGNANT DISEASE
POROKERATOSIS OF MIBELLI PATIENTS. 3344

CIGARETTE SMOKE
ARYL HYDROCARBON HYDROXYLASE INDUCTION
PULMONARY ALVEOLAR MACROPHAGES. HUMAN. 3107*

CLEAR CELL CARCINOMA
CERVIX. VAGINA
MATERNAL STILBESTROL THERAPY. VAGINAL ADENOSIS. CASE REPORTS. 3524*
ENDOMETRIUM
ULTRASTRUCTURE. PATHOLOGY. CLINICAL STUDY. 3597*

COLON CARCINOMA
CASE PRESENTATION. 3566*
EPIDEMIOLOGY
HUMAN. REVIEW. 3006
IMMUNOLOGY
RADIOIMMUNOASSAY FOR CARCINOEMBRYONIC ANTIGEN. HUMAN. 3311*

COMPLEMENT
HEMOLYTIC LEVELS
MEASUREMENT AND MANIPULATION. TUMOR BEARING MICE. 3291*

CONCANAVALIN A
AGGLUTINATION
CELL CYCLE DEPENDENCE. TRANSFORMED HUMAN LYMPHOCYTES. 3162
BINDING
TRANSFORMED. NORMAL CELLS. 3536*
EMBRYOGENESIS
CHICKEN. 3288*
IN VITRO EFFECTS
HUMAN LYMPHOCYTES. 3314*
INDUCED LYMPHOCYTE TRANSFORMATION
S-ADENOSYL-L-METHIONINE DECARBOXYLASE. 3557*
MITOCHONDRIA
COMPARATIVE STUDY. NORMAL AND MALIGNANT CELLS. 3258

CONCANOVALIN A
AGGLUTINATION
TRANSFORMED CELL. MOUSE. 3242

COPPER
SERUM
HODGKIN'S DISEASE. CLINICAL STUDY. 3556*

CORTICOSTEROID
LONG-TERM ADMINISTRATION
ASSOCIATED DWARFISM. GLAUCOMA. CHILD. 3558*

CRANIOPHARYNGIOMA
ULTRASTRUCTURE
IN VIVO. IN VITRO. 3445*

CRANIUM BIFIDUM OCCULTA
JUVENILE FIBROMATOSIS OF FACE
CASE REPORT. 3456*

CYCLIC AMP
GROWTH HORMONE-SECRETING PITUITARY TUMORS

- RAT, 3395*
- HEPATOMA, MALIGNANT
  - GLUCAGON, PROSTAGLANDIN E1
    - STIMULATION, ETHIONINE-TREATED RAT, 3055
- NEUROBLASTOMA
  - MORPHOLOGIC DIFFERENTIATION, MOUSE, 3426*
- STIMULATION BY PROSTAGLANDIN E1
  - PHOSPHODIESTERASE INHIBITORS, NEUROBLASTOMA, MOUSE, 3470*

CYTOCHEMISTRY
- LEUKEMIA CHRONIC LYMPHOID
  - HUMAN, 3285*

CYTOSINE ARABINOSIDE
- LEUKEMIA, ACUTE MYELOID
  - MYELOBLAST CELL CYCLE, CLINICAL STUDY, 3423*

CYTOTOXICITY
- HUMAN LEUKEMIC CELLS
  - ANTIBODY-DEPENDENT LYMPHOCYTE KILLING OF ALLOGENEIC MYELOBLASTS, 3289*
- IRRADIATED FOOD PRODUCTS
  - EVALUATION, 3095*

DDT
- CARCINOGENICITY
  - MOUSE, REVIEW, 3011*

2-DEOXY-D-GLUCOSE
- EFFECT ON CELL FUSION
  - NEWCASTLE DISEASE VIRUS AND HERPES SIMPLEX VIRUS-INDUCED, 3177*
- INCORPORATION INTO GLYCOPROTEINS
  - SV40-TRANSFORMED HAMSTER CELLS, 3206*

M-DIAMINOANISOL
- CARCINOGENESIS
  - RAT, 3036

DIBUTYRYL CYCLIC AMP
- HEPATOMA
  - DNA SYNTHESIS, 3571*

DIEMTHYLNITROSAMINE
- 3-METHYLCHOLANTHRENE
  - SYNCARCINOGENIC EFFECT, MOUSE, 3078

DIETHYLNITROSAMINE
- CARCINOGENESIS
  - DETECTION BY CHLORELLA, 3108*
- CELL CLASSES
  - MITOSIS IN LIVER, RAT, HAMSTER, 3100*
- DUST
  - RESPIRATORY CARCINOGENESIS, HAMSTER, 3077
- INDUCED LIVER CARCINOMAS
  - DEOXYRIBONUCLEASES, PHOSPHATASES, RAT, 3085*

DIETHYLSTILBESTROL
- ADENOCARCINOMA, CLEAR CELL
  - CASE REPORTS, YOUNG FEMALES, 3488*
- CLEAR CELL CARCINOMA, VAGINA, CERVIX
  - CASE REPORTS, 3524*

DIGESTIVE SYSTEM
- CANCER DIAGNOSIS
  - RADIOIMMUNOASSAY FOR CARCINOEMBRYONIC ANTIGEN, HUMAN, 3311*

9,10,-DIMETHYL-1-2-BANZANTHRACENE
- SALIVARY GLAND TUMOR
  - EFFECT OF ZINC, RAT, 3054

9,10-DIMETHYL-1,2-BENZANTHRACENE
- TRANSFORMATION, MALIGNANT
  - RELATION TO CYTOTOXICITY, CELLS, HAMSTER, 3070

4-DIMETHYLAMINOAZOBENZENE
- CARCINOGENESIS
  - DETECTION BY CHLORELLA, 3108*

7,12-DIMETHYLBENZ(A)ANTHRACENE
- CARCINOGENESIS
  - REGIONAL LYMPHADENECTOMIZED RATS, 3270
- GLUTATHIONE CONJUGATES FORMATION
  - LIVER HOMOGENATES, RAT, 3105*
- HYPERTENSION
  - HIGH, LOW SALT INTAKE, RAT, 3091*
- INDUCED SARCOMA
  - ISOENZYME PATTERN, RAT, 3424*
- MAMMARY CARCINOMA
  - ESTROGEN EFFECT, RAT, 3039
  - PROGESTERONE EFFECT, RAT, 3039
- MAMMARY TUMOR
  - INDUCTION, HAMSTER, 3081*
- NEPHROBLASTOMA
  - IMMUNODEPRESSED RAT, 3080*

7-12-DIMETHYLBENZ(A)ANTHRACENE
- SKIN TUMOR
  - MOUSE, 3056
  - UV ENHANCEMENT, INHIBITION, MOUSE, 3050

7,12-DIMETHYLBENZ(A)ANTHRACENE
- TUMOR INDUCTION
  - ANTILYMPHOCYTE SERUM ADMINISTRATION, MOUSE, 3088*

DISULFIDE
- REDUCTION
  - TUMOR PROLIFERATION, IMMUNNOLOGY, MOUSE, 3346

6,6'-DITHIODINICOTINIC ACID
- EHRLICH ASCITES TUMOR
  - GLYCOLYTIC MECHANISMS, MOUSE, 3458*

2,2'-DITHIODIPYRIDINE
- EHRLICH ASCITES TUMOR
  - GLYCOLYTIC MECHANISMS, MOUSE, 3458*

DNA
- 7-BROMOMETHYLBENZ(A)ANTHRACENE

- ESCHERICHIA COLI, 3071
- CROSS-LINKING AGENTS
  - CHROMOSOME ABERRATIONS, 3059
- ETHYLCARBAMATE
  - PROLONGED BINDING, LIVER, MOUSE, 3099*
- FRAGMENTATION
  - SV40 INFECTION, PSEUDOVIRION FORMATION, MONKEY CELLS, 3152
- HUMAN ADENOVIRUS 2
  - UV RADIATION, 3210*
- N-METHYL-N-NITROSOUREA
  - PHOSPHOTRIESTER FORMATION, 3048
- METHYLATION
  - N-METHYL-N-NITROSOUREA, 3060
- 7-METHYLBENZ(A)ANTHRACENE
  - HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS, CELL CULTURE, MOUSE, 3044
- 7-METHYLBENZ(A)ANTHRACENE BINDING
  - HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS, CELL CULTURE, MOUSE, 3042
- 3-METHYLCHOLANTHRENE
  - BINDING SITE, FIBROBLAST, HAMSTER, 3073
- PHOTOLESIONS
  - REPAIR, 3361
- SINGLE STRANDED
  - NUCLEOLUS, ZAJDELA ASCITES TUMOR, RAT, 3508*
- VIRUS, ADENO
  - GENOME, ELECTRON MICROSCOPY, 3143
  - HYDROXYAPATITE CHROMATOGRAPHY, FORMAMIDE DENATURATION, 3199*
- VIRUS, ADENO 5
  - TRANSFORMATION OF RAT CELLS, 3147
- VIRUS, HERPES SIMPLEX
  - DENATURATION PROPERTIES, 3186*
- VIRUS, SV40
  - ALTERATION, HOST CELL INCORPORATION, 3122
  - GENOME, ELECTRON MICROSCOPY, 3143
  - REASSORTMENT DURING SERIAL UNDILUTED PASSAGE, 3184*

DNA POLYMERASE
- HEPATOMA, MORRIS
  - SEPARABLE ACTIVITIES, HOST LIVER, RAT, 3582*
- INACTIVATION
  - POXVIRUSES, 3189*
- MULTIPLE
  - MYELOMA, ISOLATION, CHARACTERIZATION, MOUSE, 3374*
- PURIFICATION
  - HERPES SIMPLEX VIRUS-INFECTED HELA CELLS, 3138
- REVERSE TRANSCRIPTASE
  - RNA PROGRAMMED, REVIEW, 3015*
- RNA DEPENDENT
  - IMMUNE RESPONSE, RABBIT, 3259

DNA REPLICATION
- VIRUS, POLYOMA
  - DISCONTINUOUS ELONGATION OF STRANDS AT REPLICATION FORKS, 3165

DNA SYNTHESIS
- ACUTE MYELOID LEUKEMIA
  - CYTOSINE ARABINOSIDE, CLINICAL STUDY, 3423*
- BENZO(A)PYRENE-INDUCED SKIN CANCER
  - CYTOPHOTOMETRY, MOUSE, 3087*
- DISCONTINUOUS
  - EHRLICH ASCITES TUMOR, MAMMAL, 3347
- ESTROGEN, PROGESTERONE, TESTOSTERONE
  - MAMMARY TUMOR, HUMAN, 3356
- HEPATOMA
  - DIBUTYRYL CYCLIC AMP, 3571*
- INDUCTION
  - ADENOVIRUS 2-SV40 HYBRID, 3185*
- POLYOMA GENE FUNCTION
  - MOUSE CELLS, 3129
- TRIETHYLENEIMINOBENZOQUINONE
  - EHRLICH ASCITES TUMOR CELLS, 3049
- VIRUS, SV40
  - 5-BROMODEOXYURIDINE INCORPORATION, MONKEY CELLS, 3196*

DNA TRANSCRIPTION
- VIRUS, ADENO 2
  - PARTICIPATION OF EARLY PROTEINS, 3182*
- VIRUS, SV40
  - POLYADENYLIC ACID, 3126

DUST
- DIETHYLNITROSAMINE
  - RESPIRATORY CARCINOGENESIS, HAMSTER, 3077

EHRLICH ASCITES CARCINOMA
- LETTRE
  - FREEZING, THAWING, MORPHOLOGICAL STRUCTURE, 3409*

EHRLICH ASCITES TUMOR
- ACTIN-LIKE FILAMENTS
  - REACTION WITH HEAVY MEROMYOSIN, 3585*
- COMPLEMENT FACTORS
  - GROWTH, MOUSE, 3278*
- DISCONTINUOUS DNA SYNTHESIS
  - IN VIVO, MAMMALS, 3347
- 6,6'-DITHIODINICOTINIC ACID
  - GLYCOLYTIC MECHANISMS, MOUSE, 3458*
- 2,2'-DITHIODIPYRIDINE
  - GLYCOLYTIC MECHANISMS, MOUSE, 3458*
- DNA-DEPENDENT RAN POLYMERASE
  - MOUSE, 3552*
- GLUCOSE ANOMERS
  - ACTIVE TRANSPORT, MOUSE, 3507*
- HYPERDIPLOID
  - A-OXOGLUTARATE TRANSPORT, MITOCHONDRIA, 3573*
- LETTRE MUTANT
  - A-OXOGLUTARATE TRANSPORT, MITOCHONDRIA, 3573*
- PHOSPHORYLATION OF 3'-DEOXY-3'FLUOROTHYMIDINE
  - INCORPORATION INTO DNA, CELLFREE SYSTEM, 3526*
- RNA DEGRADATION

- CELL CULTURE, 3422*
- RNA SYNTHESIS
  - INHIBITION, HEAT TREATMENT, MOUSE, RAT, 3402*

ENDOMETRIAL CARCINOMA
- UTERUS
  - PEMPHIGUS VULGARIS OF CERVIX, CASE REPORT, 3316*

ENDOMETRIUM
- ADENOCARCINOMA
  - CELL CULTURE, HUMAN, 3510*
- ATYPICAL PATTERN
  - MAMMARY CARCINOMA, CLINICAL STUDY, 3420*
- CARCINOMA
  - CYTOLOGIC DETECTION, HUMAN, 3437*
- CLEAR CELL CARCINOMA
  - ULTRASTRUCTURE, PATHOLOGY, CLINICAL STUDY, 3597*
- ESTRADIOL RECEPTORS
  - CYTOPLASM, HUMAN, 3327*
- MIXED ADENOSQUAMOUS CARCINOMA
  - CLINICAL STUDY, 3525*
- SQUAMOUS CELL CARCINOMA
  - CASE REPORTS, 3377*
- XENOTRANSPLANTATION
  - HUMAN CELLS INTO HAMSTERS, 3312*

ENDOPLASMIC RETICULUM
- AFLATOXIN B1
  - LIVER, RAT, 3390*

ENDOXAN
- EFFECT ON CERVICAL CARCINOMA
  - MOUSE, 3486*

ENZYME
- SEE ALSO NAMES OF
- ACTIVITY
  - CARCINOGENESIS, 3472*
- GLUTATHIONASE
  - CHEMICAL CARCINOGENS, LIVER, RAT, 3040
- HISTOCHEMISTRY
  - LEAD-INDUCED CHRONIC KIDNEY DISEASE, RAT, 3096*
- INDUCTION
  - CHEMICAL CARCINOGENS, REVIEW, 3024*

EPENDYMOMA
- MYXOPAPILLARY, OF THE FILUM TERMINALE
  - ULTRASTRUCTURE, 3589*

EPIDEMIOLOGY
- CANCER INCIDENCE
  - ONTARIO, 3329
- CASE-CONTROL STUDIES
  - GUIDELINES, 3376*
- COLON CARCINOMA
  - HUMAN, REVIEW, 3006
- HARD PALATE CARCINOMA
  - REVERSE SMOKERS, 3335
- LEUKEMIA
  - NEBRASKA, REVIEW, 3030*
- LYMPHOMA
  - NEBRASKA, REVIEW, 3030*
- MAREK'S DISEASE
  - INCIDENCE OF PRECIPITINS, ZOO BIRDS, 3217*

EPIDERMIS
- KERATOACANTHOMA
  - TATTOO, CASE REPORT, 3567*
- PAPILLOMA, SPONTANEOUS
  - NEWT, 3599*
- TUMORS, HEAD, NECK, ESOPHAGUS
  - HYPERCALCEMIA, CLINICAL STUDY, 3503*

EPIDIDYMIS
- CARCINOMA OF MULLERIAN VESTIGAL ORIGIN
  - CASE REPORT, 3479*

EPITHELIUM
- CERVICAL CARCINOMA
  - ULTRASTRUCTURE, HUMAN, 3506*
- GROWTH FACTOR
  - CHARACTERISTICS, RABBIT, 3364*
- LOW-RESISTANT JUNCTIONS
  - OUTGROWTHS, NORMAL AND CANCER PATIENTS, 3459*

ERYTHROCYTE
- GLYCOPROTEIN ALTERATIONS
  - ERYTHROBLASTOSIS VIRUS TRANSFORMATION, CHICKEN, 3202*

ESOPHAGUS
- EPIDERMOID TUMORS
  - HYPERCALCEMIA, CLINICAL STUDY, 3503*
- POLYPLOID CARCINOMA
  - PSEUDOSARCOMATOUS FEATURES, CASE REPORT, 3414*

ESTRADIOL
- RECEPTORS
  - CYTOPLASM, PRECANCEROUS HUMAN ENDOMETRIUM, 3327*

ESTROGEN
- BREAST CANCER, MALE
  - URINARY EXCRETION, CLINICAL STUDY, 3575*
- CERVICAL CARCINOMA
  - VAGINAL SMEARS, CLINICAL STUDY, 3587*
- MAMMARY CARCINOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACE INDUCED, RAT, 3039
- MAMMARY TUMOR
  - DNA SYNTHESIS, HUMAN, 3356
- RECEPTOR
  - MAMMARY CARCINOMA, HUMAN CELL LINE, 3355
- RECEPTOR COMPLEX ASSOCIATION CONSTANT
  - MAMMARY CARCINOMA, CLINICAL STUDY, 3600*

N-ETHYL-N-NITROSOUREA
- RNA, TRANSFER
  - ALKYLATION, YEAST, ESCHERICHIA COLI, 3052

ETHYL STEARATE

- RETICULOENDOTHELIAL SYSTEM
    - IMMUNE RESPONSE, MOUSE, 3300*

ETHYLCARBAMATE
- DNA
    - PROLONGED BINDING, LIVER, MOUSE, 3099*

O-ETHYLDIALKYLNITROSIMMONIUM SALTS
- CARCINOGENESIS
    - RAT, 3047

N-ETHYLENEIMIDE
- ROUS SARCOMA VIRUS
    - REVERSE TRANSCRIPTASE, TRANSFORMING ACTIVITIES, 3127

ETHYLINITROSOUREA
- NERVOUS SYSTEM, CENTRAL
    - TUMOR INDUCTION, RAT, 3084*

ETHYLPHENYLPROPIOLATE
- CELL PROLIFERATION
    - EPIDERMAL HYPERPLASIA, MOUSE, 3076

EWING'S SARCOMA
- SERUM ALKALINE PHOSPHATASE
    - HUMAN, 3359

FECES
- BLOOD LOSS
    - RADIOACTIVELY LABELED, NORMAL HUMAN, 3462*

FECUNDITY
- CANCER MORTALITY
    - INFANTS, UNITED STATES, 3336*

FERRIC OXIDE
- TRACHEOBRONCHIAL EPITHELIUM
    - SURFACE MORPHOLOGY, MOUSE, 3068

ALPHA-FETOPROTEIN
- ENDODERMAL SINUS TUMOR
    - OVARY, CASE REPORT, 3520*
- LEVEL
    - SERA, ADULT MICE, 3260

FIBROADENOMA
- TRIPLE CARCINOMA OF BREAST
    - CASE REPORT, 3482*

FIBROBLAST
- 5-BROMO-2-DEOXYURIDINE
    - MEMBRANE FUNCTION, MOUSE, 3429*

FIBROMATOSIS
- SUBDERMAL, JUVENILE
    - CRANIUM BIFIDUM OCCULTA, CASE REPORT, 3456*

FIBROMYOMA
- PROTEIN METABOLISM
    - UTERINE TISSUE, HUMAN, 3405*

FIBROSARCOMA
- LEUKEMOID REACTION
    - MOUSE, 3305*
- LIVER
    - CYSTICERCUS FASCIOLARIS, RAT, 3543*
- PARANASAL SINUSES
    - CASE REPORT, 3385*
- SERUM ALKALINE PHOSPHATASE
    - HUMAN, 3359
- T- AND B-CELL SUBPOPULATIONS
    - SPLEEN, MOUSE, 3256
- TEMPORAL BONE
    - CASE REPORTS, 3467*

FIBROXANTHOMA
- ATYPICAL
    - CLINICAL, HISTOLOGIC, HISTOCHEMICAL STUDIES, 3428*

FLUORENYLHYDROXAMIC ACIDS
- MAMMARY CARCINOMA
    - RAT, 3051

FUCOSYLGLYCOLIPID
- METABOLISM
    - ONCORNAVIRUS-TRANSFORMED CELL LINES, 3193*

GASTRIC CANCER
- SPREAD FEATURES
    - CLINICAL SIGNIFICANCE, 3404*

GASTRIC CARCINOMA
- CLINICAL STUDY
    - LETTERKENNY COUNTY, IRELAND, 3386*
- LONG-TERM GASTROENTEROSTOMY
    - CASE REPORTS, 3541*
- POST-GASTRIC SURGERY
    - CLINICAL STUDY, 3496*

GASTRIC LESIONS
- COLD-+-RESTRAINT STRESS
    - RAT, 3427*

GASTROINTESTINAL CARCINOMA
- CARCINOEMBRYONIC ANTIGEN
    - SERUM CLINICAL STUDY, 3287*
- ETIOLOGY
    - REVIEW, 3001

GASTROINTESTINAL TRACT
- CARCINOID TUMORS
    - DIATHESIS, CASE REPORT, 3517*

GENETICS
- CHROMOSOMAL INSTABILITY
    - CANCER, HUMAN, 3545*
- MAPPING OF BASE SEQUENCE HETEROLOGIES
    - GENOMES FROM DIFFERENT ADENOVIRUS SEROTYPES, 3143
- NEOPLASTIC DIATHESIS
    - CASE REPORT, 3517*
- PHILADELPHIA CHROMOSOME
    - CHRONIC MYELOCYTIC LEUKEMIA, CLINICAL STUDY, 3369*
- PREDISPOSITION TO CANCER
    - DERMATOGLYPHIC STUDY, HUMAN, 3360
- SKIN CANCER
    - DIATHESIS, CLINICAL STUDY, 3509*
- TUMOR VARIABILITY
    - INDUCTION, HAMSTER, 3576*

- GESTATIONAL TROPHOBLASTIC DISEASE
  - NEOPLASMS, MALIGNANT
    - INCIDENCE, REVIEW, 3020*
- GLIOBLASTOMA
  - GIANT CELLS
    - ENDOEOPLASTIC TRANSFORMATION, TISSUE CULTURE, 3564*
- GLIOMA, MALIGNANT
  - HUMAN PAPOVAVIRUS(JC)-INDUCED
    - HAMSTER, 3139
- GLOMERULO NEPHRITIS
  - LEUKEMIA, MURINE
    - VIRUS-ANTIBODY COMPLEXES, MOUSE, 3266
- GLOMUS TUMOR
  - BILATERAL, TEMPORAL BONE
    - CASE REPORT, 3474*
- GLUCAGON
  - CYCLIC AMP AND ADENYLATE CYCLASE ACTIVITY
    - MALIGNANT HEPATOMA, ETHIONINE-TREATED RATS, 3055
- GLUCOCORTICOSTEROIDS
  - MONOCYTE PROLIFERATION
    - BONE MARROW, HUMAN, 3504*
- GLUCOSAMINE
  - METABOLISM
    - CULTURED NOVIKOFF HEPATOMA CELLS, RAT, 3419*
  - TRANSPORT
    - CULTURED NOVIKOFF HEPATOMA CELLS, RAT, 3419*
  - VIRUS, PICORNA
    - ISOLATION FROM CAPSIDS, 3216*
- GLUTATHIONASE
  - CARCINOGEN, CHEMICAL
    - LIVER, RAT, 3040
- GLYCERALDEHYDE-3-PHOSPHATE DEHYDROGENASE
  - SV40-TRANSFORMED CELLS
    - HUMAN DIPLOID CELLS, 3172*
- GLYCOGEN
  - METABOLISM
    - HELA CELLS, 3348
- GLYCOPROTEIN
  - ALTERATIONS
    - ERYTHROBLASTOSIS VIRUS TRANSFORMATION, CHICKEN ERYTHROCYTES, 3202*
  - INCORPORATION OF 2-DEOXY-D-GLUCOSE
    - SV40-TRANSFORMED HAMSTER CELLS, 3206*
- GRAFT-VS-HOST REACTION
  - PATHOGENESIS
    - CHICKEN EMBRYOS, 3315*
- GROWTH
  - AKR MOUSE CELLS IN SPINNER CULTURE
    - ROUS SARCOMA VIRUS-INDUCED, 3219*
  - ASCITES TUMOR
    - SELF LIMITATION, MOUSE, 3399*
  - GLUCOCORTICOIDS, MINERALOCORTICOIDS
    - CHILD, 3558*
  - POLYOMA-TRANSFORMED TUMOR CELL
    - SPIROMETRA MANSONOIDES-INFECTED HAMSTER, 3118
  - PROLIFERATION KINETICS
    - DENSITY-INHIBITED CULTURES, HUMAN CELLS, 3332
  - ROUS SARCOMA VIRUS-TRANSFORMED CELL
    - ANTIGENIC CHARACTERISTICS, HAMSTER, 3156
  - SELF REGULATION
    - THREE DIMENSIONS, TUMOR CELLS, HAMSTER, MOUSE, 3330
- GROWTH FACTOR
  - EPITHELIUM
    - CHARACTERISTICS, RABBIT, 3384*
  - FIBROBLASTS AND VIRAL TRANSFORMANTS
    - MOUSE, 3140
- GROWTH, TUMOR
  - DISULFIDE REDUCTION
    - PROLIFERATION, IMMUNOLOGY, MOUSE, 3346
  - ENHANCEMENT BY INTERFERON
    - MOLONEY SARCOMA VIRUS-INDUCED TUMORS, MOUSE, RAT, 3155
  - ENHANCEMENT BY POLY (I) POLY (C)
    - MOLONEY SARCOMA VIRUS-INDUCED TUMORS, MOUSE, RAT, 3155
  - INHIBITION
    - SILICA GEL PF 254 ELUATE, MOUSE, 3190*
  - INHIBITION BY BACTERIUM
    - MOLONEY LEUKEMIA VIRUS-INDUCED TUMOR, MOUSE, 3282*
  - INVASIVE
    - MECHANICAL ASPECTS, 3324*
- HARD PALATE CARCINOMA
  - TOBACCO SMOKING
    - INCIDENCE, REVERSE SMOKERS, 3335
- HEAD
  - CANCER
    - ALCOHOLICS, SMOKING DIET, CLINICAL STUDY, 3411*
  - EPIDERMOID TUMORS
    - HYPERCALCEMIA, CLINICAL STUDY, 3503*
  - RHABDOMYOSARCOMA
    - CHILDREN, 3480*
  - SYNOVIAL SARCOMA
    - CASE REPORTS, 3454*
- HEART
  - METASTASES
    - ANITSCHKOW NUCLEAR STRUCTURE, CLINICAL STUDY, 3523*
- HELA CELLS
  - METABOLISM
    - GLYCOGEN, 3348
  - MITOCHONDRIAL PROTEIN SYNTHESIS
    - ATYPICAL AMINO ACID UTILIZATION,

  3593*

HEMANGIOBLASTOMA
  LINDAU'S DISEASE
    FAMILIAL OCCURRENCE, CASE REPORT, 3351

HEMANGIOMA
  MANDIBLE, CENTRAL
    CASE REPORT, 3465*
  ORAL CAVITY, CAVERNOUS
    REVIEW, CASE REPORT, 3464*

HEPATOCELLULAR CARCINOMA
  CHORIONIC GONADOTROPIN
    ECTOPIC PRODUCTION, CLINICAL STUDY, 3516*

HEPATOMA
  SEE ALSO LIVER
  DNA SYNTHESIS
    DIBUTYRYL CYCLIC AMP, 3571*
  LYSOSOMES
    ELECTRON-HISTOCHEMICAL INVESTIGATION, MOUSE, 3447*
  3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    ENZYME PATTERN, RAT, 3038
    RAT, 3037
  5'-NUCLEOTIDE PHOSPHODIESTERASE
    ISOENZYME PATTERN, HUMAN, 3353
  PRIMARY, MALIGNANT
    BANTU MINEWORKERS, CLINICAL STUDY, 3595*
  STIMULATION
    SYNTHETIC TRIPEPTIDE, HUMAN, 3339
  THOROTRAST-INDUCED
    PRESENTING AS HYPERPARATHYROIDISM, CASE REPORT, 3542*
  TRABECULAR TYPE
    ULTRASTRUCTURE, HUMAN, 3530*
  TRANSPLANTATION
    CELL PROLIFERATION, HOST TISSUE, MOUSE, 3432*

HEPATOMA, ASCITES
  BACK TRANSPLANTABILITY
    TISSUE CULTURE, RAT, 3379*

HEPATOMA, MALIGNANT
  ADENYLATE CYCLASE
    GLUCAGON, PROSTAGLANDIN E1 STIMULATION, ETHIONINE-TREATED RAT, 3055
  CYCLIC AMP
    GLUCAGON, PROSTAGLANDIN E1 STIMULATION, ETHIONINE-TREATED RAT, 3055

HEPATOMA, MORRIS
  DNA POLYMERASE
    SEPARABLE ACTIVITIES, HOST LIVER, RAT, 3582*
  MITOCHONDRIA, MICROSOMES
    CHOLESTEROL, PHOSPHOLIPID COMPOSITION, RAT, 3584*
  PROTEIN TURNOVER
    MICROSOMAL SUBFRACTIONS, RAT, 3583*

HEPATOMA, NOVIKOFF
  ARGININE ACCEPTOR-PROTEINS
    LIVER, RAT, 3392*
  GLUCOSAMINE METABOLISM, TRANSPORT
    CULTURED CELLS, RAT, 3419*

HEPATOMA, SEIDEL ASCITIC
  CELL PROTEINS
    FRACTIONAL CONTENT, 3363*

HEPTAMETHYLENEIMINE
  WITH NITRITE
    LIVER, LUNG MALIGNANT TUMORS, RAT, 3090*

HISTIOCYTOMA, FIBROUS
  PARANASAL SINUSES
    CASE REPORT, 3468*

HISTIOCYTOSIS, MALIGNANT
  ROBB-SMITH TYPE
    CASE REPORT, 3430*

HODGKIN'S DISEASE
  CLINICOPATHOLOGY
    HUMAN, 3497*
  FERRITINEMIA
    CLINICAL STUDY, 3400*
  LAPAROTOMY, SPLENECTOMY
    CLINICAL STUDY, 3493*
  MELKERSSON-ROSENTHAL SYNDROME
    CORNEAL KERATOPATHY, CASE REPORT, 3579*
  SERUM COPPER LEVELS
    CLINICAL STUDY, 3556*
  SPLEEN SCANNING
    CLINICAL STUDY, 3565*
  THYMUS-DERIVED LYMPHOCYTE DEPLETION
    CLINICAL STUDY, 3299*

HORMONE
  SEE ALSO NAMES OF
  CONTROL OF ENZYME ACTIVITY
    CARCINOGENESIS, 3472*
  ECTOPIC PRODUCTION IN TUMORS
    WATER, ELECTROLYTE BALANCE, HUMAN, REVIEW, 3394*
  GLUCOCORTICOSTEROIDS
    MONOCYTE PROLIFERATION, BONE MARROW, HUMAN, 3504*
  MELANOCYTE STIMULATING
    CONTROL PHENOTYPIC EXPRESSION, MELANOMA, MOUSE, 3533*

HORMONE, ADRENOCORTICOTROPHIC
  SEROTONIN
    OAT-CELL CARCINOMA, LUNG, CLINICAL STUDY, 3455*

HYALURONIC ACID
  LYMPHOMA
    CELL AGGREGATION, CAT, 3435*

HYCANTHONE
  TRANSFORMATION OF RAUSCHER VIRUS-INFECTED CELL CULTURES
    RAT EMBRYO CELLS, 3149

HYDANTOIN

- LEUKEMIA
  - HUMAN, 3079*

HYDROCARBON, POLYCYCLIC
- MAXIMALLY PERMISSIBLE DOSES, CONCENTRATIONS
  - REVIEW, 3027*

HYDROCEPHALUS
- NITROSAMIDE-INDUCED
  - PREVENTION BY ASCORBIC ACID, RAT, 3086*

HYDROCORTISONE
- CELL VIABILITY
  - HUMAN LYMPHOBLASTOID CELL LINES, 3119
- EPSTEIN-BARR VIRUS GENOME EXPRESSION
  - HUMAN LYMPHOBLASTOID CELL LINES, 3119
- INTERFERON SYNTHESIS
  - HUMAN LYMPHOBLASTOID CELL LINES, 3119

N-HYDROXY-2-FLUORENYLACETAMIDE
- MAMMARY CARCINOMA
  - RAT, 3051

HYPERCALCEMIA
- ACUTE MYELOMONOCYTIC LEUKEMIA
  - BENCE JONES PROTEINURIA, MACROGLOBULINEMIA, CASE REPORT, 3498*
- EPIDERMOID TUMORS
  - HEAD, NECK, ESOPHAGUS, CLINICAL STUDY, 3503*

IMMUNE RESPONSE
- ADENOVIRUS 12-INDUCED ANTIGENS
  - MACROPHAGE INHIBITION TEST, MOUSE, 3263
- ANTI-TUMOR ACTIVITY
  - FETAL TISSUES, MOUSE, 3244
- ANTI-TYROSINASE ANTIBODY PRODUCTION
  - RABBIT, 3302*
- ASCITES, TUMOR CELLS
  - DEPENDENCE ON LYMPHOCYTE AND ANTISERA ORIGIN, 3262
- DELAYED CUTANEOUS HYPERSENSITIVITY REACTIONS
  - MALIGNANT AND NORMAL HUMAN BREAST CELLS, 3261
- FAMILY
  - ACUTE LYMPHOBLASTIC LEUKEMIA, CHILDREN, 3237
- INHIBITION BY 7S ANTIBODY
  - MECHANISM AND SITE OF ACTION, MOUSE, 3255
- LYMPHOCYTE
  - INITIATING MECHANISM, MOUSE, 3227
  - PLASMA CELL TUMORS, MOUSE, 3253
  - SERUM-INHIBITORY FACTORS, CANCER PATIENTS, 3247
- RETICULOENDOTHELIAL SYSTEM
  - ETHYL STEARATE, METHYL PALMITATE, MOUSE, 3300*
- RNA DEPENDENT-DNA POLYMERASE
  - RABBIT, 3259
- SUPPRESSION
  - 6- MERCAPTOPURINE, GUINEA PIG, 3250
- TRANSPLANTED TUMOR
  - LYMPHOID CELL DISTRIBUTION, MOUSE, 3239

IMMUNITY
- CELL-MEDIATED
  - DEPENDENCE ON LYMPHOCYTE AND ANTISERA ORIGIN, 3262
  - 6- MERCAPTOPURINE, GUINEA PIG, 3250
- CELL-MEDIATED TUMOR
  - SPECIFICITY CELL DESTRUCTION, MOLONEY SARCOMA REGRESSORS, MOUSE, 3236
- PLASMA CELL TUMOR
  - MOUSE, 3253
- SERUM-MEDIATED
  - DEPENDENCE ON LYMPHOCYTE AND ANTISERA ORIGIN, 3262
- SYNGENEIC TUMOR
  - ANTIGEN-SPECIFIC IMMUNOGLOBULIN, CYTOTOXIC EFFECTOR CELLS, MOUS, 3269
- WALKER CARCINOSARCOMA
  - SERUM BLOCKING FACTOR, MACROPHAGE INACTIVATION, RAT, 3249

IMMUNITY, CELLULAR
- ADENOVIRUS 12-INDUCED ANTIGENS
  - MACROPHAGE INHIBITION TEST, MOUSE, 3263
- MALIGNANCY, HUMAN
  - EMBRYONAL CELLS, HUMAN, 3254

IMMUNODEFICIENCY
- LYMPHORETICULAR SARCOMA
  - MOUSE, 3295*

IMMUNOGENICITY
- ONCOGENIC POTENTIAL
  - SV40-TRANSFORMED CELLS, MOUSE, 3251

IMMUNOGLOBULIN
- ANTIGEN-SPECIFIC
  - SYNGENEIC TUMOR IMMUNITY, CYTOTOXIC EFFECTOR CELLS, MOUSE, 3269
- DISTRIBUTION ON B CELLS
  - HUMAN, 3229
- LEUKEMIA, CHRONIC LYMPHOCYTIC
  - HUMAN, 3228
- SURFACE
  - HUMAN LEUKEMIA CELLS, 3240

IMMUNOGLOBULIN E
- ISOLATION
  - SERUM, MYELOMA PATIENT, 3313*

IMMUNOLOGY
- ANTI-IGG
  - ISOLATION AND IMMUNOCHARACTERIZATION, RABBIT, 3264
- BRAIN TUMOR
  - IMMUNOTHERAPY, REVIEW, 3023*
- CANCER

    - REVIEW. 3031*
  - COLON CARCINOMA
    - RADIOIMMUNOASSAY FOR CARCINOEMBRYONIC ANTIGEN. HUMAN. 3311*
  - IMMUNE PROLIFERATION
    - PARAPROTEINS. REVIEW. 3029*
  - IMMUNOLOGICAL TOLERANCE BREAKDOWN
    - GROSS VIRUS-INDUCED CELL-SURFACE ANTIGENS. RAT. 3235
  - LEUKEMIA
    - REVIEW. 3009. 3016*
  - LEUKEMIA CHRONIC LYMPHOID
    - HUMAN. 3285*
  - PRIMARY IMMUNODEFICIENCY DISEASES
    - THE IMMUNODEFICIENCY-CANCER REGISTRY. 3277
  - TUMOR-SPECIFIC PROTEINS
    - BRAIN TUMORS. HUMAN. 3274

- IMMUNOSENSITIVITY
  - ONCOGENIC POTENTIAL
    - SV40-TRANSFORMED CELLS. MOUSE. 3251

- IMMUNOSUPPRESSION
  - CHEMICAL CARCINOGENESIS
    - REGIONAL LYMPHADENECTOMIZED RATS. 3270

- IMMUNOSURVEILLANCE
  - BURSA- AND THYMUS-DEPENDENT
    - AVIAN RETICULOENDOTHELIOSIS VIRUS-INDUCED TUMOR. CHICKEN. 3192*

- IMMUNOTHERAPY
  - MOLONEY SARCOMA VIRUS-INDUCED TUMORS
    - MOUSE. 3173*

- INFECTIOUS MONONUCLEOSIS
  - VIRUS. EPSTEIN-BARR
    - CLINICAL STUDY. 3144

- INTERFERON
  - LEUKEMIA
    - MACROMOLECULAR SYNTHESIS IN VITRO. MOUSE. 3501*
    - RNA. PROTEIN. POLYRIBOSOME SYNTHESIS. MOUSE. 3591*
  - SYNTHESIS
    - EFFECT OF HYDROCORTISONE. HUMAN LYMPHOBLASTOID CELL LINES. 3119
  - TUMOR GROWTH ENHANCEMENT
    - MOLONEY SARCOMA VIRUS-INDUCED TUMORS. MOUSE. RAT. 3155

- INTESTINE
  - TUMOR INDUCTION
    - BENZIDINE. RAT. 3297*

- INTESTINE. LARGE
  - COLON CARCINOMA
    - CASE PRESENTATION. 3566*

- IODODEOXYURIDINE
  - HERPESVIRUS HOMINIS INFECTION
    - THERAPY. MOUSE. 3221*

- JAW
  - ADENOMATOID ODONTOGENIC TUMOR
    - CASE REPORT. 3450*
  - CENTRAL HEMANGIOMA
    - CASE REPORT. 3465*

- JENSEN SARCOMA
  - DIAGNOSIS
    - SEROLOGICAL INVESTIGATIONS WITH CLOSTRIDIAL SPORES. RAT. HAMSTE. 3301*

- KERATOACANTHOMA
  - TATTOO
    - CASE REPORT. 3567*

- KIDNEY
  - HYPERNEPHROMA
    - LANDAU'S DISEASE. FAMILIAL OCCURRENCE. 3351
  - LEAD-INDUCED CHRONIC DISEASE
    - ENZYME HISTOCHEMISTRY. RAT. 3096*
  - RENAL CARCINOMA
    - PREGNANCY. CASE REPORTS. 3433*

- LACTATE DEHYDROGENASE
  - SERUM
    - CHANGE. CANCER PATIENTS. 3471*

- LARYNGEAL PAPILLOMA
  - VIRUS. PAPOVA
    - ELECTRON MICROSCOPIC IDENTIFICATION. 3142

- LARYNX
  - CARCINOMA
    - HISTOLOGIC CLASSIFICATION. GRADING OF MALIGNANCY. HUMAN. 3537*
  - OXYPHIL ADENOMA
    - CASE REPORTS. 3457*
  - PAPILLOMA
    - PAPOVA VIRUS. ELECTRON MICROSCOPIC IDENTIFICATION. 3142

- LEAD
  - CHRONIC KIDNEY DISEASE INDUCTION
    - ENZYME HISTOCHEMISTRY. RAT. 3096*

- LECTIN
  - DETECTION OF SURFACE ALTERATIONS
    - TRANSFORMED CELLS. 3265
  - ROTATIONAL DIFFUSION
    - LYMPHOCYTE MEMBRANE. RAT. 3341

- LEUKEMIA
  - AUTOIMMUNE DISORDERS
    - CYTOTOXICITY. IMMUNOFLUORESCENCE. CLINICAL STUDY. 3280*
  - BONE MARROW
    - OSTEOGENIC POTENTIAL. RAT. 3555*
  - BONE MARROW CELLS
    - ELECTROPHORETIC CELL SEPARATION. HUMAN. 3531*
  - EPIDEMIOLOGY
    - NEBRASKA. REVIEW. 3030*
  - ERYTHROCYTIC. LEUKOCYTIC. SERUM ALLOTYPES
    - ALTERED EXPRESSION. 3284*
  - HYDANTOIN
    - HUMAN. 3079*

- IMMUNOLOGY
  - REVIEW, 3009, 3016*
- INCIDENCE
  - CHILDREN, CHILE, 3331
- INTERFERON
  - MACROMOLECULAR SYNTHESIS IN VITRO, MOUSE, 3501*
  - RNA, PROTEIN, POLYRIBOSOME SYNTHESIS, MOUSE, 3591*
- LEUKOCYTE KINETICS
  - REVIEW, 3003
- LIPID ANTIOXIDATIVE ACTIVITY
  - MOUSE, 3362*
- LYMPHOCYTE ALPHA AMINOISOBUTYRIC ACID TRANSPORT
  - CORTISOL-INDUCED CHANGES, HUMAN, 3393*
- MYELOBLAST ALPHA AMINOISOBUTYRIC ACID TRANSPORT
  - CORTISOL-INDUCED CHANGES, HUMAN, 3393*
- PARALYMPHOBLASTS
  - ABO SYSTEM TRANSPLANTATION ANTIGEN, HUMAN, 3283*
- PHAGOCYTIC ACTIVITY
  - CLINICAL STUDY, 3375*
- PREVENTION
  - MAREK'S DISEASE, REVIEW, 3022*
- RADIATION, X-RAY
  - SYNERGISTIC EFFECT OF URETHANE, RAT, 3110
- REDUCTION FOLLOWING SPLENECTOMY
  - RAT, 3388*
- REGRESSION, SPONTANEOUS
  - AVIAN MYELOBLASTOSIS VIRUS-INFECTED CHICKEN, 3159
- REVERSE TRANSCRIPTASE
  - PLASMA, MOUSE, CHICKEN, 3169
- RNA ISOLATION
  - HUMAN, 3349
- RNA SYNTHESIS
  - INTERFERON EFFECT, MOUSE, 3591*
- SCREENING
  - CAPILLARY HEMATOCRIT, MICROSCOPIC OBSERVATION, RAT, 3371*
- SOMATOTROPIC ACTIVITY
  - BLOOD SERUM, CLINICAL STUDY, 3406*
- 6-THIOPURINE SENSITIVE, RESISTANT
  - PURINE PHOSPHORIBOSYLTRANSFERASE, HUMAN, 3502*
- VIRUS
  - REVIEW, 3017*

LEUKEMIA, ACUTE
- BACTEREMIA
  - CLINICAL STUDY, 3581*
- DIFFERENTIAL DIAGNOSIS
  - LEUKOCYTE MEAN CORPUSCULAR VOLUME, CLINICAL STUDY, 3453*
- HEMATOLOGICAL DISORDERS
  - CYTOGENETIC STUDIES, HUMAN, 3580*
- LACTIC ACIDOSIS
  - METABOLIC, THERAPEUTIC STUDIES, CASE REPORT, 3592*
- LEUKOCYTE KINETICS
  - REVIEW, 3003
- PLATELET METABOLISM
  - CLINICAL STUDY, 3538*
- T AND B LYMPHOCYTES
  - BLOOD, CLINICAL STUDY, 3290*

LEUKEMIA, ACUTE LYMPHOBLASTIC
- BLAST CELLS
  - GLUCOCORTICOID-BINDING, HUMAN, 3492*
- CHILDREN
  - IMMUNE RESPONSE IN FAMILIES, 3237
- GLUCOCORTICOID BINDING PROTEINS
  - LYMPHOBLAST, HUMAN, 3534*

LEUKEMIA, ACUTE LYMPHOID
- ALLOGENEIC MARROW GRAFTING DURING MARROW REMISSION
  - HUMAN, 3292*

LEUKEMIA, ACUTE MYELOBLASTIC
- FERRITINEMIA
  - CLINICAL STUDY, 3400*

LEUKEMIA, ACUTE MYELOID
- CYTOSINE ARABINOSIDE
  - MYELOBLAST CELL CYCLE, CLINICAL STUDY, 3423*

LEUKEMIA, ACUTE MYELOMONOCYTIC
- MACROGLOBULINEMIA, BENCE JONES PROTEINURIA, HYPERCALCEMIA
  - CASE REPORT, 3498*

LEUKEMIA, ACUTE PROMYELOCYTIC
- AUER BODIES
  - ULTRASTRUCTURE, HUMAN, 3365*

LEUKEMIA BASOPHILIC
- BASOPROTEIN
  - RAT, 3505*

LEUKEMIA, CHRONIC
- BACTEREMIA
  - CLINICAL STUDY, 3581*

LEUKEMIA CHRONIC
- FREE NUCLEOTIDES IN LEUKOCYTES
  - CLINICAL STUDY, 3380*

LEUKEMIA, CHRONIC GRANULOCYTIC
- LEUKOCYTE KINETICS
  - REVIEW, 3003
- PROLONGED SURVIVAL
  - MUSCLE INFILTRATION, SEA-BLUE HISTIOCYTOSIS, CASE REPORT, 3577*

LEUKEMIA, CHRONIC LYMPHATIC
- NON-IMMUNOGLOBULIN-BEARING B LYMPHOCYTES
  - CLINICAL STUDY, 3308*

LEUKEMIA, CHRONIC LYMPHOCYTIC
- ANNULATE LAMELLAE
  - CLINICAL STUDY, 3387*
- B-LYMPHOCYTES
  - MALIGNANT TRANSFORMATION, HUMAN, 3574*
- IMMUNOGLOBULIN
  - HUMAN, 3228
- LEUKOCYTE KINETICS
  - REVIEW, 3003

THYMUS-DERIVED PROLIFERATING
LYMPHOCYTES
HUMAN, 3461*

LEUKEMIA CHRONIC LYMPHOID
CYTOCHEMISTRY
HUMAN, 3285*
IMMUNOLOGY
HUMAN, 3285*

LEUKEMIA, CHRONIC MYELOCYTIC
PHILADELPHIA CHROMOSOME
CLINICAL STUDY, 3369*

LEUKEMIA, CHRONIC MYELOID
MYELOBLASTS, LIPID-CONTAINING VACUOLES
ULTRASTRUCTURE, CASE REPORT, 3487*
PLATELET FUNCTIONS
CLINICAL STUDY, 3378*

LEUKEMIA, LYMPHOCYTIC
ASCITES TUMOR
ZINC DEFICIENCY, MOUSE, 3352

LEUKEMIA, LYMPHOID
C-TYPE RNA VIRUS ISOLATION
CATTLE, 3158

LEUKEMIA, MONOCYTIC
HYPERLYSOZYMEMIA
BACTERIOLYTIC, BACTERICIDAL
ACTIVITY, CLINICAL STUDY, 3391*

LEUKEMIA, MURINE
GLOMERULO NEPHRITIS
VIRUS-ANTIBODY COMPLEXES, MOUSE,
3266
SELF REGULATION OF GROWTH
MOUSE, 3330

LEUKEMIA, MYELOMONOCYTIC
HYPERLYSOZYMEMIA
BACTERIOLYTIC, BACTERICIDAL
ACTIVITY, CLINICAL STUDY, 3391*

LEUKEMIA, PROLYMPHOCYTIC
B AND T CELL TYPE
CLINICAL STUDY, 3286*

LEUKEMIA, RADIOGENIC
CHILDHOOD CANCER
IN UTERO DEATHS, 3115*

LEUKOCYTE
KINETICS
LEUKEMIA, REVIEW, 3003

LINDAU'S DISEASE
FAMILIAL INCIDENCE
CASE REPORT, 3351

LIPID
ANTIOXIDATIVE ACTIVITY
LEUKEMIA, MOUSE, 3362*

LIPOPOLYSACCHARIDE
EFFECT ON RESISTANCE
EHRLICH TUMOR CELLS, MOUSE, 3307*

LIVER
DIETHYLNITROSAMINE-INDUCED CARCINOMA
DEOXYRIBONUCLEASES, PHOSPHATASES,
RAT, 3085*
MALIGNANT TUMOR
AMINOPYRINE, HEPTAMETHYLENEIMINE
WITH NITRITE, RAT, 3090*
METASTATIC TUMOR
GAMMAGLUTAMYL TRANSPEPTIDASE,
CLINICAL STUDY, 3418*
MITOSIS
CELL CLASSES, RAT, HAMSTER, 3100*
REGENERATION
HEMATOPOIETIC STEM CELLS, MOUSE,
3544*

LUCANTHONE
TRANSFORMATION OF RAUSCHER VIRUS-
INFECTED CELL CULTURES
RAT EMBRYO CELLS, 3149

LUCKE TUMOR
VIRUS, HERPES
RNA TRANSCRIPTION, FROG, 3124

LUNG
ADENOMA
CHOLESTERYL-14-METHYLHEXADECANOATE,
TRANSPLACENTAL EFFECT, MOUS,
3043
ASBESTOS
PLEURAL PLAQUES, HUMAN, 3045
HORMONE PRODUCING TUMORS
REVIEW, 3012*
HUMAN PERIPHERAL TUMORS
LIGHT AND ELECTRON MICROSCOPE
CORRELATION, 3519*
MALIGNANT TUMOR
AMINOPYRINE, HEPTAMETHYLENEIMINE
WITH NITRITE, RAT, 3090*
NICKEL SUBSULFIDE, BENZO(A)PYRENE
INJECTION
PATHOLOGICAL REACTION, RAT, 3104*
OAT-CELL CARCINOMA
ACTH, SEROTONIN PRODUCTION,
CLINICAL STUDY, 3455*

LUNG CANCER
CARCINOEMBRYONIC ANTIGEN
SERUM CLINICAL STUDY, 3287*
INCIDENCE
URANIUM MINERS, UNITED STATES,
3334
METASTASIS
TRITON WR1339 INJECTION, MOUSE,
3396*
OCCUPATIONAL HAZARD
URANIUM MINERS, UNITED STATES,
3334
PRIMARY SCAR CARCINOMA
HUMAN, 3319

GAMMAGLUTAMYL TRANSPEPTIDASE
LIVER DISEASE
METASTATIC, NON-METASTATIC,
CLINICAL STUDY, 3418*

LYMPH NODE CELL
RESPONSE TO ABELSON LEUKEMIA VIRUS

    - ULTRASTRUCTURE, MOUSE, 3151

- LYMPHADENOPATHY
  - SINUS HISTIOCYTOSIS
    - ELEVATED EBV ANTIBODY TITERS, CASE REPORT, 3225*

- LYMPHOCYTE
  - BLASTOGENIC RESPONSE
    - AUTOCHTHONOUS TUMOR CELLS, HUMAN, 3243
  - CONCANAVALIN A-INDUCED TRANSFORMATION
    - S-ADENOSYL-L-METHIONINE DECARBOXYLASE, 3557*
  - EFFECT OF CONCANAVALIN A
    - HUMAN, 3314*
  - IMMUNE RESPONSE
    - INITIATING MECHANISM, MOUSE, 3227
    - PLASMA CELL TUMORS, MOUSE, 3253
    - SERUM-INHIBITORY FACTORS, CANCER PATIENTS, 3247
  - MIXED REACTIONS
    - ABSENCE OF STIMULATION BY IRRADIATED ALLOGENEIC CELLS, 3293*
  - NON-IMMUNOGLOBULIN-BEARING B
    - CHRONIC LYMPHATIC LEUKEMIA PATIENTS, 3308*
  - SEPARATION
    - FLOTATION ON FICOLL TRIOSIL MIXTURE, 3543*
  - SEPARATION OF LYMPHOID CELLS
    - VELOCITY SEDIMENTATION TECHNIQUE, HUMAN, 3559*
  - SURFACE MEMBRANE LECTINS
    - ROTATIONAL DIFFUSION, MOUSE, 3341
  - T AND B CELL
    - ANTI-IG ANTIBODY, ANTIGEN RECEPTOR BLOCKING, REDISTRIBUTION, 3271
    - DISTRIBUTION OF HISTOCOMPATIBILITY ANTIGENS, GUINEA PIG, 3306*
    - LYMPHOMA, ACUTE LEUKEMIC PATIENT, 3290*
  - T- AND B-CELL SUBPOPULATIONS
    - FIBROSARCOMA, MOUSE SPLEEN, 3256
  - T-CELL DEPLETION
    - HODGKIN'S DISEASE, CLINICAL STUDY, 3299*
  - THYMUS-DERIVED PROLIFERATING
    - CHRONIC LYMPHOCYTIC LEUKEMIA, HUMAN, 3461*
  - TRANSFORMATION
    - STANDARDIZATION TO PHYTOHEMAGGLUTININ, 3296*
  - TUMOR-CELL DESTRUCTION
    - SERA, CANCER PATIENTS, 3232

- LYMPHOGRANULOMATOSIS
  - BLOOD COAGULATION
    - CLINICAL STUDY, CHILDREN, 3410*

- LYMPHOMA
  - BACTEREMIA
    - CLINICAL STUDY, 3581*
  - BREAST, PRIMARY
    - CASE REPORTS, 3578*
  - CUTIS
    - EXPERIMENTAL MOUSE, HUMAN DISEASE COMPARISON, 3550*
  - EPIDEMIOLOGY
    - NEBRASKA, REVIEW, 3030*
  - HYALURONIC ACID
    - CELL AGGREGATION, CAT, 3435*
  - LYMPHOCYTE CHARACTERISTICS
    - CORTISOL, PHYTOHEMAGGLUTININ, MOUSE, 3413*
  - NUCLEIC ACID METABOLISM
    - CYTOCHEMICAL STUDY, HUMAN, 3417*
  - NUCLEOTIDE POOL SIZES
    - MOUSE, 3358
  - PITUITARY TUMOR
    - VIRUS MORPHOGENESIS, MOUSE, 3167
  - PREVENTION
    - MAREK'S DISEASE, REVIEW, 3022*
  - PURINE NUCLEOTIDE METABOLISM
    - MOUSE, 3358
  - RETROPERITONEAL
    - GLOMERULAR LESIONS, IMMUNOHISTOCHEMICAL STUDY, 3310*
  - SPLENIC POOL SIZE
    - CLINICAL STUDY, 3570*
  - T AND B LYMPHOCYTES
    - BLOOD, CLINICAL STUDY, 3290*

- LYMPHOMA, MALIGNANT
  - HISTOPATHOLOGIC DIAGNOSIS
    - RELIABILITY, FINLAND, 3451*
  - INCIDENCE
    - CHILDREN, CHILE, 3331
  - MONOCLONAL GAMMOPATHIES
    - CASE REPORTS, 3309*

- LYMPHORETICULAR NEOPLASM
  - VIRUS, C-TYPE RNA
    - MOUSE, 3125
  - VIRUS, MURINE LEUKEMIA
    - MOUSE, 3125

- LYMPHORETICULAR SARCOMA
  - BONE MARROW ALTERATIONS
    - CLINICAL STUDY, 3446*
  - IMMUNODEFICIENCY
    - MOUSE, 3295*

- LYMPHORETICULAR SYSTEM
  - TUMOR INDUCTION
    - MURINE C-TYPE RNA VIRUS, MOUSE, 3134

- LYMPHOSARCOMA
  - ANNULATE LAMELLAE
    - CLINICAL STUDY, 3387*
  - VIRUS, C-TYPE PARTICLE
    - GIBBON, 3222*

- MACROPHAGE
  - ELECTROPHORETIC MIGRATION TEST
    - CANCER PATIENTS, 3294*

- MALIGNANCY
  - BLOOD CLOT RETRACTION
    - CLINICAL STUDY, 3407*
  - GRADING
    - CARCINOMA OF LARYNX, HUMAN, 3537*

- MAMMARY ADENOCARCINOMA
  - CELL POPULATION KINETICS
    - RAT, 3328

MAMMARY CARCINOMA
- ANDROGEN
  - ATYPICAL ENDOMETRIAL PATTERN, CLINICAL STUDY, 3420*
- CARCINOEMBRYONIC ANTIGEN
  - SERUM CLINICAL STUDY, 3287*
- CLASSIFICATION
  - TNM SYSTEM, CANADA, 3434*
- CYTOLOGIC DETECTION
  - CLINICAL-PATHOLOGIC APPROACHES, 3437*
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - ESTROGEN EFFECT, RAT, 3039
  - PROGESTERONE EFFECT, RAT, 3039
- DISSEMINATION ROUTES
  - AUTOPSY STUDY, HUMAN, 3389*
- ESTRADIOL BINDING
  - CLINICAL STUDY, 3600*
- ESTROGEN RECEPTOR
  - HUMAN CELL LINE, 3355
- FLUORENYLHYDROXAMIC ACIDS
  - RAT, 3051
- N-HYDROXY-2-FLUORENYLACETAMIDE
  - RAT, 3051
- MAST CELLS
  - CLINICAL STUDY, 3372*
- PLASMA PROLACTIN
  - CLINICAL STUDY, 3401*
- TESTOSTERONE
  - ATYPICAL ENDOMETRIAL PATTERN, CLINICAL STUDY, 3420*
- TRIPLE
  - FIBROADENOMA, CASE REPORT, 3482*
- TRYPTOPHAN METABOLISM
  - URINARY STEROID EXCRETION, CLINICAL STUDY, 3416*

MAMMARY GLAND
- ADENOCARCINOMA
  - CELL POPULATION KINETICS, RAT, 3328
- CELL PROLIFERATION
  - HORMONAL EFFECTS, IN VITRO STUDIES, 3321*

MAMMARY TUMOR
- 7,12-DIMETHYLBENZ(A)ANTHRACENE
  - INDUCTION, HAMSTER, 3081*
- ESTROGEN
  - DNA SYNTHESIS, HUMAN, 3356
- PROGESTERONE
  - DNA SYNTHESIS, HUMAN, 3356
- TESTOSTERONE
  - DNA SYNTHESIS, HUMAN, 3356

MAREK'S DISEASE
- EPIDEMIOLOGY
  - INCIDENCE OF PRECIPITINS, ZOO BIRDS, 3217*
- LEUKEMIA PREVENTION
  - REVIEW, 3022*
- LYMPHOMA PREVENTION
  - REVIEW, 3022*

MASTOCYTOMA
- LEUKOKININ-FORMING SYSTEM
  - ASCITIC FLUID, MOUSE, 3522*

MEDULLARY CARCINOMA
- THYROID
  - ORAL MUCOSAL NEUROMA, CASE REPORT, 3452*

MELANOMA
- CONTROL OF PHENOTYPIC EXPRESSION
  - MELANOCYTE STIMULATING HORMONES, MOUSE, 3533*
- PEROXIDATIVE OXIDATION, TYROSINE TO MELANIN
  - HYDROXYFUMARATE COFACTOR, MOUSE, 3469*
- RADIATION
  - MOUSE, 3116*
- SELF REGULATION OF GROWTH
  - MOUSE, 3330
- TREATMENT WITH HIGH-SPEED ELECTRONS
  - MOUSE, 3116*
- VIRAL ETIOLOGY
  - HUMAN, 3194*

MELANOMA, AMELANOTIC
- DIAGNOSIS
  - SEROLOGICAL INVESTIGATIONS WITH CLOSTRIDIAL SPORES, RAT, HAMSTE, 3301*

MELANOMA, MALIGNANT
- CHROMOSOME ABERRATIONS
  - CLINICAL STUDY, 3484*
- CYTOLOGIC DIAGNOSIS
  - CLINICAL STUDY, 3440*
- DIAGNOSIS
  - CLINICAL STUDY, AUSTRALIA, 3560*
- ECTOPIC ADRENOCORTICOTROPIN
  - CLONAL CELL LINE, HUMAN, 3535*
- ETIOLOGY
  - SUNLIGHT, REVIEW, 3025*
- INCIDENCE
  - THAILAND, 3333
- RADIATION, UV
  - SUPERSONIC TRANSPORT, REVIEW, 3002

MEMBRANE
- 5-BROMO-2-DEOXYURIDINE
  - MOUSE FIBROBLASTS, 3429*
- ENDOPLASMIC RETICULUM
  - AFLATOXIN B1, LIVER, RAT, 3390*

MENINGIOMA
- CHROMOSOME ABERRATIONS
  - ACROCENTRICITY, NUCLEOLAR AREA, HUMAN, 3586*
- HYPERDIPLOID-HYPOTRIPLOID STEMLINES
  - FLUORESCENCE KARYOTYPES, HUMAN, 3561*

6- MERCAPTOPURINE
- IMMUNE RESPONSE SUPPRESSION
  - GUINEA PIG, 3250

MESONEPHRIC ADENOCARCINOMA
- CASE REPORT, 3513*

MESOTHELIOMA
- DIFFUSE, PLEURAL
  - ULTRASTRUCTURE, CASE REPORTS, 3415*

METABOLISM
    A-OXOGLUTARATE TRANSPORT
        MITOCHONDRIA, ASCITES TUMOR, 3573*
    BLOOD MALATE, OXALOACETATE LEVEL
        BENIGN SURGICAL MALIGNANT DISEASE, HUMAN, 3490*
    FUCOSYLGLYCOLIPID
        ONCORNAVIRUS-TRANSFORMED CELL LINES, 3193*
    GLUCOSAMINE
        CULTURED NOVIKOFF HEPATOMA CELLS, RAT, 3419*
    GLYCOGEN
        HELA CELLS, 3348
    GLYCOLYTIC MECHANISM
        6,6'-DITHIODINICOTINIC ACID, 2,2'-DITHIODIPYRIDINE, MOUSE, 3458*
    LIPID ANTIOXIDATIVE ACTIVITY
        LEUKEMIA, MOUSE, 3362*
    NUCLEIC ACID
        LYMPHOMA, HUMAN, 3417*
    PHOSPHORYLATION OF 3'-DEOXY-3'FLUOROTHYMIDINE
        INCORPORATION INTO DNA, EHRLICH ASCITES CELLFREE SYSTEM, 3526*
    PLATELET
        ACUTE LEUKEMIA, CLINICAL STUDY, 3538*

METALS
    NICKEL REFINERY
        RESPIRATORY SYSTEM CANCER, NORWAY, 3053
    ZINC DEFICIENCY
        LYMPHOCYTIC LEUKEMIA, ASCITES TUMOR, MOUSE, 3352

METASTASIS
    CARCINOEMBRYONIC ANTIGEN
        PLASMA LEVEL, CLINICAL STUDY, 3572*
    CARDIAC
        ANITSCHKOW NUCLEAR STRUCTURE, CLINICAL STUDY, 3523*
    CERVICAL CARCINOMA
        CALCIFIED LYMPH NODE, CASE REPORT, 3512*
    LUNG CANCER
        TRITON WR1339 INJECTION, MOUSE, 3396*
    NASOPHARYNGEAL CARCINOMA
        TEMPORAL BONE, CASE REPORT, 3478*
    PROSTATE CARCINOMA
        CASE REPORTS, 3338
        CERVICAL LYMPH NODES, CLINICAL STUDY, 3475*
    PULMONARY CARCINOMA
        PENIS, CASE REPORT, 3431*
    CHEMODECTOMA TO THE LIVER
        CATECHOLAMINE STORAGE, CASE REPORT, 3551*

3'-METHYL-4-DIMETHYLAMINOAZOBENZENE
    HEPATOMA
        ENZYME PATTERN, RAT, 3038
        RAT, 3037

N-METHYL-N'-NITRO-N-NITROSOGUANIDINE
    SUPERNUMERARY LENS FORMATION
        LENTECTOMIZED NEWT, 3098*

N-METHYL-N-NITROSOUREA
    DNA
        METHYLATION, 3060
        PHOSPHOTRIESTER FORMATION, 3048
    RNA, TRANSFER
        ALKYLATION, YEAST, ESCHERICHIA COLI, 3052

METHYL PALMITATE
    RETICULOENDOTHELIAL SYSTEM
        IMMUNE RESPONSE, MOUSE, 3300*

METHYLATION
    DNA
        N-METHYL-N-NITROSOUREA, 3060

7-METHYLBENZ(A)ANTHRACENE
    BINDING TO DNA
        HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS, CELL CULTURE, MOUSE, 3042
    DNA
        HYDROCARBON-DEOXYRIBONUCLEOSIDE PRODUCTS, CELL CULTURE, MOUSE, 3044

METHYLCHOLANTHRANE
    INDUCED TRANSPLANTED SARCOMA
        IMMUNITY, MOUSE, 3239

METHYLCHOLANTHRENE
    SARCOMA
        CIRCADIAN RHYTHM OF CELL DIVISION, MOUSE, 3097*
        CYCLOPHOSPHAMIDE TREATMENT, CIRCADIAN RHYTHM OF CELL DIVISION, 3083*

3-METHYLCHOLANTHRENE
    BENZO(A)PYRENE METABOLISM
        LIVER MICROSOMES, RAT, 3069
    DIEMTHYLNITROSAMINE
        SYNCARCINOGENIC EFFECT, MOUSE, 3078
    DNA BINDING SITE
        FIBROBLAST, HAMSTER, 3073
    LIVER GROWTH
        RNA METABOLISM, RAT, 3075
    SARCOMA
        EFFECT OF RADIATION LEUKEMIA VIRUS, MOUSE, 3072

20-METHYLCHOLANTHRENE
    CERVICAL CARCINOMA
        EFFECT OF ENDOXAN, BLEOMYCIN, MOUSE, 3486*
    EPIDERMAL CYTOSOL PROTEIN
        MICROSOMAL METABOLIZING SYSTEM, MOUSE, 3035

METHYLNITROSOUREA
    NERVOUS SYSTEM, CENTRAL
        TUMOR INDUCTION, RAT, 3084*

5-METHYLRESORCINOL

  EFFECT ON BENZO(A)PYRENE CARCINOGENESIS
    MICE, 3106*

METIAPINE
  NERVOUS SYSTEM, CENTRAL
    TOXICITY STUDY, MOUSE, RAT, DOG, 3102*

MITOCHONDRIA
  CONCANAVALIN A
    COMPARATIVE STUDY, NORMAL AND MALIGNANT CELLS, 3258

MITOMYCIN C
  CHROMOSOME ABERRATIONS
    FANCONI'S ANEMIA PATIENTS, 3059

MORTALITY
  INFANTILE CANCER
    FECUNDITY, UNITED STATES, 3336*

MUTAGENESIS
  BENZ(A)ANTHRACENE
    OXIDATIVE ACTIVATION, DROSOPHILA, 3074
  5'-BROMODEOXYURIDINE
    MURINE SARCOMA VIRUS-TRANSFORMED NONPRODUCER CELL LINES, 3157
  7-BROMOMETHYLBENZ(A)ANTHRACENE
    ESCHERICHIA COLI, 3071
  IRRADIATED FOOD PRODUCTS
    EVALUATION, 3095*

MUTAGENESIS, CHEMICAL
  GENERAL ASPECTS
    REVIEW, 3021*

MYCOPLASMA
  VIRUS, LEUKOSIS
    INFECTION, MOUSE, 3200*

MYCOSTATIN
  INHIBITORY EFFECT
    PLAQUE FORMATION, MAREK'S DISEASE HERPESVIRUS, 3214*

MYELOFIBROSIS
  MYELOID SARCOMA
    CASE REPORT, 3514*
  PLATELET FUNCTIONS
    CLINICAL STUDY, 3378*

MYELOID METAPLASIA
  MYELOID SARCOMA
    CASE REPORT, 3514*

MYELOID SARCOMA
  CASE REPORT, 3514*

MYELOMA
  BONE MARROW CELLS
    ELECTROPHORETIC CELL SEPARATION, HUMAN, 3531*
  DNA POLYMERASE, MULTIPLE
    ISOLATION, CHARACTERIZATION, MOUSE, 3374*
  ISOLATION OF IGE
    SERUM, HUMAN, 3313*
  LYMPHOCYTE CHARACTERISTICS
    CORTISOL, PHYTOHEMAGGLUTININ, MOUSE, 3413*
  PLASMACYTES
    ULTRASTRUCTURE, HUMAN, 3343
  PROTEIN
    L-CHAIN, RECOMBINATION EXPERIMENTS, 3304*
  SPLENIC POOL SIZE
    CLINICAL STUDY, 3570*

MYELOMA, PLASMA-CELL
  B-LYMPHOCYTES
    MALIGNANT TRANSFORMATION, HUMAN, 3574*

MYELOPROLIFERATIVE DISEASE
  BONE MARROW
    MORPHOLOGICAL ANOMALIES, HUMAN, 3521*

MYXOMA
  PRIMARY INTRALUMINAL AORTIC
    VERTEBRAL INVOLVEMENT, CASE REPORT, 3354

NASOPHARYNGEAL CARCINOMA
  ANTIBODIES TO EPSTEIN-BARR VIRUS-RELATED ANTIGENS
    HUMAN, 3248
  COMPLEMENT-FIXING ANTIBODIES
    CLINICAL STUDY, 3230
  MIDDLE EAR EXTENSION
    CASE REPORT, 3478*
  VIRUS, EPSTEIN-BARR
    EPITHELIAL CELLS, 3160

NASOPHARYNGEAL FIBROMA
  OCCURRENCE
    MEXICO, 3489*

NECK
  CANCER
    ALCOHOLICS, SMOKING DIET, CLINICAL STUDY, 3411*
  EPIDERMOID TUMORS
    HYPERCALCEMIA, CLINICAL STUDY, 3503*
  RHABDOMYOSARCOMA
    CHILDREN, 3480*
  SYNOVIAL SARCOMA
    CASE REPORTS, 3454*

NEOPLASMS
  PRIMARY AORTIC
    VERTEBRAL INVOLVEMENT, CASE REPORT, 3354

NEOPLASMS, MALIGNANT
  GESTATIONAL TROPHOBLASTIC DISEASE
    INCIDENCE, REVIEW, 3020*

NEPHROBLASTOMA
  7,12-DIMETHYLBENZ(A)ANTHRACENE
    IMMUNODEPRESSED RAT, 3080*

NERVOUS SYSTEM, CENTRAL
  BRAIN TUMOR
    MENINGITIS, CASE REPORT, 3463*

- BRAIN, TUMOR-LIKE LESION
    - TRYPANOSOMA CRUZI, CASE REPORT, 3562*
- CAROTID ARTERY BIFURCATION TUMORS
    - ANGIOGRAPHIC DIFFERENTIATION, HUMAN, 3500*
- CEREBRAL VASCULAR NEOPLASMS
    - IMPAIRED REGIONAL CIRCULATION, HUMAN, 3546*
- CHEMICAL CARCINOGENESIS
    - REVIEW, 3032*
- ETHYLNITROSOUREA
    - TUMOR INDUCTION, RAT, 3084*
- METHYLNITROSOUREA
    - TUMOR INDUCTION, RAT, 3084*
- METIAPINE
    - TOXICITY STUDY, MOUSE, RAT, DOG, 3102*
- MYXOPAPILLARY EPENDYMOMA OF THE FILUM TERMINALE
    - ULTRASTRUCTURE, 3589*
- NEUROBLASTOMA
    - NERVOUS-SYSTEM SPECIFIC PROTEIN, HUMAN, 3532*
- SPINAL CORD
    - MALIGNANT EXTRADURAL TUMORS, CLINICAL STUDY, 3483*
- TUMORS
    - CLINICAL STUDY, ZAMBIA, 3367*
    - OCCURRENCE, IVORY COAST, 3381*

NERVOUS SYSTEM TUMORS
- INCIDENCE
    - CHILDREN, CHILE, 3331

NEURILEMMOMA
- CAROTID ARTERY BIFURCATION TUMORS
    - ANGIOGRAPHIC DIFFERENTIATION, HUMAN, 3500*

NEUROBLASTOMA
- CATECHOLAMINES
    - CLINICAL STUDY, 3554*
- CYCLIC AMP
    - MORPHOLOGIC DIFFERENTIATION, MOUSE, 3426*
- CYCLIC AMP STIMULATION BY PROSTAGLANDIN E1
    - PHOSPHODIESTERASE INHIBITORS, MOUSE, 3470*
- DIFFERENTIATION
    - INDUCTION BY NONCYCLIC AMP AGENTS, MOUSE, 3345
- NERVOUS-SYSTEM SPECIFIC PROTEIN
    - HUMAN, 3532*
- SOMATIC CELL HYBRIDS
    - ELECTRICAL EXCITABILITY, CHEMOSENSITIVITY, MOUSE, HUMAN, 3370*

NICKEL
- REFINERY WORKERS
    - RESPIRATORY SYSTEM CANCER, NORWAY, 3053

NICKEL SUBSULFIDE
- INTRATRACHEAL INJECTION
    - PATHOLOGICAL REACTION, RAT, 3104*

4-NITROBENZENE
- CARCINOGENESIS
    - RAT, 3066

5-NITROFURANS
- CARCINOGENESIS
    - RAT, 3066

NITROGEN MUSTARD
- CHROMOSOME ABERRATIONS
    - FANCONI'S ANEMIA PATIENTS, 3059

5-NITROIMIDAZOLES
- CARCINOGENESIS
    - RAT, 3066

4-NITROQUINOLINE-1-OXIDE
- CARCINOGENICITY
    - TEST METHOD, MUTANT YEAST, 3041

NITROSAMIDE
- HYDROCEPHALI INDUCTION
    - PREVENTION BY ASCORBIC ACID, RAT, 3086*

NITROSAMINES
- ACCUMULATION
    - NITRATE, SECONDARY AMINE FERTILIZATION, WHEA, 3046
- FORMATION
    - MEAT CURING MIXTURE, 3064
- N-NITROSOPYRROLIDINE
    - BACON, OTHER FRIED FOODS, 3062

N-NITROSOPYRROLIDINE
- POTENTIAL PRECURSORS
    - BACON, OTHER FRIED FOODS, 3062

NOVIKOFF ACITES TUMOR
- GUANOSINE ANABOLISM
    - NUCLEIC ACID BIOSYNTHESIS, RAT, 3357

NUCLEIC ACID
- HUMAN LEUKEMIC OR NEOPLASTIC TISSUES
    - EFFECT ON VARIOUS MOUSE STRAINS, 3223*
- METABOLISM
    - LYMPHOMA, HUMAN, 3417*

5'-NUCLEOTIDE PHOSPHODIESTERASE
- HEPATOMA
    - ISOENZYME PATTERN, HUMAN, 3353

12-O-TETRADECANOYL-PHORBOL-13-ACETATE
- PHOSPHOLIPID METABOLISM
    - TUMOR PROMOTION, MOUSE SKIN, 3056

OAT CELL CARCINOMA
- TRACHEA
    - CASE REPORT, 3540*

OCCUPATIONAL HAZARD
- CARCINOGENS
    - INDUSTRIAL HEALTH STANDARDS, U.S. DEPARTMENT OF LABOR, 3460*
- LUNG CANCER
    - URANIUM MINERS, UNITED STATES, 3334

- RESPIRATORY SYSTEM CANCER
  - NORWAY NICKEL REFINERY, 3053

ODONTOGENIC TUMOR
- ADENOMATOID
  - CASE REPORT, 3450*

ORAL CAVITY
- CAVERNOUS HEMANGIOMA
  - REVIEW, CASE REPORT, 3464*
- MUCOSAL NEUROMA
  - MEDULLARY THYROID CARCINOMA, CASE REPORT, 3452*
- SQUAMOUS CELL CARCINOMA
  - CLINICAL STUDY, 3443*

OSTEOSARCOMA
- RADIATION
  - SKELETAL LOCATION, MAN AND DOG, 3113
- SERUM ALKALINE PHOSPHATASE
  - HUMAN, 3359

OVARIAN CARCINOMA
- BRENNER TUMOR, MALIGNANT
  - CASE REPORT, 3373*
- CHROMOSOME ABERRATIONS
  - HUMAN, 3425*
- CLEAR CELL
  - PERITONEAL CAVITY TRANSPLANT, HUMAN, MOUSE, 3448*
- CYTOLOGIC DETECTION
  - CLINICAL-PATHOLOGIC APPROACHES, 3437*
- DIAGNOSIS, BIOCHEMICAL
  - HUMAN, 3473*
- MUCINOUS CYSTADENOCARCINOMA
  - CLINICAL STUDY, 3412*
- SKIN LESIONS
  - CASE REPORTS, 3441*
- STRUCTURAL AND FUNCTIONAL CONDITIONS
  - CLINICAL STUDY, 3326*

OVARY
- ANDROBLASTOMA
  - HETEROTOPIC ELEMENTS, CASE HISTORY, 3481*
- ARRHENOBLASTOMA
  - ANDROGENS, CLINICAL STUDY, 3421*
- ENDODERMAL SINUS TUMOR
  - ALPHA-FETOPROTEIN, CASE REPORT, 3520*
- SECRETORY TUMORS
  - HISTOENZYMOLOGY, HUMAN, 3563*
- TUMORIGENESIS
  - GROWTH RATE, RAT, 3317

OXYPHIL ADENOMA
- LARYNX
  - CASE REPORTS, 3457*

OZONE
- SKIN CANCER
  - SUPERSONIC TRANSPORT, REVIEW, 3002

1-(P-CARBOXYETHYLPHENYL)-3,3-DIMETHYLTRIAZENE
- CARCINOGENIC ACTION
  - RAT, 3101*

PANCREAS
- CANCER
  - EPIDEMIOLOGY, INTERNATIONAL, 3598*
- PRIMARY CARCINOMA
  - JAPAN, CHILDREN, CASE REPORTS, 3594*

PAPILLARY CARCINOMA
- URINARY BLADDER
  - CLINICAL STUDY, 3325*

PAPILLOMA
- LARYNX
  - PAPOVA VIRUS, ELECTRON MICROSCOPIC IDENTIFICATION, 3142
- SPONTANEOUS EPIDERMAL
  - NEWT, 3599*

PAPILLOMA, BENIGN
- URINARY BLADDER
  - CLINICAL STUDY, 3325*

PARANASAL SINUSES
- FIBROSARCOMA
  - CASE REPORT, 3385*
- HISTIOCYTOMA, FIBROUS
  - CASE REPORT, 3468*

PARAPROTEIN
- IMMUNE PROLIFERATION
  - IMMUNOLOGY, REVIEW, 3029*

PARATHYROID GLAND
- CARCINOMA
  - MORPHOLOGY, ULTRASTRUCTURE, CASE REPORTS, 3342
- CARCINOMAS, HORMONE-SECRETING
  - ULTRASTRUCTURE, HUMAN, 3442*

PENIS
- SCLEROSING LIPOGRANULOMA
  - SILICONE-INDUCED, HUMAN, 3082*
- SECONDARY BRONCHOGENIC CARCINOMA
  - CASE REPORT, 3431*

PHENOL
- EFFECT ON BENZO(A)PYRENE CARCINOGENESIS
  - MICE, 3106*

PHEOCHROMOCYTOMA
- CATECHOLAMINES
  - CLINICAL STUDY, 3554*
- TRANSPLANTABLE
  - ULTRASTRUCTURE, GROWTH, RAT, 3515*

PHOSPHODIESTERASE
- INHIBITORS
  - PROSTAGLANDIN E1 STIMULATION OF CYCLIC AMP, NEUROBLASTOMA, MOUS, 3470*

PHOSPHOLIPID
- UPTAKE
  - EXPERIMENTAL TUMORS, MOUSE, 3340

PHYTOHEMAGGLUTININ
- STANDARDIZATION
  - LYMPHOCYTE TRANSFORMATION, 3296*

PHYTOHEMAGGLUTINNIN R
- EMBRYOGENESIS
  - CHICKEN, 3288*

PITUITARY GLAND
- CARCINOMA
  - CASE REPORT, 3527*
- PARANEOPLASTIC HYPERCORTICISM
  - ULTRASTRUCTURE, CASE REPORT, 3568*
- TUMOR, HORMONE-SECRETING
  - CYCLIC AMP, RAT, 3395*
- TUMORS
  - CLINICAL STUDY, IBADAN, NIGERIA, 3383*
  - OCCURRENCE, KAMPALA, UGANDA, 3382*

PITUITARY TUMOR
- LYMPHOMA
  - VIRUS MORPHOGENESIS, MOUSE, 3167

PLASMA CELL TUMOR
- IMMUNITY
  - MOUSE, 3253

PLASMA MEMBRANE
- PROTEIN
  - VIRUS-TRANSFORMED CHICK CELLS, 3137

PLASMACYTOMA
- CELLULAR IMMUNITY
  - SERUM FACTORS, MOUSE, 3279*
- COLONY FORMATION
  - POTENTIATION BY HEMOPOIETIC CELLS, SERUM, MOUSE, 3547*

PLASTICS
- TUMORIGENESIS
  - MOUSE INOCULATED WITH HUMAN NEOPLASTIC CELLS, 3061

POLYADENYLIC ACID
- DNA TRANSCRIPTION
  - VIRUS, SV40, 3126

POLYCYTHEMIA
- PLATELET FUNCTIONS
  - CLINICAL STUDY, 3378*

POLYDEOXYADENYLIC ACID
- RADIATION, UV
  - DNA PHOTOLESIONS, REPAIR, 3361

POLYINOSINIC: POLYCYTIDYLIC ACID
- TUMOR GROWTH ENHANCEMENT
  - MOLONEY SARCOMA VIRUS-INDUCED TUMORS, MOUSE, RAT, 3155

POXVIRUS
- BIOGENESIS
  - INACTIVATION OF HOST DNA POLYMERASE, 3189*

PRELEUKEMIA
- MYELODYSPLASTIC SYNDROME
  - CASE REPORTS, 3323*

PRIMARY SCAR CARCINOMA
- LUNG CANCER
  - HUMAN, 3319

PROGESTERONE
- MAMMARY CARCINOMA
  - 7,12-DIMETHYLBENZ(A)ANTHRACE INDUCED, RAT, 3039
- MAMMARY TUMOR
  - DNA SYNTHESIS, HUMAN, 3356

PROSTAGLANDIN E1
- CYCLIC AMP AND ADENYLATE CYCLASE ACTIVITY
  - MALIGNANT HEPATOMA, ETHIONINE-TREATED RATS, 3055
- CYCLIC AMP STIMULATION
  - PHOSPHODIESTERASE INHIBITORS, NEUROBLASTOMA, MOUSE, 3470*

PROSTATE
- BENIGN HYPERPLASIA
  - MORPHOLOGICAL CHANGES, ORGAN CULTURE, 3397*
- CARCINOMA
  - BONE MARROW SERUM PHOSPHATASE, CLINICAL STUDY, 3494*
  - METASTASIS TO CERVICAL LYMPH NODES, CLINICAL STUDY, 3475*
  - RECTAL MUCOSAL INVOLVEMENT, CASE REPORTS, 3338

PROTEIN
- BRAIN TUMOR
  - IMMUNOLOGICAL STUDY, HUMAN, 3274
- DEFICIENCY, EXCESS
  - TUMORIGENESIS, RAT, 3466*
- METABOLISM
  - FIBROMYOMA, UTERINE TISSUE, HUMAN, 3405*
- MYELOMA
  - L-CHAIN, RECOMBINATION EXPERIMENTS, 3304*
- PARTICIPATION OF EARLY PROTEINS
  - ADENOVIRUS 2 DNA TRANSCRIPTION, 3182*
- PLASMA MEMBRANE
  - VIRUS-TRANSFORMED CHICK CELLS, 3137
- VIRUS, SV40
  - PHOSPHORYLATION, CELL-FREE SYSTEM, 3170*

PROTEIN, CAPSID
- SV40 PLAQUE MUTANTS
  - ELECTROPHORETIC DIFFERENCES, 3181*

PROTEIN SYNTHESIS
- ACETYLCHOLINE
  - LIMB REGENERATION, AMPHIBIAN, 3398*
- HELA CELL MITOCHONDRIA
  - ATYPICAL AMINO ACID UTILIZATION, 3593*
- HERPESVIRUS-INFECTED CELLS
  - MOUSE, RAT, 3176*

PULMONARY CARCINOMA
- INCIDENCE AND THERAPY
  - GEISINGER MEDICAL CENTER, 3449*
- METASTASIS

    - PENIS. CASE REPORT. 3431*

PURINE PHOSPHORIBOSYL TRANSFERASE
- LEUKEMIA. 6-THIOPURINE SENSITIVE. RESISTANT
    - HUMAN. 3502*

RADIATION
- CYTOTOXICITY
    - FOOD PRODUCTS. 3095*
- MELANOMA
    - MOUSE. 3116*
- MUTAGENESIS
    - FOOD PRODUCTS. 3095*
- OSTEOSARCOMA
    - SKELETAL LOCATION. MAN AND DOG. 3113
- SKIN CANCER
    - MOUSE. 3116*
- STRONTIUM 90
    - THYMUS. MOUSE. 3111

RADIATION. UV
- BASAL CELL CARCINOMA
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- BENZO(A)PYRENE COOXIDATION
    - PHENOLS. KINETICS. 3058
- CARCINOGENESIS
    - REVIEW. 3010
- CHROMOSOME ABERRATIONS
    - FANCONI'S ANEMIA PATIENTS. 3059
- MALIGNANT MELANOMA
    - ETIOLOGY. REVIEW. 3025*
- MELANOMA. MALIGNANT
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- POLYDEOXYADENYLIC ACID
    - DNA PHOTOLESIONS. REPAIR. 3361
- SKIN CANCER
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- SKIN TUMOR
    - 7,12-DIMETHYLBENZ(A)ANTHRACENE-INDUCED. RAT. 3050
    - REVIEW. 3010
- SQUAMOUS CELL CARCINOMA
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- VIRUS. ADENO 2
    - DNA DAMAGE. BIOLOGICAL FUNCTION. 3210*

RADIATION. X-RAY
- BONE MARROW
    - CYTOGENETIC EFFECT. MOUSE. 3112
- LEUKEMIA
    - SYNERGISTIC EFFECT OF URETHANE. RAT. 3110
- THYROID CARCINOMA
    - DOG. 3109

226 RADIUM
- COLONY FORMING UNITS
    - BONE MARROW. SPLENECTOMIZED MICE. 3114*

RECTAL CARCINOMA
- INVOLVEMENT IN PROSTATE CARCINOMA
    - CASE REPORTS. 3338

REGENERATION
- LIVER
    - HEMATOPOIETIC STEM CELLS. MOUSE. 3544*

REGENERATION. LIMB
- PROTEIN SYNTHESIS
    - ACETYLCHOLINE. AMPHIBIAN. 3398*

REGRESSION. SPONTANEOUS
- LEUKEMIA
    - AVIAN MYELOBLASTOSIS VIRUS-INFECTED CHICKEN. 3159

RENAL CARCINOMA
- PREGNANCY
    - CASE REPORTS. 3433*

RESISTANCE
- EFFECT OF LIPOPOLYSACCHARIDE
    - EHRLICH TUMOR CELLS. MOUSE. 3307*
- TRANSMISSION
    - TRANSPLANTED TUMORS. RAT. 3245

RESORCINOL
- CARCINOGENESIS
    - RAT. 3036

RESPIRATORY SYSTEM
- CANCER
    - NICKEL REFINERY WORKERS. NORWAY. 3053
- CANCER INCIDENCE
    - URANIUM MINERS. UNITED STATES. 3334
- CARCINOGENESIS
    - DUST. DIETHYLNITROSAMINE. HAMSTER. 3077
- OAT CELL CARCINOMA. TRACHEA
    - CASE REPORT. 3540*

RETICULOENDOTHELIAL SYSTEM
- ANERYTHREMIC. ALEUKEMIC DI GUGLIELMO SYNDROME
    - CASE REPORT. 3499*
- IMMUNE RESPONSE
    - ETHYL STEARATE. METHYL PALMITATE. MOUSE. 3300*
- MALIGNANT HISTIOCYTOSIS. ROBB-SMITH TYPE
    - CASE REPORT. 3430*

RETICULOPROLIFERATIVE DISEASE
- VIRUS. EPSTEIN-BARR
    - OWL MONKEY. 3150. 3153

RETICULOSARCOMA
- BLOOD COAGULATION
    - CLINICAL STUDY. CHILDREN. 3410*

RETICULOSIS
- HISTIOCYTIC MEDULLARY
    - CASE REPORT. 3430*

RETINOPATHY
- SV40-INDUCED
    - RAT. 3211*

REVERSE TRANSCRIPTASE
- DNA POLYMERASE
    - RNA PROGRAMMED. REVIEW. 3015*

- HUMAN ACUTE MYELOBLASTIC LEUKEMIA CELLS
    - COMPARISON WITH PRIMATE AND MOUSE C-TYPE VIRUSES. 3246
- LEUKEMIA
    - PLASMA. MOUSE. CHICKEN. 3169
- VIRUS. ROUS SARCOMA
    - N-ETHYLENEIMIDE. TRANSFORMING ACTIVITIES. 3127

RHABDOMYOMA
- VAGINA
    - BENIGN. POLYPLOID. ULTRASTRUCTURE. CASE REPORT. 3553*

RHABDOMYOSARCOMA
- EMBRYONAL. HEAD. NECK
    - CHILDREN. 3480*
- FELINE LEUKEMIA VIRUS INFECTION
    - MORPHOLOGY. GROWTH CHARACTERISTICS. HUMAN CELLS. 3136
- MURINE SARCOMA VIRUS INFECTION
    - MORPHOLOGY. GROWTH CHARACTERISTICS. HUMAN CELLS. 3136
- RD-114 VIRUS
    - CELL-SURFACE ANTIGENS. HUMAN. 3267
- RD-114 VIRUS INFECTION
    - MORPHOLOGY. GROWTH CHARACTERISTICS. HUMAN CELLS. 3136
- TEMPORAL BONE
    - CASE REPORT. 3476*
    - CASE REPORTS. 3467*

RNA
- 7-BROMOMETHYLBENZ(A)ANTHRACENE
    - ESCHERICHIA COLI. 3071
- DEGRADATION
    - EHRLICH ASCITES TUMOR CELLS. 3422*
- DNA POLYMERASE
    - REVIEW. 3015*
- ISOLATION
    - LEUKEMIC CELLS. HUMAN. 3349
- LUCKE TUMOR HERPESVIRUS-SPECIFIC
    - FROG. 3124
- METABOLISM
    - LIVER. 3-METHYLCHOLANTHRENE EFFECT. RAT. 3075
- REVERSE TRANSCRIPTASE
    - REVIEW. 3015*
- "8S" COMPONENTS
    - ULTRASTRUCTURE. MOLONEY SARCOMA VIRUS. MOUSE. 3204*
- VIRUS. SV40
    - CHARACTERIZATION. INFECTED BS-C-1 CELLS. 3198*
- VIRUS. WOUND TUMOR
    - ELECTROPHORETIC SEPARATION OF ALL COMPONENTS. 3209*

RNA. MESSENGER
- VIRUS. ADENO
    - ATP REQUIREMENT FOR RELEASE FROM HUMAN CELL NUCLEI. 3133

RNA METHYLASE
- TRANSFER RNA-METHYLATED BASES
    - POLYOMA VIRUS-TRANSFORMED MOUSE CULTURE CELLS. 3203*

RNA POLYMERASE
- DNA DEPENDENT
    - EHRLICH ASCITES TUMOR. MOUSE. 3552*
- RNASE H AND A EFFECT
    - DNA TEMPLATE SPECIFICITY. FRIEND VIRUS-INFECTED SPLEEN. MOUSE. 3132

RNA SYNTHESIS
- EHRLICH ASCITES TUMOR
    - INHIBITION. HEAT TREATMENT. MOUSE. RAT. 3402*
- HERPES SIMPLEX VIRUS INFECTION
    - HUMAN EPIDERMOID CELLS. 3180*
- LEUKEMIA
    - INTERFERON EFFECT. MOUSE. 3591*
- YOSHIDA ASCITES TUMOR
    - INHIBITION. HEAT TREATMENT. MOUSE. RAT. 3402*

RNA TRANSCRIPTION
- LUCKE TUMOR HERPESVIRUS
    - VIRUS-FREE ADENOCARCINOMA. FROG. 3124

RNA. TRANSFER
- N-ETHYL-N-NITROSOUREA
    - ALKYLATION. YEAST. ESCHERICHIA COLI. 3052
- N-METHYL-N-NITROSOUREA
    - ALKYLATION. YEAST. ESCHERICHIA COLI. 3052

RUTHENIUM RED
- STAINING
    - TRANSFORMED CELL. MOUSE. 3242

SALIVARY GLAND
- ADENOLYMPHOMA
    - CASE REPORT. 3539*
- 9,10-DIMETHYL-1,2-BENZANTHRACENE-INDUCED TUMOR
    - EFFECT OF ZINC. RAT. 3054

SARCOMA
- HOMOGENATES AND NUCLEIC ACID EXTRACTS
    - COMPARATIVE PATHOGENICITY STUDIES. MOUSE. 3120
- ISOENZYME PATTERN
    - RAT. 3424*
- METHYLCHOLANTHRENE
    - CIRCADIAN RHYTHM OF CELL DIVISION. MOUSE. 3097*
    - CYCLOPHOSPHAMIDE TREATMENT. CIRCADIAN RHYTHM OF CELL DIVISION. 3083*
- 3-METHYLCHOLANTHRENE
    - EFFECT OF RADIATION LEUKEMIA VIRUS. MOUSE. 3072
- SYNOVIAL
    - SULFATED MUCOPOLYSACCHARIDE PRODUCTION. HUMAN. 3403*
- UTERINE CERVIX
    - CLINICAL STUDY. 3408*

SCHIZOGENY
- AMNION CELLS
    - HUMAN. 3337

- SV40 VIRUS-TRANSFORMED CELLS
    - MOUSE. 3337

SEROTONIN
- HORMONE. ADRENOCORTICOTROPHIC
    - OAT-CELL CARCINOMA. LUNG. CLINICAL STUDY. 3455*

SERUM
- COPPER
    - HODGKIN'S DISEASE. CLINICAL STUDY. 3556*
- ERYTHROCYTIC. LEUKOCYTIC. SERUM ALLOTYPES
    - ALTERED EXPRESSION. MALIGNANT DISEASES. 3284*
- LACTATE DEHYDROGENASE ISOENZYME CHANGES
    - CANCER PATIENTS. 3471*
- LYMPHOCYTE-MEDIATED TUMOR-CELL DESTRUCTION
    - CANCER PATIENTS. 3232
- RNA-RICH FRACTION INCUBATION
    - ELECTROPHORETIC ANALYSIS. CANCER PATIENTS. 3444*

SICKLE-CELL ANEMIA
- IMMUNODEFICIENCY
    - GHANA. 3322*

SILICA GEL
- PF 254 ELUATE
    - ANTITUMOR ACTIVITY. MOUSE. 3190*

SILICONE
- PENILE SCLEROSING LIPOGRANULOMA
    - HUMAN. 3082*

SINUS HISTIOCYTOSIS
- CANCER DETECTION
    - CLINICAL STUDY. 3495*
- LYMPHADENOPATHY
    - ELEVATED EBV ANTIBODY TITERS. CASE REPORT. 3225*

SKIN
- ATYPICAL FIBROXANTHOMA
    - CLINICAL. HISTOLOGIC. HISTOCHEMICAL STUDIES. 3428*
- DELAYED CUTANEOUS HYPERSENSITIVITY REACTIONS
    - MALIGNANT AND NORMAL HUMAN BREAST CELLS. 3261
- LESIONS
    - DIAGNOSIS OF UTERINE. OVARIAN CARCINOMA. CASE REPORTS. 3441*
- LYMPHOMA CUTIS
    - EXPERIMENTAL MOUSE. HUMAN DISEASE COMPARISON. 3550*

SKIN CANCER
- BENZO(A)PYRENE
    - NUCLEAR DNA SYNTHESIS. CELL PROLIFERATION. MOUSE. 3087*
- OZONE
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- RADIATION
    - MOUSE. 3116*
- RADIATION. UV
    - SUPERSONIC TRANSPORT. REVIEW. 3002

SKIN TUMOR
- 7-12-DIMETHYLBENZ(A)ANTHRACENE
    - MOUSE. 3056
    - UV ENHANCEMENT. INHIBITION. MOUSE. 3050
- PIGMENTED
    - CLINICAL STUDY. AUSTRALIA. 3560*
- RADIATION. UV
    - REVIEW. 3010

SKULL
- CRANIUM BIFIDUM OCCULTA
    - JUVENILE FIBROMATOSIS OF FACE. CASE REPORT. 3456*
- PRIMARY SARCOMA. TEMPORAL BONE
    - CASE REPORTS. 3467*

SPINAL CANAL
- MALIGNANT EXTRADURAL TUMORS
    - CLINICAL STUDY. 3483*

SPLEEN
- ANTIBODY-PRODUCING CELLS
    - CHANGES DURING CARCINOGENESIS. MOUSE. 3272
- COLONY-FORMING UNITS
    - LIVER REGENERATION. MOUSE. 3544*
- POOL SIZE
    - MYELO- AND LYMPHO- PROLIFERATIVE SYNDROMES. CLINICAL STUDY. 3570*
- T- AND B-CELL SUBPOPULATIONS
    - FIBROSARCOMA. MOUSE. 3256

SQUAMOUS CELL CARCINOMA
- ENDOMETRIUM
    - CASE REPORTS. 3377*
- GENETIC DIATHESIS
    - CLINICAL STUDY. 3509*
- ORAL CAVITY
    - CLINICAL STUDY. 3443*
- RADIATION. UV
    - SUPERSONIC TRANSPORT. REVIEW. 3002
- UTERUS
    - ULTRASTRUCTURE. HUMAN. 3569*

STEROIDS
- GLUCOCORTICO-
    - MONOCYTE PROLIFERATION. BONE MARROW. HUMAN. 3504*

STREPTONIGRIN
- INHIBITORY EFFECT
    - MURINE SARCOMA VIRUS-INDUCED TUMOR CELL LINE. 3208*

STRONTIUM 90
- RADIATION
    - THYMUS. MOUSE. 3111

SUNLIGHT
- MALIGNANT MELANOMA
    - ETIOLOGY. REVIEW. 3025*

SYNOVIAL SARCOMA
- HEAD. NECK
    - CASE REPORTS. 3454*
- SULFATED MUCOPOLYSACCHARIDE PRODUCTION

- HUMAN. 3403*

TEMPORAL BONE
- BILATERAL GLOMUS TUMOR
  - CASE REPORT. 3474*
- NASOPHARYNGEAL CARCINOMA METASTASIS
  - CASE REPORT. 3478*
- PRIMARY SARCOMA
  - CASE REPORTS. 3467*
- RHABDOMYOSARCOMA
  - CASE REPORT. 3476*

TERATOMA
- MALIGNANT
  - DIFFERENTIATION RESTRICTED TO NEURO-ECTODERMAL TISSUE. MOUSE. 3511*
- Y BODIES
  - FLUORESCENT CHROMOCENTERS. HUMAN. 3518*

TESTIS
- CARCINOMA OF MULLERIAN VESTIGAL ORIGIN
  - CASE REPORT. 3479*

TESTOSTERONE
- MAMMARY CARCINOMA
  - ATYPICAL ENDOMETRIAL PATTERN. CLINICAL STUDY. 3420*
- MAMMARY TUMOR
  - DNA SYNTHESIS. HUMAN. 3356

TESTOSTERONE PROPIONATE
- CELL PROLIFERATION
  - SEMINAL VESICLE. CASTRATE MOUSE. 3491*

THOROTRAST
- TUMOR DEVELOPMENT
  - CASE REPORTS. REVIEW. 3019*

THROMBOCYTHEMIA
- PLATELET FUNCTIONS
  - CLINICAL STUDY. 3378*

THYMUS
- STRONTIUM. 90 RADIATION
  - REGENERATION. MOUSE. 3111

THYROGLOBULIN
- THYROID TUMOR
  - IMMUNOMORPHOLOGICAL INVESTIGATION. RAT. 3303*

THYROID
- FUNCTION. REGRESSION OF EXPERIMENTAL TUMORS
  - HYPOTHALAMUS STIMULATION. RAT. 3103*

THYROID CARCINOMA
- RADIATION. X-RAY
  - DOG. 3109

THYROID GLAND
- MEDULLARY CARCINOMA
  - DIARRHEA. CASE REPORT. 3596*
  - ORAL MUCOSAL NEUROMA. CASE REPORT. 3452*

- TRANSPLANTABLE TUMORS
  - IMMUNOMORPHOLOGY. RAT. 3364*

THYROID TUMOR
- THYROGLOBULIN
  - IMMUNOMORPHOLOGICAL INVESTIGATION. RAT. 3303*

TOBACCO SMOKING
- HARD PALATE CARCINOMA
  - INCIDENCE. REVERSE SMOKERS. 3335
- HEAD AND NECK CANCER
  - ALCOHOLICS. CLINICAL STUDY. 3411*

P-TOLUENEDIAMINE
- CARCINOGENESIS
  - RAT. 3036

TRACHEA
- OAT CELL CARCINOMA
  - CASE REPORT. 3540*

TRANSFORMATION
- ADENOVIRUS 5 DNA
  - RAT CELLS. 3147
- CELL-SURFACE
  - DETECTION OF ALTERATIONS BY LECTINS. 3265
- LYMPHOCYTE
  - STANDARDIZATION TO PHYTOHEMAGGLUTININ. 3296*
- LYMPHOCYTE. CONCANAVALIN A-INDUCED
  - S-ADENOSYL-L-METHIONINE DECARBOXYLASE. 3557*
- PHOTODYNAMIC INACTIVATED SV40
  - HAMSTER CELLS. 3164
- VIRUS
  - SERUM GROWTH FACTORS. MOUSE. 3140
- VIRUS. ADENO
  - CLONAL VARIABILITY OF CELLS. 3148
- VIRUS. ADENO 12
  - SPECIFIC SURFACE ANTIGEN DETECTION. MOUSE. HAMSTER. 3276
- VIRUS. EPSTEIN-BARR
  - LYMPHOCYTES. HUMAN. 3162
- VIRUS. ERYTHROBLASTOSIS
  - GLYCOPROTEIN ALTERATIONS. CHICKEN ERYTHROCYTES. 3202*
- VIRUS. MURINE SARCOMA
  - 5'-BROMODEOXYURIDINE. MUTAGENESIS. NONPRODUCER CELL LINES. 3157
- VIRUS. POLYOMA
  - RNA METHYLASES. TRNA-METHYLATED BASES. MOUSE CULTURE CELLS. 3203*
- VIRUS. RAUSCHER LEUKEMIA
  - ANTISCHISTOSOMAL DRUGS. RAT EMBRYO CELLS. 3149
- VIRUS. ROUS SARCOMA
  - N-ETHYLENEIMIDE. 3127
  - GROWTH. ANTIGENIC CHARACTERISTICS. HAMSTER CELLS. 3156
- VIRUS. SV40
  - CELL SURFACE PROPERTIES. 3242
  - FETAL ANTIGENS. MOUSE. 3273
  - IMMUNOGENICITY. IMMUNOSENSITIVITY. MOUSE CELLS. 3251

TRANSFORMATION. MALIGNANT

- CARCINOGEN, CHEMICAL
  - QUANTITATIVE SYSTEM FOR ASSAY, MOUSE, 3065
- 9,10-DIMETHYL-1,2-BENZANTHRACENE
  - RELATION TO CYTOTOXICITY, CELLS, HAMSTER, 3070
- VIRUS
  - MODEL, GENETIC IMBALANCE, REVIEW, 3008

TRANSPLACENTAL CARCINOGENESIS
- CHOLESTERYL-14-METHYLHEXADECANOATE
  - MOUSE, 3043

TRANSPLANTATION
- ANTIGEN
  - MURINE TUMOR, 3239
- HEPATOMA
  - CELL PROLIFERATION, HOST TISSUE, MOUSE, 3432*

TRIETHYLENEIMINOBENZOQUINONE
- DNA SYNTHESIS
  - EHRLICH ASCITES TUMOR CELLS, 3049
- LYSINE INCORPORATION, NUCLEAR PROTEIN
  - EHRLICH ASCITES TUMOR CELLS, 3049

TRIPEPTIDE
- SYNTHETIC
  - HEPATOMA STIMULATION, HUMAN, 3339

TUMOR
- DIAGNOSIS
  - SEROLOGICAL INVESTIGATIONS WITH CLOSTRIDIAL SPORES, RAT, HAMSTE, 3301*
- TISSUE CULTURE TECHNIQUE
  - MAINTAINING CHARACTERISTICS, HUMAN, 3436*
- Y BODIES
  - FLUORESCENT CHROMOCENTERS, HUMAN, 3518*

TUMOR VIRUS
- SEE VIRUS

TUMORIGENESIS
- ELEVATION MECHANISM OF AGING
  - REVIEW, 3028*
- PLASTICS
  - MOUSE INOCULATED WITH HUMAN NEOPLASTIC CELLS, 3061
- PROTEIN DEFICIENCY, EXCESS
  - RAT, 3466*
- VERTEBRATE AND INVERTEBRATE
  - COMPARITIVE PATHOLOGY, REVIEW, 3033*

URETHANE
- RADIATION-INDUCED LEUKEMIA
  - SYNERGISTIC EFFECT, RAT, 3110

URINARY BLADDER
- CARCINOGENESIS
  - BANANA DIET, RAT, GUINEA PIG, 3057
- PAPILLARY CARCINOMA
  - CLINICAL STUDY, 3325*
- PAPILLOMA, BENIGN
  - CLINICAL STUDY, 3325*

UTERINE CERVIX
- CARCINOSARCOMA
  - CLINICAL STUDY, 3408*
- SARCOMA
  - CLINICAL STUDY, 3408*

UTERUS
- CARCINOMA DIAGNOSIS
  - SKIN LESIONS, CASE REPORTS, 3441*
- ENDOMETRIAL CARCINOMA
  - PEMPHIGUS VULGARIS OF CERVIX, CASE REPORT, 3316*
- FIBROMYOMA
  - PROTEIN METABOLISM, HUMAN, 3405*
- MESONEPHRIC ADENOCARCINOMA
  - CASE REPORT, 3513*
- PRIMARY NEOPLASMS
  - CLINICAL STUDY, 3525*
- SQUAMOUS CELL CARCINOMA
  - ULTRASTRUCTURE, HUMAN, 3569*

VAGINA
- ADENOCARCINOMA, CLEAR CELL
  - DIETHYLSTILBESTROL, CASE REPORTS, 3488*
- CLEAR CELL CARCINOMA
  - MATERNAL STILBESTROL THERAPY, VAGINAL ADENOSIS, CASE REPORTS, 3524*
- RHABDOMYOMA
  - BENIGN, POLYPLOID, ULTRASTRUCTURE, CASE REPORT, 3553*

VIRUS
- ADENOVIRUS 2-SV40 HYBRID
  - CELLULAR DNA SYNTHESIS INDUCTION, 3185*
- CARCINOGENESIS
  - HUMAN, REVIEW, 3018*
- LEUKEMIA
  - REVIEW, 3017*
- MORPHOGENESIS
  - LYMPHOMA, MOUSE, 3167
- ONCOGENESIS
  - BIOLOGIC CONCEPTS, REVIEW, 3014*
- TRANSFORMATION
  - SERUM GROWTH FACTORS, MOUSE, 3140
- TRANSFORMATION, MALIGNANT
  - MODEL, GENETIC IMBALANCE, REVIEW, 3008
- VIRAL PATHOLOGY
  - REVIEW, 3013*

VIRUS, ABELSON
- ACTIVITY
  - ANTI-RAUSCHER VIRUS SERUM NEUTRALIZED MICE AND RATS, 3226*

VIRUS, ABELSON LEUKEMIA
- LYMPH NODE CELLS RESPONSE
  - ULTRASTRUCTURE, MOUSE, 3151

VIRUS, ADENO
- DNA
  - GENOME, ELECTRON MICROSCOPY, 3143
  - HYDROXYAPATITE CHROMATOGRAPHY, FORMAMIDE DENATURATION, 3199*
- RNA, MESSENGER
  - ATP REQUIREMENT FOR RELEASE FROM

    - HUMAN CELL NUCLEI, 3133
  - TRANSFORMATION
    - CLONAL VARIABILITY OF CELLS, 3148

- VIRUS, ADENO 2
  - DNA TRANSCRIPTION
    - PARTICIPATION OF EARLY PROTEINS, 3182*
  - RADIATION, UV
    - DNA DAMAGE, BIOLOGICAL FUNCTION, 3210*

- VIRUS, ADENO 5
  - DNA
    - TRANSFORMATION OF RAT CELLS, 3147

- VIRUS, ADENO 12
  - ANTIGEN
    - IMMUNE RESPONSE, MACROPHAGE INHIBITION TEST, MOUSE, 3263
  - TRANSFORMATION
    - SPECIFIC SURFACE ANTIGEN DETECTION, MOUSE, HAMSTER, 3276

- VIRUS, ADENO 16
  - INCOMPLETE PARTICLES
    - CHARACTERIZATION, 3154

- VIRUS, AMPHIBIAN LEUKOCYTE
  - ULTRASTRUCTURE
    - FROG, 3201*

- VIRUS, AVIAN MYELOBLASTOSIS
  - -INDUCED LEUKEMIA
    - REVERSE TRANSCRIPTASE, PLASMA, CHICKEN, 3169
  - INFECTION
    - SPONTANEOUS REGRESSION OF LEUKEMIA, CHICKEN, 3159

- VIRUS, AVIAN RETICULOENDOTHELIOSIS
  - TUMOR INDUCTION
    - BURSA- AND THYMUS-DEPENDENT IMMUNOSURVEILLANCE, CHICKEN, 3192*

- VIRUS, AVIAN SARCOMA
  - GENOME TRANSFER BY FUSION
    - TRANSFORMED MAMMALIAN CELLS, 3171*

- VIRUS, C-TYPE
  - ANTIGEN
    - PASSIVE HEMAGGLUTINATION TEST, 3268
  - ANTIGEN, CELL-SURFACE
    - RAT, 3234
  - COMPARATIVE CHARACTERIZATION WITH A VIRUS-FREE CELL LINE
    - VIPERA RUSSELLI, 3146
  - GROUP-SPECIFIC ANTIGENS
    - CHICKEN CELLS AND TISSUES, 3231
  - SUPERINDUCTION
    - 5-BROMODEOXYURIDINE, TRANSFORMED MOUSE CELLS, 3145

- VIRUS, C-TYPE PARTICLE
  - LYMPHOSARCOMA
    - GIBBON, 3222*

- VIRUS, C-TYPE RNA
  - HOST-CELL TROPIC
    - LYMPHORETICULAR NEOPLASMS, MOUSE, 3134
  - ISOLATION
    - LYMPHOID LEUKEMIA, CATTLE, 3158
  - LYMPHORETICULAR NEOPLASM
    - MOUSE, 3125

- VIRUS, EPSTEIN-BARR
  - ANTIGEN
    - NASOPHARYNGEAL CARCINOMA, HUMAN, 3248
  - BURKITT'S LYMPHOMA
    - ETIOLOGY, AMERICAN AND AFRICAN, 3168
    - MODES OF INFECTION, HUMAN, 3130
  - CELL VIABILITY
    - BURKITT'S CELLS, TEST METHOD, 3123
  - CHILDHOOD DISEASES
    - CASE REPORTS, 3183*
  - COINFECTION WITH RAUSCHER LEUKEMIA VIRUS
    - LYMPHOBLASTOID CELLS, HUMAN, 3121
  - ELEVATED ANTIBODY TITERS
    - SINUS HISTIOCYTOSIS, LYMPHADENOPATHY, CASE REPORT, 3225*
  - GENOME EXPRESSION
    - EFFECT OF HYDROCORTISONE, HUMAN LYMPHOBLASTOID CELL LINES, 3119
  - INFECTIOUS MONONUCLEOSIS
    - CLINICAL STUDY, 3144
  - NASOPHARYNGEAL CARCINOMA
    - EPITHELIAL CELLS, 3160
  - RETICULOPROLIFERATIVE DISEASE
    - OWL MONKEY, 3150, 3153
  - SEROLOGICAL INVESTIGATION
    - CLINICAL STUDY, 3207*
  - SUPERINFECTION
    - HUMAN LYMPHOID CELL LINES, 3128
    - SENSITIVITY OF BURKITT LYMPHOMA-DERIVED CELL LINES, 3166
  - SYNTHESIS
    - BURKITT'S CELLS, TEST METHOD, 3123
  - TRANSFORMATION
    - LYMPHOCYTES, HUMAN, 3162

- VIRUS, ERYTHROBLASTOSIS
  - TRANSFORMATION
    - GLYCOPROTEIN ALTERATIONS, CHICKEN ERYTHROCYTES, 3202*

- VIRUS, FELINE LEUKEMIA
  - HEMAGGLUTINATION-INHIBITION
    - INDIRECT HEMAGGLUTINATION TESTS, 3135
  - HOST RANGE
    - HUMAN, DOG, 3161
  - INFECTION
    - GROWTH CHARACTERISTICS, HUMAN RHABDOMYOSARCOMA CELLS, 3136

- VIRUS, FELINE SARCOMA
  - ATTEMPTED SARCOMA INDUCTION
    - CAPE CHACMA BABOON, 3220*

- VIRUS, FRIEND LEUKEMIA
  - B-TROPIC

  - HOST RANGE PSEUDOTYPE. SPLEEN FOCUS-FORMING VIRUS. 3195*
- EXTRINSIC INCUBATION POTENTIAL
  - ARTHROPODS. 3178*
- QUANTITATIVE INFECTIVITY DETERMINATIONS
  - ARTHROPODS. 3179*
- SERUM ALBUMIN SYNTHESIS
  - MOUSE LIVER. 3212*
- TRANSMISSION
  - ARTHROPODS. 3188*
- ULTRASTRUCTURE
  - CRITICAL POINT DRYING TECHNIQUE. 3197*

VIRUS. GROSS
- ANTIGEN. CELL-SURFACE
  - IMMUNOLOGICAL TOLERANCE BREAKDOWN. RAT. 3235

VIRUS. GROSS LEUKEMIA
- HEMAGGLUTINATION-INHIBITION
  - INDIRECT HEMAGGLUTINATION TESTS. 3135

VIRUS. HERPES
- INFECTION
  - PROTEIN SYNTHESIS. MOUSE. RAT CELLS. 3176*
- INFECTION. LATENT
  - CULTIVATION OF GANGLIA ON MONOFIL FABRIC. 3175*
- LUCKE TUMOR
  - RNA TRANSCRIPTION. FROG. 3124

VIRUS. HERPES ATELES
- ONCOGENICITY
  - MONKEY. 3215*

VIRUS. HERPES HOMINIS
- INFECTION
  - IODODEOXYURIDINE THERAPY. MOUSE. 3221*

VIRUS. HERPES SAIMIRI
- INFECTION
  - SQUIRREL MONKEY. 3191*

VIRUS. HERPES SIMPLEX
- CELL FUSION
  - EFFECT OF 2-DEOXY-D-GLUCOSE. 3177*
- DNA
  - DENATURATION PROPERTIES. 3186*
- -INFECTED HELA CELLS
  - DNA POLYMERASE PURIFICATION. 3138
- INFECTION
  - REVIEW. 3004
  - RNA SYNTHESIS. HUMAN EPIDERMOID CELLS. 3180*

VIRUS. LEUKOSIS
- MYCOPLASMA
  - INFECTION. MOUSE. 3200*

VIRUS. MAMMARY TUMOR
- BIOLOGICAL PROPERTIES
  - CLONES. MAMMARY TUMOR. MOUSE. 3163
- ELECTROPHORETIC MOBILITY
  - EFFECT OF PH. MOUSE. 3205*
- INFECTIVITY
  - EFFECT OF PH. MOUSE. 3205*
- MORPHOLOGY
  - EFFECT OF PH. MOUSE. 3205*
- REVIEW
  - MOUSE. HUMAN. 3005

VIRUS. MAREK'S DISEASE
- ANTIGEN
  - LYMPHOID CELLS. CHICKEN. 3257
- INFECTION
  - AUTORADIOGRAPHY OF TISSUES. 3H-THYMIDINE. CHICKS. 3174*
- INFLUENCE OF MATERNAL EFFECTS
  - FAST AND SLOW GROWING CHICKENS. 3218*

VIRUS. MAREK'S DISEASE HERPES
- PLAGUE FORMATION
  - INHIBITORY EFFECT OF MYCOSTATIN. 3214*

VIRUS. MOLONEY LEUKEMIA
- TUMOR INDUCTION
  - GROWTH INHIBITION BY BACTERIUM. MOUSE. 3282*

VIRUS. MOLONEY SARCOMA
- "8S" RNA COMPONENTS
  - ULTRASTRUCTURE. MOUSE. 3204*
- TUMOR GROWTH INHIBITION
  - SILICA GEL PF 254 ELUATE. MOUSE. 3190*
- TUMOR INDUCTION
  - COMBINED IMMUNOTHERAPY AND CHEMOTHERAPY. MOUSE. 3173*

VIRUS. MURINE LEUKEMIA
- HEMAGGLUTINATION-INHIBITION
  - INDIRECT HEMAGGLUTINATION TESTS. 3135
- HOST-CELL TROPIC
  - LYMPHORETICULAR NEOPLASMS. MOUSE. 3134
- LYMPHORETICULAR NEOPLASM
  - MOUSE. 3125

VIRUS. MURINE SARCOMA
- -INDUCED TUMOR CELL LINE
  - INHIBITORY EFFECT OF STREPTONIGRIN. 3208*
- INFECTION
  - GROWTH CHARACTERISTICS. HUMAN RHABDOMYOSARCOMA CELLS. 3136
- TRANSFORMATION
  - 5'-BROMODEOXYURIDINE. MUTAGENESIS. NONPRODUCER CELL LINES. 3157
- TRANSFORMED EMBRYO CELLS
  - NONPRODUCER CLONES. GUINEA PIG. 3187*

VIRUS. NEWCASTLE DISEASE
- CELL FUSION
  - EFFECT OF 2-DEOXY-D-GLUCOSE. 3177*

VIRUS. ONCORNA
- TRANSFORMED CELL LINES
  - FUCOSYLGLYCOLIPID METABOLISM. 3193*

    ULTRASTRUCTURE
        CRITICAL POINT DRYING TECHNIQUE, 3197*

VIRUS, ONCORNA B-TYPE
    ISOLATION
        HUMAN CANCER CELL LINES, 3131

VIRUS, PAPOVA
    -INDUCED BRAIN TUMORS
        HAMSTER, 3139
    LARYNGEAL PAPILLOMA
        ELECTRON MICROSCOPIC IDENTIFICATION, 3142

VIRUS, PICORNA
    GLUCOSAMINE
        ISOLATION FROM CAPSIDS, 3216*

VIRUS, POLYOMA
    DNA REPLICATION
        DISCONTINUOUS ELONGATION OF STRANDS AT REPLICATION FORKS, 3165
    GENE FUNCTION
        DNA SYNTHESIS, MOUSE CELLS, 3129
    TRANSFORMATION
        RNA METHYLASES, TRNA-METHYLATED BASES, MOUSE CULTURE CELLS, 3203*
    TRANSFORMED CELL LINE
        CONCANAVALIN A BINDING, 3536*
    TRANSFORMED TUMOR CELLS
        GROWTH, SPIROMETRA MANSONOIDES-INFECTED HAMSTER, 3118

VIRUS, POXVIRUS
    BIOGENESIS
        INACTIVATION OF HOST DNA POLYMERASE, 3189*

VIRUS, RADIATION LEUKEMIA
    ANTIGEN, CELL-SURFACE
        RAT, 3234
    ANTIGEN, VIRION-ENVELOPE
        RAT, 3234
    EFFECT ON CHEMICAL CARCINOGENESIS
        MOUSE, 3072

VIRUS, RAUSCHER LEUKEMIA
    ANTIBODY TO CELL-SURFACE ANTIGENS
        IMMUNIZED C57BL/6 MICE, 3117
    COINFECTION WITH EPSTEIN-BARR VIRUS
        LYMPHOBLASTOID CELLS, HUMAN, 3121
    -INDUCED LEUKEMIA
        REVERSE TRANSCRIPTASE, PLASMA, MOUSE, 3169
    LOW-LEUKEMOGENIC VARIANTS
        LONG TERM CULTURES, SUPRA-OPTIMAL TEMPERATURES, MOUSE, 3141
    PROPAGATION
        LYMPHOBLASTOID CELLS, HUMAN, 3121
    TRANSFORMATION
        ANTISCHISTOSOMAL DRUGS, RAT EMBRYO CELLS, 3149
    ULTRASTRUCTURE
        CRITICAL POINT DRYING TECHNIQUE, 3197*

VIRUS, RD-114
    CELL-SURFACE ANTIGENS
        RHABDOMYOSARCOMA, HUMAN, 3267
    INFECTION
        GROWTH CHARACTERISTICS, HUMAN RHABDOMYOSARCOMA CELLS, 3136

VIRUS, ROUS SARCOMA
    ANTIGEN, SURFACE
        HUMORAL CYTOTOXICITY TEST, HAMSTER, 3281*
    CARCINOMA PRODUCTION
        MOUSE, 3224*
    GROWTH OF AKR MOUSE CELLS
        SPINNER CULTURE, 3219*
    INDUCED SARCOMA
        ISOENZYME PATTERN, RAT, 3424*
    REVERSE TRANSCRIPTASE
        N-ETHYLENEIMIDE, TRANSFORMING ACTIVITIES, 3127
    TRANSFORMATION
        N-ETHYLENEIMIDE, 3127
        GROWTH, ANTIGENIC CHARACTERISTICS, HAMSTER CELLS, 3156
    TUMOR INDUCTION
        CHICKEN, 3120

VIRUS, SPLEEN FOCUS-FORMING
    HOST RANGE PSEUDOTYPE
        B-TROPIC FRIEND VIRUS, 3195*

VIRUS, SV40
    ANTIBODY
        T ANTIGEN, HUMAN SERA, 3238
    DNA
        GENOME, ELECTRON MICROSCOPY, 3143
        REASSORTMENT DURING SERIAL UNDILUTED PASSAGE, 3184*
    DNA ALTERATION
        HOST CELL DNA INCORPORATION, 3122
    DNA SYNTHESIS
        5-BROMODEOXYURIDINE INCORPORATION, MONKEY CELLS, 3196*
    DNA TRANSCRIPTION
        POLYADENYLIC ACID, 3126
    -INDUCED RETINOPATHY
        RAT, 3211*
    INFECTION
        DNA FRAGMENTATION, PSEUDOVIRION FORMATION, MONKEY CELLS, 3152
    PHOTODYNAMIC INACTIVATION
        TRANSFORMATION, HAMSTER CELLS, 3164
    PLAQUE MUTANTS
        CAPSID PROTEINS, ELECTROPHORETIC DIFFERENCES, 3181*
    PROTEIN
        PHOSPHORYLATION, CELL-FREE SYSTEM, 3170*
    RNA
        CHARACTERIZATION, INFECTED BS-C-1 CELLS, 3198*
    TRANSFORMATION
        CELL SURFACE PROPERTIES, 3242
        FETAL ANTIGENS, MOUSE, 3273
        IMMUNOGENICITY, IMMUNOSENSITIVITY, MOUSE CELLS, 3251
    TRANSFORMED CELL LINE
        CONCANAVALIN A BINDING, 3536*

TRANSFORMED EMBRYO CELLS
    INTRACELL FORMATION, MOUSE, 3337
TRANSFORMED HAMSTER CELLS
    INCORPORATION OF 2-DEOXY-D-GLUCOSE INTO GLYCOPROTEIN, 3206*
TRANSFORMED HUMAN DIPLOID CELLS
    GLYCERALDEHYDE-3-PHOSPHATE DEHYDROGENASE, 3172*

VIRUS, TURKEY HERPES
  SUSCEPTIBILITY TO INFECTION
    GUINEA PIG CELL CULTURES, 3213*

VIRUS, WOUND TUMOR
  RNA
    ELECTROPHORETIC SEPARATION OF ALL COMPONENTS, 3209*

WALKER CARCINOSARCOMA
  IMMUNITY
    SERUM BLOCKING FACTOR, MACROPHAGE INACTIVATION, RAT, 3249

WALKER'S TUMOR
  FORMATION OF PROTEIN GLOBULES
    TIME-LAPSE MICROCINEMATOGRAPHY, RAT, 3439*

WILM'S TUMOR
  BLOOD COAGULATION
    CLINICAL STUDY, CHILDREN, 3410*

XANTHOMA
  ERUPTIVE, TUBEROUS, PLANAR
    ULTRASTRUCTURE, LIPID BIOCHEMISTRY, HUMAN, 3528*

XENOTRANSPLANTATION
  ENDOMETRIUM
    HUMAN CELLS INTO HAMSTERS, 3312*

YOSHIDA ASCITES HEPATOMA
  MITOCHONDRIA VOLUME, FUNCTION
    TRYPSIN EFFECT, RAT, 3529*

YOSHIDA ASCITES TUMOR
  RNA SYNTHESIS
    INHIBITION, HEAT TREATMENT, MOUSE, RAT, 3402*

YOSHIDA SARCOMA
  ENDOTOXIN-LIKE SUBSTANCES
    RAT, 3590*

ZAJDELA ASCITES TUMOR
  DNA, SINGLE STRANDED
    NUCLEOLUS, RAT, 3508*

ZINC
  DEFICIENCY
    LYMPHOCYTIC LEUKEMIA, ASCITES TUMOR, MOUSE, 3352
  9,10-DIMETHYL-1,2-BENZANTHRACENE-INDUCED TUMOR
    SALIVARY GLAND, RAT, 3054

☆U.S. GOVERNMENT PRINTING OFFICE: 1974-544-915/6

LIBRARY U. OF I. URBANA

LIBRARY U. OF I. URBANA

LIBRARY U. OF I. URBANA [illegible]

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
BETHESDA, MARYLAND 20014

OFFICIAL BUSINESS

PENALTY FOR PRIVATE USE, $300

If you do not desire to continue receiving this publication, please CHECK HERE ☐; tear off this label and return it to the above address. Your name will then be promptly removed from the appropriate mailing list

DHEW Publications No. (NIH) 72-301

LIBRARY U. OF I. URBANA

UNIVERSITY OF ILLINOIS-URBANA
3 0112 084231684